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**Title**

## Innovative Stochastic Methods in the Study of Dynamical Systems

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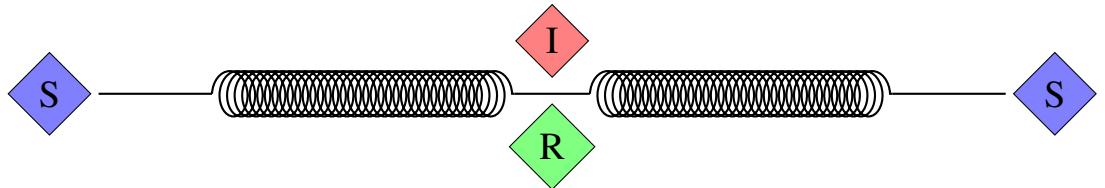
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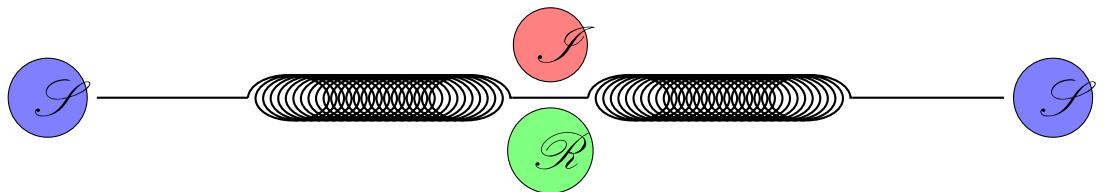
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*Innovative Stochastic Methods in the  
Study of Dynamical Systems  
Stochastic Differential Equation Systems*

# Abstract

The development of stochastic epidemiological models represents a critical advance in addressing the limitations of classical deterministic approaches. By incorporating random perturbations, these models capture the inherent variability observed in real-world epidemic data, such as fluctuations in transmission rates and heterogeneous population responses. The integration of both continuous and discontinuous stochastic processes allows for a more nuanced representation of complex phenomena, including superspreading events and the emergence of novel variants. This framework is particularly relevant in the context of rapidly evolving pathogens, where deterministic models often fail to account for the unpredictability of disease dynamics.

The analytical tractability of stochastic differential equations with Lévy jumps and Markovian switching provides a powerful tool for studying epidemic thresholds and stability properties. Through the construction of appropriate Lyapunov functions, we establish rigorous criteria for disease persistence or extinction, which are fundamentally different from their deterministic counterparts. These theoretical results are complemented by numerical simulations that reveal how noise-induced transitions can lead to unexpected epidemiological outcomes. For instance, stochastic resonance phenomena may explain the recurrent epidemic waves observed in certain diseases, even when the basic reproduction number suggests stable equilibrium.

The methodological contributions of this work extend beyond theoretical analysis to include practical computational algorithms for epidemic forecasting. We develop adaptive numerical schemes that efficiently handle the multiscale nature of stochastic epidemic models, particularly in scenarios with sudden parameter changes or regime shifts. These algorithms incorporate variance reduction techniques to improve the reliability of long-term predictions while maintaining computational feasibility. The application of these methods to historical outbreak data demonstrates their superior performance compared to conventional deterministic models, especially in capturing the timing and magnitude of epidemic peaks.

The implications of this research for public health decision-making are manifold. The stochastic framework provides quantitative measures of uncertainty in epidemic projections, enabling more informed risk assessment and resource allocation. Furthermore, explicit modeling of noise sources facilitates the identification of key drivers of epidemic variability, which can inform targeted intervention strategies. By bridging the gap between mathematical theory and epidemiological applications, this work establishes a foundation for the next generation of infectious disease models that are both mathematically rigorous and operationally relevant.

The broader significance of this research lies in its potential to transform our understanding of epidemic dynamics across multiple scales. From individual-level transmission events to population-wide spread patterns, the stochastic perspective offers unifying principles that connect microscopic variability with macroscopic outcomes. Future extensions of this work could incorporate network structures, spatial heterogeneity, or time-varying contact patterns, further enhancing the realism and predictive power of stochastic epidemic models. These developments will be crucial to address emerging challenges in global health security and pandemic preparedness.

## Keywords:

Stochastic SIRS model, Lévy jumps, hybrid switching diffusion, nonlinear incidence rate, persistence, extinction, Lyapunov function, threshold analysis, Markov chains, asymptotic stability, numerical epidemiology, public health decision-making.

# Résumé

L'élaboration de modèles épidémiologiques stochastiques constitue une avancée majeure pour pallier les limites des approches déterministes classiques. En intégrant des perturbations aléatoires, ces modèles capturent la variabilité intrinsèque des données épidémiques réelles, telle que les fluctuations des taux de transmission ou les réponses hétérogènes des populations. L'incorporation simultanée de processus stochastiques continus et discontinus permet de représenter avec finesse des phénomènes complexes comme les événements de super-propagation ou l'émergence de variants. Ce cadre théorique s'avère particulièrement pertinent pour les pathogènes à évolution rapide, où les modèles déterministes peinent à rendre compte de l'imprévisibilité des dynamiques infectieuses.

L'analyse des équations différentielles stochastiques avec sauts de Lévy et commutation markovienne offre des outils puissants pour étudier les seuils épidémiques et les propriétés de stabilité. Par la construction de fonctions de Lyapunov appropriées, nous établissons des critères rigoureux distinguant persistance et extinction des maladies, qui diffèrent fondamentalement de leurs analogues déterministes. Ces résultats théoriques s'accompagnent de simulations numériques révélant comment les transitions induites par le bruit peuvent générer des comportements épidémiques inattendus. Les phénomènes de résonance stochastique, par exemple, pourraient expliquer les vagues épidémiques récurrentes observées pour certaines maladies, même lorsque le nombre de reproduction de base suggère un équilibre stable.

L'originalité méthodologique de ce travail réside dans le développement d'algorithmes numériques adaptatifs pour la prévision épidémique. Nos schémas de calcul traitent efficacement la nature multi-échelle des modèles stochastiques, notamment lors de changements brutaux de paramètres ou de régimes épidémiques. Intégrant des techniques de réduction de variance, ces algorithmes améliorent la fiabilité des prédictions à long terme tout en conservant une faisabilité computationnelle. Leur application à des données historiques d'épidémies démontre leur supériorité par rapport aux modèles déterministes conventionnels, particulièrement pour anticiper l'amplitude et la temporalité des pics épidémiques.

Les implications de cette recherche pour la santé publique sont multiples. Le cadre stochastique fournit des mesures quantitatives de l'incertitude des projections, permettant une évaluation des risques et une allocation des ressources plus éclairées. Par ailleurs, la modélisation explicite des sources de variabilité facilite l'identification des facteurs clés influençant la dynamique épidémique, ce qui peut guider des stratégies d'intervention ciblées. En établissant un pont entre théorie mathématique et applications épidémiologiques, ce travail jette les bases d'une nouvelle génération de modèles à la fois rigoureux et opérationnels.

L'importance fondamentale de ces recherches réside dans leur capacité à renouveler notre compréhension des dynamiques infectieuses à toutes les échelles. Des événements de transmission individuels aux patterns de diffusion populationnelle, la perspective stochastique offre des principes unificateurs reliant variabilité microscopique et comportements macroscopiques. Des extensions futures pourraient intégrer des structures de réseau, l'hétérogénéité spatiale, ou la temporalité des contacts, renforçant ainsi le réalisme et la puissance prédictive des modèles. Ces développements s'avéreront cruciaux pour relever les défis émergents en sécurité sanitaire mondiale et préparation pandémique.

## Mots-clés :

Modèle SIRS stochastique, sauts de Lévy, diffusion à commutation, taux d'incidence non-linéaire, persistance, extinction, fonction de Lyapunov, analyse de seuil, chaînes de Markov, stabilité asymptotique, épidémiologie numérique, décision en santé publique.

## ملخص

مثل تطوير النماذج الوبائية العشوائية تقدماً حاسماً في معالجة قيود النهج الختمية الكلاسيكية. من خلال دمج الاضطرابات العشوائية، تلقط هذه النماذج التباين الجوهرى الملاحظ في بيانات الأوبئة الواقعية، مثل تقلبات معدلات الانتقال واستجابات السكان غير المتتجانسة. يسمح دمج كل من العمليات العشوائية المستمرة وغير المستمرة بتقريب أدق للظواهر المعقّدة، بما في ذلك أحداث الانتشار الفائق وظهور المتحورات الجديدة. هذا الإطار ذو صلة خاصة في سياق مسببات الأمراض سريعة التطور، حيث تفشل النماذج الختمية في حساب عدم القدرة على التنبؤ بدیناميکات المرض.

توفر قابلية التحليل للمعادلات التفاضلية العشوائية مع قفزات ليفي والتبديل الماركوفى أداة قوية لدراسة عتبات الوباء وخصائص الاستقرار. من خلال بناء دوال ليابونوف المناسبة، نضع معايير صارمة لاستقرار المرض أو انقراضه، والتي تختلف جوهرياً عن نظيراتها الختمية. تكل هذه النتائج النظرية محايكات عدديّة تكشف كيف يمكن للتحولات الناتجة عن الضوابط أن تؤدي إلى نتائج وباية غير متوقعة. على سبيل المثال، قد تفسر ظواهر الرنين العشوائي موجات الوباء المتكررة التي لوحظت في بعض الأمراض، حتى عندما يشير عدد التكاثر الأساسي إلى توازن مستقر.

تجاور المساهمات المنهجية لهذا العمل التحليل النظري لشمول خوارزميات حسابية عملية للتنبؤ الوبائي. تطور مخططات عدديّة تكيفية تعالج بكفاءة الطبيعة متعددة المقاييس للنماذج الوبائية العشوائية، خاصة في السيناريوهات ذات التغيرات المفاجئة في المعلومات أو التحولات النظامية. تتضمن هذه الخوارزميات تقنيات تقليل التباين لتحسين موثوقية التنبؤات طويلة المدى مع الحفاظ على الجدوى الحسالية. يوضح تطبيق هذه الطرق على بيانات تفصيّة تاريخية أداءها المتفوق مقارنة بالنماذج الختمية التقليدية، خاصة في التفاصيل تقوية وحجم الدروات الوبائية.

تطوي مسامين هذا البحث لصنع القرار في الصحة العامة على جوانب متعددة. يوفر الإطار العشوائي مقاييس كمية لعدم اليقين في التوقعات الوبائية، مما يمكن من تقييم المخاطر وتخصيص الموارد بشكل أكثر استنارة.علاوة على ذلك، فإن المندقة الصريحة لمصادر الضوضاء تسهل تحديد الحركات الرئيسية للتباين الوبائي، والتي يمكن أن توجه استراتيجيات التدخل المستهدفة. من خلال سد الفجوة بين النظرية الرياضية والتطبيقات الوبائية، يضع هذا العمل أساساً للجيبل القادم من نماذج الأمراض المعديّة التي تكون صارمة رياضياً وذات صلة عملية.

تكمن الأهمية الأوسع لهذا البحث في إمكاناته لتحويل فهمنا لدیناميکيات الوباء عبر مقاييس متعددة. من أحداث الانتقال على مستوى الفرد إلى أنماط الانتشار على مستوى السكان، يقدم المنظور العشوائي مبادئ موحدة تربط التباين المجهري بالنتائج العينانية. يمكن للتوسعات المستقبلية لهذا العمل أن تدمج هيكل الشبكات، أو عدم التجانس المكاني، أو أنماط الاتصال المتغيرة مع الزمن، مما يعزز واقعية وقوة النماذج الوبائية العشوائية التنبؤية. ستكون هذه التطورات حاسمة لمعالجة التحديات الناشئة في الأمن الصحي العالمي والاستعداد للجائح.

## كلمات مفتاحية:

نموذج SIRS العشوائي، قفزات ليفي، الانتشار المجهري بالتبديل، معدل الإصابة غير الخططي، الاستمرارية، الانقراض، دالة ليابونوف، تحليل العتبة، السلاسل الماركوفية، الاستقرار التقاربى، الوبائيات العددية، صنع القرار في الصحة العامة.

# Declaration

The author hereby declares that this thesis and the work presented here are my own. I confirm the following:

- ◆ This thesis represents my original contribution to the field of mathematics, except where explicitly stated in the preface or acknowledgments.
- ◆ Proper attribution has been given to all sources, including previously published work, ideas, and results that are not my own.
- ◆ The thesis does not exceed 400,000 *characters*, excluding tables, figures, bibliographies, and appendices.

KHALID EL BAKKIOUI, September 1, 2025.

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If, inadvertently, I have omitted to mention someone who contributed directly or indirectly to this work, I beg their forgiveness for this oversight. As the saying rightly goes: "*To err is human*"... and this modest work certainly bears its mark.

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# General Introduction

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*E*xcellent works and numerous research themes have been dedicated to the integration of mathematical models in epidemiological studies and the preventive approach to epidemics and infectious diseases. The primary motivation behind this thesis, titled Innovative Stochastic Methods in the Study of Dynamical Systems, is to incorporate the fascinating and rigorous aspects of mathematics into epidemiology, biology and, more broadly, into understanding the mechanisms underlying the dynamics of infectious disease spread, such as COVID-19. We believe that theoretical challenges, through the proposal of mathematical models whose formulation, resolution and constraints are influenced by the choice of epidemiological parameters, are inspired by the factors governing the infectious dynamics of an epidemic [1–4].

The proposed models offer investigative perspectives to understand pandemic situations, where the severity and extinction of the disease are linked to specific factors. These factors are precisely highlighted by mathematical models, particularly in the interpretation of epidemic curves derived from their solutions. These curves are fundamental for understanding the factors involved in the spread of a pandemic.

In the mathematical modeling of epidemic spread or disease contagion, there is a preliminary concern about the reliability of the model itself, which is tied to the mechanisms of disease transmission [5–7]. In real-world epidemiological situations, these mechanisms are numerous and sometimes complex, raising the persistent question of whether the proposed mathematical model accurately reflects real-world conditions.

It is unquestionable that the spread of an epidemic is, first and foremost, a social phenomenon rooted in the population, with its diversity, modes of functioning, geographical organization, culture and socioeconomic structure [5, 7]. An obvious question arises: Can a mathematical model or any other form of modeling incorporate this complexity into its equations or representations? Therefore, it is necessary to categorize the models into two classes: simple models and complex or comprehensive models.

Chronologically, the **SIR** model (standing for Susceptible Infected Recovered) is a pioneering and classic model in epidemiological modeling. It is one of the simplest mathematical models for spreading disease. During an epidemic, the SIR model divides the population into three (3) basic categories or compartments (3) based on the disease status. This model is not absolute, but relies on simplifying assumptions that remain realistic in an epidemiological context. One such assumption is that every individual or group of individuals is vulnerable and susceptible to contracting the disease. They are potentially infectable but not symptomatic. In other words, there is no innate immunity. This category of individuals forms the **S** compartment of susceptibles [8–10].

The dynamics of epidemic spread occurs through interindividual contacts (in any form) and the infection process itself. Infected or symptomatic individuals, those exhibiting external symp-

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toms due to the pathogen, form the second compartment, known as the "infected" compartment (**I**). The stay of infected individuals in this compartment is transient, as they are subject to two possible outcomes: recovery or immunity, and death. Individuals falling into these two categories are grouped in a third compartment labeled **R** for recovered, although deaths are also counted here.

The environment of the SIR model can take two forms: a closed environment in which the model is isolated, meaning that it is not subject to any demographic constraints, such as birth or death rates upstream of the **S** and **I** compartments. This is the simplest case to formulate mathematically through straightforward logical reasoning. The other form slightly complicates the model by adding a demographic component of birth or death rates; even if negligible, the inclusion of this component affects the shape of the epidemic curves **S**, **I**, and **R**.

To understand the principle of epidemic spread, the concept of system dynamics from physics is invoked, where the notion of flow (incoming or outgoing) underlies the exchanges between system components. The SIR model adheres to this principle by taking the **S** compartment as the reference upstream compartment (flow of susceptible individuals leaving this compartment to enter the **I** compartment) and a downstream flow consisting of immunized or deceased individuals, who then enter the **R** compartment [8–11]. The arithmetic signs in the model's equations account for this migratory movement.

As mentioned above, a mathematical model of epidemic spread must demonstrate its reliability and reflect, as much as possible, the prevailing health situation during an epidemic crisis. The notion of migratory flows between susceptible, infected, and recovered individuals requires the definition of driving parameters that allow some individuals to leave one compartment and join another. This transition occurs through the definition and selection of specific state parameters, which are fundamental once they are integrated into the mathematical equations of the model. In epidemiological modeling, these parameters are more similar to rates or even probabilities [12–15].

The spread of an epidemic has been observed to originate from the nature and frequency of contacts between asymptomatic individuals and infected, symptomatic individuals. These contacts are a societal phenomenon and can take many forms: handshakes, hugs, social proximity (referred to as distancing), professional relationships, etc. It would be tedious to enumerate them all, as they are numerous and complex. However, they are related to the type of social organization of the population, its density, cultural and religious foundations, and economic activities [16, 17].

In the simplified SIR model, two fundamental parameters must be specified: the contamination rate or infection parameter of a susceptible individual by an infected or infectious individual, and the recovery rate coupled with the proportion of deaths resulting from the epidemic. These parameters are challenging to define as they can be random. For the infection rate, it is necessary to consider both the number and the nature of contacts between susceptible and infected individuals. Regarding the recovery rate, its value is strongly influenced, on the one hand, by the treatment administered to infected individuals and, on the other hand, by preventive health measures such as barrier measures (mask wearing and social distancing). Quantifying the effectiveness of these measures remains a significant challenge in epidemiological modeling. However, mathematics, with its rigor and objectivity, can assist epidemiologists by providing arguments that contribute to their decision-making in terms of health policy [18].

The SIR model mentioned earlier can be considered a prototype in the mathematical modeling of epidemic spread. However, it cannot claim to be a universal model because of its inability to represent all or even part of the situations that prevail during an epidemic crisis. The idea of incorporating additional compartments deemed relevant to this basic model could lead to a more realistic approach to the epidemiological situation. This is how a **E** compartment (E: Exposed) was introduced, inserted between the **S** and **I** compartments, comprising individuals who host

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the pathogen but have not yet reached the infected status. The resulting model is called the **SEIR** model. In addition, a **H** compartment (H: Hospitalized) can be linked to the infected compartment **I** and upstream of the **R** compartment. This would give rise to models such as **SIHR** or **SEIHR** [17].

Among the other assumptions made a priori in the SIR model is that a recovered individual acquires permanent immunity to the disease. In cases where this immunity is partial or time limited, an immunized individual can become susceptible to contracting the disease again, in which case they would reenter the **S** compartment. These models are referred to as **SIRS** or **SEIRS** [10, 19]. It is worth noting that this involves an operation of increasing the complexity of the basic SIR model, accompanied by a corresponding complexity in the mathematical model and its equations, which must nevertheless be solved.

Other configurations with minimal impact on the epidemic spread scenario can also be considered. These configurations, of local or limited scope, are obtained by removing compartments from the models mentioned above. Examples include the **SI** (Susceptible-Infected), **SIS**, **SEI**, **SEIS**, and **SRIS** models, among others.

Regarding the vaccination strategy, two approaches can be distinguished. The first involves vaccination as a routine operation that forces the recovery of an infected individual [20–22].

In the corresponding mathematical model, it suffices to introduce a parameter called the "vaccination rate" to observe the effects of vaccination efficacy on epidemic curves. The other aspect of vaccination to which mathematical modeling of an epidemic can contribute is related to large-scale vaccination strategies at the level of a country or a geographical region. What percentage of the global population needs to be vaccinated to slow the progression of the epidemic? How is this percentage calculated? What parameters should be included in its calculation? Is there a unique and universal mathematical expression to determine this percentage?

The mathematical approach to partial or total lockdown of a population in the case of a virulent epidemic is possible but subjective, depending on the mathematical model and the choice of parameters describing the lockdown. If lockdown is understood as a restriction on the activities or movement of a population, how can this restriction be quantified and expressed in equations? Can a confinement coefficient be defined and what percentage of the population should be confined? Total or partial lockdown? A mathematical model is proposed with the choice of a coefficient, but it is primarily based on the time shift (delayed time) of the appearance or situational effects of an epidemic. The effectiveness of a lockdown will be measured by the degree of flattening of the epidemic curve of infected individuals and the time required to achieve this flattening, also referred to as the lockdown period.

Various approaches and models used in this field are often cited in the scientific literature [1, 5–10, 15–20, 22–24].

The assertion that the spread of an epidemic resembles the principles of dynamical systems, as defined in physics, opens new avenues and provides an extended methodology for analyzing epidemiological processes. Currently, the most relevant dynamical systems are those that are non-linear and exhibit specific evolutionary characteristics over time. We will focus on the following two key aspects.

- The mathematical formulation of partial differential equations that govern both the evolution of nonlinear dynamical systems and the spread of epidemics is inherently nonlinear. Analytical solutions to these equations are often challenging because of the lack of a universal method for solving them.
- Computational tools are particularly well suited for graphically representing solutions, such as epidemic curves  $S(t)$ ,  $I(t)$ , and  $R(t)$ , among others. In this context, software such as Python [Download Python](#) or MATLAB, with its specialized toolbox [Download MATLAB](#), is highly recommended to solve the equations of the epidemiological models discussed above.

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In physics, non-linear dynamical systems are precursors to chaotic behavior and the unpredictability of phase trajectories. This phenomenon is accentuated by the choice of initial conditions when solving differential equations. This is referred to as the "sensitivity to initial conditions" of phase trajectories, and the observed phenomenon is known as the "butterfly effect" [25, 26].

These observations can be extrapolated to the non-linear process of epidemic propagation, provided that we identify what could represent an initial condition capable of promoting large-scale spread. This factor is the basic reproduction number  $R_0$ . Its definition is based on the following considerations. The origin of an infection stems from the existence of an infectious individual, referred to as the source of the infection or generation 0 of infected individuals. The driving force behind the transmission of disease has been the degree and nature of interpersonal contacts. If the "source" individual initially infects  $n$  others, the epidemic propagation process is triggered, as each of these  $n$  individuals (generation 1) becomes a new infectious source and infects  $n$  others. This process continues as long as the epidemic persists and preventive measures (mainly sanitary measures) do not significantly impact the main epidemic curve  $I(t)$  [10, 17, 18, 27–29].

Determining the basic reproduction number  $R_0$  is crucial, as its value serves as a guide to implement preventive sanitary measures. However, accurately estimating  $R_0$  is challenging due to the complexity of social interactions between individuals. For example, during the COVID-19 pandemic, some epidemiologists estimated  $R_0$  between 2 and 2.5 ( $2 < R_0 \leq 2.5$ ), although this value is not universal. An intermediate value of  $R_0 = 2.3$  has also been suggested.

A fundamental limitation of the basic reproduction number, as defined above, is that it is implicitly assumed to remain constant throughout the epidemic. However, in an endemic situation, preventive sanitary measures are implemented during specific periods. These measures aim to reduce the number of new infections, necessitating an update of  $R_0$  over time. This updated measure is known as the "effective reproduction number  $R_{\text{eff}}$ " [16, 30–32].

The basic reproduction number  $R_0$ : A warning tool. It should be noted that, assuming a constant basic reproduction number during an epidemic, the cumulative number of infected individuals over a given period follows a geometric progression with a ratio equal to  $R_0$ . Extrapolating the conditions for the convergence (or divergence) of this geometric progression in epidemiology leads to the following reasoning [16, 30–32]:

- It is known that a geometric progression converges when the ratio is less than one, that is,  $0 < R_0 < 1$ . In epidemiological terms, this translates into a decreasing trend in the number of infected individuals or, in the extreme case, the absence of infections, which means that there is no epidemic.
- When the ratio exceeds one, that is,  $R_0 > 1$ , the geometric progression diverges. In epidemiological terms, this indicates an increasing trend in the cumulative number of infected individuals, indicating the expansion of the epidemic.

The threshold value  $R_0 = 1$  therefore serves as a warning indicator for the onset of an epidemic when  $R_0 > 1$ . The closer the basic reproduction number is to this threshold, the more virulent the epidemic, as seen in the case of COVID-19. In contrast, if  $0 < R_0 < 1$ , the trend confirms a regression of the disease. For COVID-19, determining  $R_0$  could help assess the effectiveness of preventive measures or guide public health policies.

A recurring challenge during mass vaccination campaigns in the context of an epidemic is to determine the optimal vaccination coverage rate. Vaccinating the entire population is often impractical due to organizational and economic constraints [33]. A balance must be struck between the vaccination coverage rate and the desired outcomes of the vaccination effort [34]. In the context of an epidemic, a vaccination campaign can be designed to achieve two complementary objectives.

- Vaccinating a specific proportion of the population to ensure immunity and reduce sus-

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ceptibility to the pathogen over time, resulting in acquired immunity. In this case, the proportion  $p$  of the population must satisfy

$$p > \left(1 - \frac{1}{R_0}\right),$$

where  $R_0$  is the basic reproduction number.

- If the goal of vaccination is to slow the progression of the disease, the proportion  $p$  of the population that will be vaccinated while remaining susceptible to infection is given by

$$p = \frac{1}{R_0}.$$

For example, assuming  $R_0 = 2.3$  for COVID-19, the required vaccination coverage rates would be  $p_1 = 56.52\%$  for the first objective and  $p_2 = 43.47\%$  for the second. In a country with a population of 37,000,000 (estimated population of Morocco in 2023), this would translate to the following approximate numbers of individuals to be vaccinated:

- First case:  $N_1 \approx 20,912,400$  individuals to be vaccinated.
- Second case:  $N_2 \approx 16,083,900$  individuals to be vaccinated.

It is important to note that these values are not definitive and should be adjusted downward, as not all individuals in a population are eligible for vaccination (for example, due to age restrictions). Furthermore, these values depend on the determination of  $R_0$ , which itself is influenced by factors such as infection and recovery rates. Calculating  $R_0$  for a given epidemic remains a topic of ongoing research and discussion in epidemiology and mathematics [16].

The temporal evolution of the infected population is described by the curve  $I(t)$ . Analyzing its trends, variations, and specific characteristics provides valuable information on the diagnosis of the epidemic and the prognosis of its future trajectory. For example, based on fixed epidemiological parameters, it is possible to predict the maximum number of infected individuals during a given period, estimate the duration of the epidemic, and conclude that every epidemic will eventually end (i.e. reach extinction). These conclusions, while perhaps intuitive, are derived from the function  $I(t)$ , making it a critical tool for understanding the temporal dynamics of a pandemic.

The concept of percolation is inherently mathematical and is deeply rooted in probability theory. In physics, percolation is similar to a phase-transition phenomenon that occurs in a network where vertices (or sites) and edges (or links) are randomly removed. Beyond a certain threshold, this removal process leads to the formation of clusters of increasingly smaller sizes. For a network with  $N$  sites, if  $N_1$  denotes the number of elements (individuals) that occupy a site, the probability of occupying a site is given by  $p = \frac{N_1}{N}$ , where  $0 \leq p \leq 1$ . A cluster represents a group of individuals connected through at least one neighbor. If  $A_{\max}$  denotes the size of the largest cluster, there exists a critical probability  $p = p_c$ , known as the "percolation threshold", at which a percolation transition occurs. In the limit where the number of sites  $N$  approaches infinity ( $N \rightarrow \infty$ ),  $A_{\max}$  becomes finite if  $p < p_c$  and infinite if  $p > p_c$ .

In epidemiology, the transition of a disease that affects a population to an epidemic regime resembles a phase transition. There exists a probability, called the "epidemic threshold", below which the disease remains localized and does not escalate into an epidemic but above which it mutates into an epidemic. The value of the critical probability  $p_c$  depends on the geometry of the network. For example, in a square grid under site percolation, the critical threshold is  $p_c = 0.593$ .

The study of physical quantities, such as the number of epidemiological infections, near the percolation threshold  $p_c$  is of significant interest. These quantities often follow a power-law behavior of the form  $(p - p_c)^\alpha$ , where  $\alpha$  is the "critical exponent" [16]. Similar reasoning can be applied to bond percolation.

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The propagation of forest fires can be seen as a simplified analogy to percolation, where the network (square grid) is replaced by a forest and the sites represent trees. A propagation rule is defined to simulate the spread of fire from one tree to another. The clusters in this context represent groups of burned trees. The percolation threshold then serves as a reference point indicating the state of the forest fire.

The application of network theory (or graph theory) to the study of epidemic propagation arises from the social organization and spatial distribution of individuals within a population. In this context, the concept of a "node" is extended to represent various social structures, such as individuals, families, tribes, communities, and so on. The notion of an "edge" or "link" refers to the social relationships between individuals or communities, collectively termed communication.

The spread of an epidemic through a network depends largely on the internal structure of the network, including the density of nodes (population density, number of communities, etc.) and the links governing the coexistence of individuals and communities (in epidemiology, the number and nature of contacts and interactions between individuals). The extrapolation of network concepts, particularly their structural characteristics (network architecture), naturally aligns with the organization of society.

Epidemic propagation is a contact-based phenomenon, much like the dissemination of information in a network. The spatial distribution patterns of a population are reflected in the geometry of the network and the types of connections between nodes. However, network theory introduces concepts that are often overlooked in traditional social analyses but are crucial to understanding how epidemics spread within a population. For example, the notions of distance and shortcuts between nodes or groups of nodes can shed light on alternative forms of contact between individuals. The diameter of a network can correspond to the size of a population and the disparities in spatial occupation. Certain types of network can reveal potential modes of disease transmission. For example, the "small world" network or the concept of six degrees of separation highlights both the mode and speed of epidemic propagation, which traditional epidemiological methods might not capture [35].

The spatiotemporal aspect of epidemic development and infectious diseases is a critical concern in epidemiological studies [16, 36]. A key question arises: How can spatial effects of propagation be incorporated and quantified? A reasonable approach would be to include a spatial variable  $x$  along with the time variable  $t$  in mathematical models of epidemic spread, such as the SIR model or others. In this case, the "susceptible" and "infected" functions  $S$  and  $I$  would depend on both variables, becoming  $S(x, t)$  and  $I(x, t)$ , respectively. The resulting differential equations would be more complex. However, spatial propagation requires a driving mechanism to sustain its dynamics. Here, a well-known phenomenon in the dynamics of sciences comes into play: diffusion. The propagation process would then be modeled by combining a two-dimensional framework with the diffusion phenomenon.

Diffusion refers to the process by which an expansive agent spreads through a favorable medium to achieve a uniform state or distribution. This phenomenon is widely used in various fields, from chemistry to the biological and epidemiological sciences. In nature, diffusion describes the tendency of particles, atoms, or molecules to spread spatially due to energetic excitation. Epidemics, for example, are characterized by their extensive geographical spread, often reaching multiple continents [37, 38].

Diffusion is fundamentally based on the process of random motion, which has strong probabilistic connotations. It operates on variable spatial and/or temporal scales, ranging from microscopic levels to significantly larger dimensions.

In the context of epidemic propagation, two key factors describe the spread of a disease:

- **Territoriality or Diffusion Medium:** This involves the geometry and dimensions of the environment, concepts that are also central to solving diffusion equations.
- **Mode of Diffusion:** This refers to the fundamental mechanism that drives the spread of

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diseases.

The following illustrations provide an overview of these concepts.

By solving the integro-differential system describing the phenomenon of spatial epidemic propagation, we gain insight into the qualitative nature of this propagation. This understanding comes from the nature of the solutions and their graphical representation. In the context of the present study, it is shown that the spatial spread of an epidemic follows a wave-like law of the form  $z = x - ct$ , where  $c$  represents the speed of the wave or the velocity of propagation. It is further highlighted that for the propagation phenomenon to occur, certain conditions must be satisfied by this velocity, particularly its dependence on the basic reproduction number  $R_0$  [39–41].

**In this thesis**, advanced mathematical approaches to epidemiological modeling are developed through seven interconnected research components, each offering unique insights into the field.

Chapter 1 sets the foundational framework for this research by introducing key concepts such as population dynamics, core epidemiological principles, and disease transmission models. These theoretical elements provide the necessary background for the analytical developments presented in the subsequent chapters.

Chapter 1 provides a detailed overview of dynamical systems and their applications in mathematical biology. It begins by distinguishing between deterministic and stochastic frameworks and explores essential principles of model classification and interpretation within epidemiology. Classical population interaction models, such as the Lotka–Volterra system, are introduced along with dynamic formulations applicable to disease propagation. The chapter places particular emphasis on compartmental models, progressing from the basic SIR framework to extended versions such as SIS, SIRS, and SEIR. These variants incorporate demographic processes, declining immunity, and stochastic effects. The chapter concludes with an analysis of equilibrium states, epidemic curves, and the influence of noise and diffusion on transmission dynamics.

Chapter 2 advances the modeling framework by introducing sophisticated stochastic methods suitable for dynamic epidemiological systems. It begins with foundational concepts such as Brownian motion, Lévy processes, and general stochastic processes, paving the way for stochastic integration, including multidimensional Itô calculus and related inequalities. The chapter then examines stochastic differential equations, focusing on the existence and uniqueness of solutions, as well as numerical schemes such as Euler–Maruyama. In addition, it investigates various notions of stability, including probabilistic and exponential stability, and addresses the ergodic properties of solutions, discussing recurrence, transience, and long-term statistical behavior of stochastic models.

Chapter 3 presents one of the core theoretical contributions of this thesis by analyzing the effect of jump perturbations on stochastic SIRS models. Provides rigorous analytical results concerning the positivity, existence, persistence, and extinction of solutions in the presence of random jumps. The chapter includes extensive numerical simulations that illustrate these results, offering graphical interpretations of both persistence and extinction scenarios. By combining mathematical proofs with computational experiments, we underscore the complex interaction between stochasticity and epidemic dynamics. The discussion concludes with perspectives for further model refinement.

Chapter 4 delivers another major theoretical advancement by establishing necessary and sufficient conditions for the behavior of stochastic SIRS models with switched transmission rate exponents. It offers rigorous results on extinction and persistence, supported by detailed analyses of disease-free equilibrium stability and various stochastic stability criteria. Using two-state Markov chains, the chapter explores several parameter regimes to illustrate transitions between extinction and persistence. These theoretical results are complemented by comprehensive simulations that reveal the dynamic complexity of the model. This contribution significantly improves

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our understanding of stochastic influences in variable transmission environments.

Chapter 4.6 synthesizes the key findings of this work, offering a reflective overview of their implications for mathematical epidemiology. It contrasts the strengths and limitations of compartmental versus agent-based models, highlighting their suitability depending on the complexity and objectives of the study. The chapter revisits critical questions related to epidemic control, such as herd immunity, vaccination strategies, spontaneous resolution, and uncertainties about the duration of immunity. It emphasizes the value of adaptable modeling frameworks to support public health decision making and highlights the role of simulations and accessible communication in improving public understanding of epidemic dynamics.

Appendix A contains a structured collection of Python implementations corresponding to the main chapters. Each section documents the relevant code used for simulations, analyses, and visualizations, ensuring transparency and reproducibility of the computational results. The appendix begins with a general overview, followed by sections aligned with Chapters 1 through 5. These implementations not only reinforce the theoretical developments but also provide a foundation for further research and practical applications. The appendix concludes with remarks on potential enhancements and extensions to the code base.

Appendix B provides a systematically organized reference table of common stochastic integrals, including Itô-type identities and key results from stochastic calculus. These formulas are essential tools for the analytical derivations presented in Chapters 2–4, particularly for stochastic differential equations with jumps and switching regimes.

Appendix C offers a dual historical and lexical resource, featuring: (C.2) a bilingual English/French glossary of stochastic calculus terminology, preserving original naming conventions and standardizing abbreviations across linguistic traditions; (C.3) a reverse chronological timeline of key contributors to Brownian motion and stochastic calculus, contextualizing the theoretical foundations used throughout this work. This appendix bridges the mathematical content with its interdisciplinary origins in physics, biology, and finance.

This thesis successfully integrates theoretical innovation with practical applicability, offers novel mathematical methodologies for the analysis of epidemic dynamics, and contributes robust tools for public health research. The findings improve our capacity to model the complexity of infectious disease transmission while maintaining high standards of mathematical rigor and analytical clarity.

# CHAPTER 1

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## Introduction to Dynamical Systems and the SIR Model with Its Variations

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### 1.1 Dynamical Systems

#### 1.1.1 Concept of Dynamical Systems

Dynamical systems are a branch of mathematics that is focused on the study of systems governed by a coherent set of laws, typically involving time-dependent processes such as difference and differential equations. The primary goal of studying dynamical systems is to understand the geometric properties of trajectories and long-term behavior. These systems can model a wide range of behaviors, including the motion of planets in solar systems, the spread of diseases in populations, the growth and form of plants, the interaction of optical pulses, and the processes regulating electronic circuits and the heart [42–47].

A dynamical system can be defined as a set of entities, or components of the system, that interact with each other, ensuring its evolution over time, *that is*, its temporal dynamic evolution. A unified and descriptive theory can be formulated. A dynamical system consists of an abstract phase space or a state space, whose coordinates describe the state at any given time, and a dynamic rule that specifies the immediate future of all state variables based solely on their current values.

A dynamical system is described by a set of dynamic variables, which can be represented as a column vector  $\mathbf{X} = (x_1, x_2, \dots, x_n)^T$  in  $\mathbb{R}^n$ . The state of the system at a given time  $t$  is uniquely described by a point  $\mathbf{X}$  in the phase space. The terms  $x_i$  are generalized coordinates that can represent various quantities. Collectively, the values associated with the quantities of the system's entities evolve over time. The system's behavior arises from the temporal variations of these values, making it essential to understand and predict the system's dynamics. The formal study of dynamical systems involves examining mathematical models specific to a particular discipline, such as epidemiology, as explored in this work through the process of epidemic spread. Some systems can be derived from fundamental principles and tested to demonstrate sufficient experimental precision, allowing the development of analytical tools and potentially unraveling their complexity.

## 1.1.2 Classes of Dynamical Systems

Dynamical systems can be classified into three main categories: deterministic systems, stochastic or random systems, and systems exhibiting chaotic behavior.

### 1.1.2.1 Deterministic and Stochastic Dynamical Systems

The global evolution  $\varphi(t)$  of a deterministic dynamical system is uniquely determined once an initial state  $\varphi(t_0)$  is specified. The system's evolution over time is described by a first-order differential equation of the form

$$\frac{d\varphi(t)}{dt} = f[\varphi(t), u], \quad (1.1)$$

where  $f$  is a nonlinear function of the coordinates  $\varphi$ . Furthermore,  $f$  can explicitly depend on time  $t$ . If  $f$  does not depend on time, the system is termed *autonomous*. The parameter  $u$  serves as a control parameter, and its values can significantly impact the dynamics of the system, potentially leading to changes in its qualitative behavior. Stochastic dynamical systems, also known as random systems, are characterized by a probability distribution of possible states. Stochastic dynamics has opened up numerous avenues for modeling and analysis, yielding results on invariant manifolds, global attractors, and invariant measures for such systems.

Stochastic effects are of paramount importance in model development. Random events occur in the physical world and throughout our daily experiences. Incorporating the mathematics of complex phenomena under uncertainty is essential for practical applications. Macroscopic models for these systems incorporate randomness in various ways, such as stochastic forcing, uncertain parameters, random sources or inputs, and random initial or boundary conditions. The theory of stochastic dynamical systems and stochastic differential equations provides fundamental insights and tools for modeling, analyzing, and predicting complex phenomena [48–50].

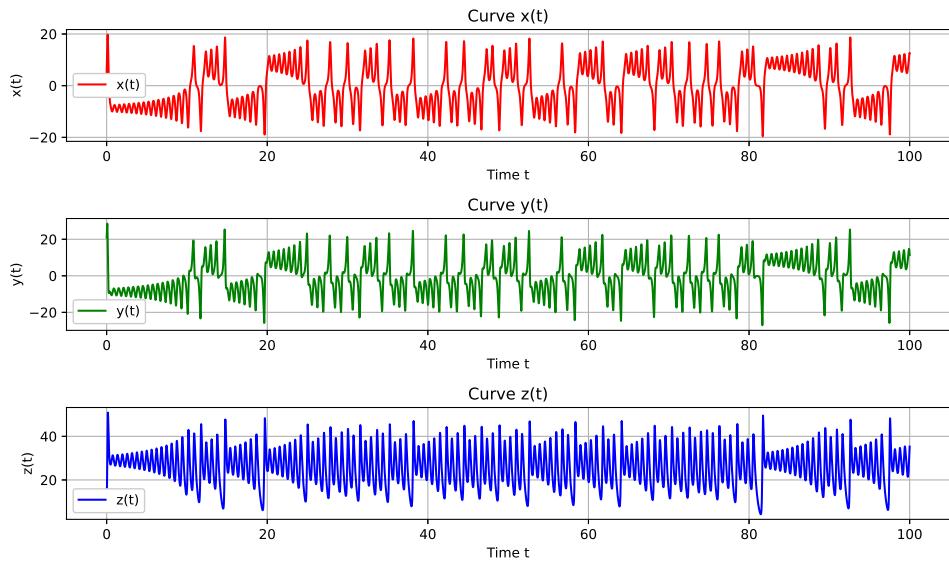
### 1.1.2.2 A Classic Model of a Nonlinear Dynamical System

The Lorenz model, a well-known nonlinear system often referred to as the Lorenz attractor, is defined by the following system of equations:

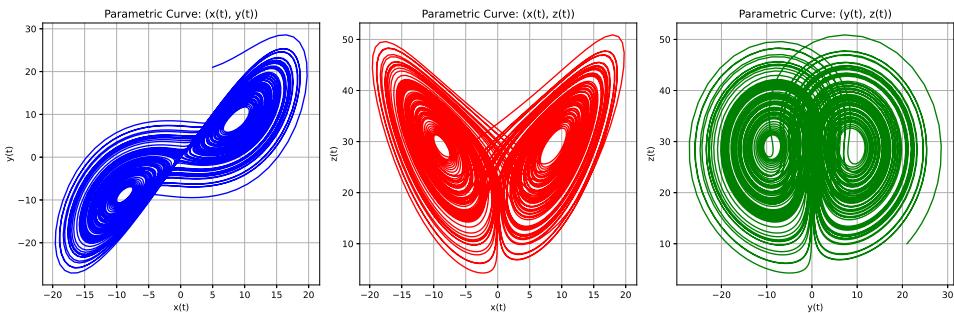
$$\begin{cases} \frac{dx}{dt} &= \sigma(y - x), \\ \frac{dy}{dt} &= x(\rho - z) - y, \\ \frac{dz}{dt} &= xy - \beta z, \end{cases}$$

where  $x = x(t)$ ,  $y = y(t)$ , and  $z = z(t)$ . The time interval is set to  $t \in [0, 100]$ , with initial conditions  $x(0) = 5$ ,  $y(0) = 21$ , and  $z(0) = 10$ . The system parameters are  $\sigma = 10$ ,  $\rho = 30$ , and  $\beta = 8/3$ . The following figures show the functions  $t \mapsto x(t)$ ,  $t \mapsto y(t)$ , and  $t \mapsto z(t)$  (Figure 1.1). For the code, see A.1. The following figures illustrate cross-sectional representations of unknown functions  $x(t)$ ,  $y(t)$ , and  $z(t)$  (Figure 1.2). For the code, see A.2. In this work, this procedure for partial phase trajectory representations, such as  $(x(t), y(t))$ ,  $(y(t), z(t))$ , and  $(x(t), z(t))$ , will be applied to the epidemiological SIR model. In the Lorenz example, it is also possible to provide a three-dimensional representation  $t \mapsto (x(t), y(t), z(t))$  of the phase trajectories (Figure 1.3). For the code, see A.3.

## 1.2 General Concepts on Mathematical Modeling



**Figure 1.1.** Representation of the functions  $t \mapsto x(t)$ ,  $t \mapsto y(t)$ , and  $t \mapsto z(t)$ .

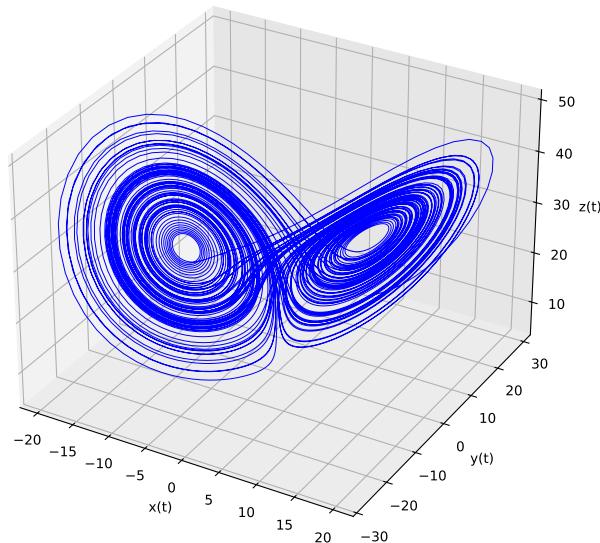


**Figure 1.2.** A complex dynamical model: the Lorenz model with parametric curves  $t \mapsto (x(t), y(t))$ ,  $t \mapsto (x(t), z(t))$ , and  $t \mapsto (y(t), z(t))$ .

### 1.2.1 Mathematical Modeling

Mathematical modeling is an approach that generates a model. Today, it is considered an effective method for reproducing real-world phenomena in the form of equations, dealing with variables and parameters. As such, modeling can be seen as a symbolic representation of certain aspects of an object or a real-world phenomenon, such as the spread of an epidemic. It is worth noting that mathematical models are not the only form of modeling. In the absence of measurements, schematic representations can serve as an alternative [44, 45]. In both cases, modeling proves to be an effective approach, especially when combined with rigorous experimental methods and their associated constraints. The process of modeling a physical phenomenon or event is delicate, time-consuming, and often subject to criticism [43, 49].

3D Parametric Curve:  $(x(t), y(t), z(t))$



**Figure 1.3.** Three-dimensional representation of the phase trajectories of the parametric curve  $t \mapsto (x(t), y(t), z(t))$ .

## 1.2.2 Model Classification and Characterization in Mathematical Modeling

Although it can vary in formality, the act of mathematical modeling follows certain principles that reinforce its status as a scientific approach [43–45, 49].

- Define the problem of the phenomenon to be modeled, highlighting the questions to be addressed.
- Identify the actions to be taken.
- Take into account the available data and knowledge, accessible through experiments and observations.

A mathematical model is a description of a system or process (epidemiology is no exception) using mathematical tools and language. The process of developing mathematical models is called mathematical modeling. We will focus on modeling infectious diseases and their spread. Mathematical models are developed to help explain a system, study the effects of its various components, and make predictions about its behavior [42–44]. Some advantages include:

- Mathematics is a highly precise language that helps us to formulate ideas and identify assumptions.
- Mathematics is a concise language with well-defined rules for manipulation.
- We have access to a set of concepts, both theoretical and practical, that are the legacy of the discoveries of mathematicians' work and throughout history.
- Advances in technology have provided numerical computation, solution, and visualiza-

tion tools that complement our curiosity to understand even the most minute or discrete processes.

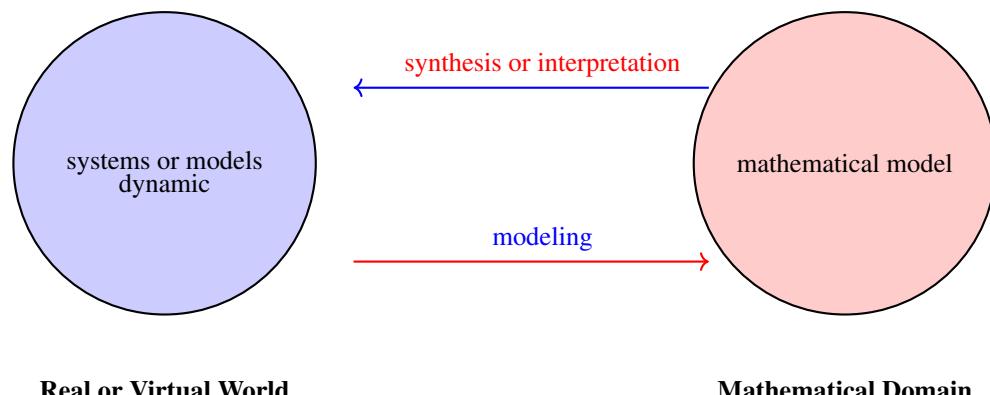
Mathematical models typically consist of parameters and variables connected by precise and well-thought-out formulations: state equations. Variables are abstractions of the properties of the system that can be quantified or measured. Models can be classified on the basis of the process being modeled, such as dynamic/static models. A dynamic model accounts for changes in the state of the system over time, while a static model calculates system quantities under the assumption that the system does not change over time and is therefore time invariant. Dynamic models often use differential equations or difference equations.

The models we will consider in this work are dynamic models [43, 44]. Discrete models treat time or system states as discrete. Continuous models integrate the time and system states as continuous. A deterministic model is one in which each set of variables is uniquely determined by the model parameters and the initial state of the variables. Stochastic models are characterized by randomness, and the states of variables are described by probability distributions. In addition to these two main classes of models, the following specific forms of modeling are also commonly applied. Geometric models Simulation models and Data structure-based models

### **1.3 Requirements, Development, and Interpretation of Mathematical Models in Epidemiology**

The modeling process involves translating a given scenario (biological, epidemiological, etc.) into a mathematical problem. This process typically begins with a clear description of processes or systems based on the scientist's understanding of the system. Translating into mathematical equations must be done with a specific objective in mind. The mathematical model should only incorporate features that are relevant to the specific goals at hand. Solving the model leads to solutions that are intended for:

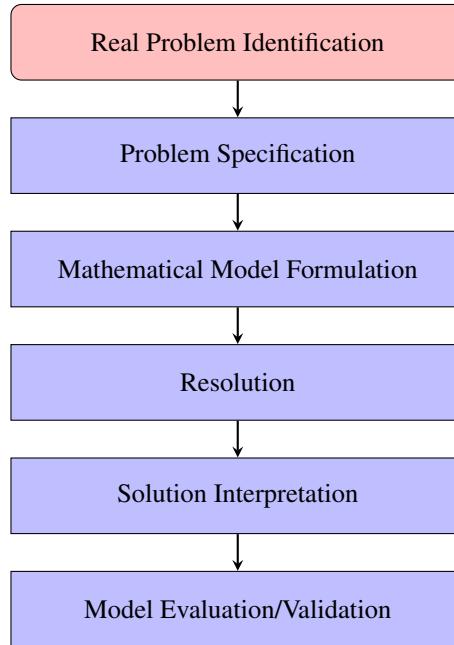
- Analysis to produce critical quantities governing the overall behavior of the solutions
- Estimation of the model parameters
- Manipulation of these parameters through simulation to assess their importance in the nature of the solution
- Adaptation to available data or data used to simulate experiments that are likely to produce results



**Figure 1.4.** Schematic diagram of modeling and its synthesis

Once the model is understood, its results must be interpreted in the context of the considered sce-

nario, potentially addressing the initial question. At the very least, we must answer the following questions: What have we learned about the real world through the model? Is the message of our model supported by information about the system? The following flowchart describes a type of mathematical modeling process for a specified phenomenon with the ultimate goal of evaluating or validating the initiated mathematical model.



**Figure 1.5.** Workflow in a modeling process

Infectious agents have shaped the world, driving public health toward simplicity throughout history. The selection and adoption of a mathematical model in epidemiology must be approached with care. These models are subject to specific criteria, the most important of which is to clarify the intended purpose of a mathematical model for the spread of epidemic, that is, "a mathematical model for what purpose?" Mathematical models are indeed research tools, but they can also address critical public health questions about an epidemic, such as its scale, temporal evolution, or potential control strategies. To this end, an epidemic model must rely on realism oriented toward simplicity. This does not mean excluding various possible aspects of the epidemic, but rather incorporating the key components that best represent the spread of the epidemic [48, 50, 51].

## 1.4 Specificities of Mathematical Models in Epidemiology

Epidemiological modeling faces the challenge of not being unique, even though the goal remains the same: to alter the rate of spread of infection. This results in two impacts on the population: a direct real impact due to the epidemic (primarily contamination) and an indirect impact conveyed by the model, with a major issue being their corroboration, if it exists [46, 52]. As mentioned earlier, the hierarchy of considerations for establishing a mathematical model depends primarily on the epidemiological phenomenon being understood and established, which can be descriptive, qualitative, observational, and interpretable [47, 52].

At this stage, the quantitative aspect comes into play, in the mathematical sense comprising equations, initial conditions, functions to compute, and representations come into play. A mathematical model can only be established based on hypotheses closely tied to the epidemiological phenomenon. The development of a mathematical model in a mathematical discipline, epidemiology being no exception, requires a fundamental step in the modeling process. A mathematical model is not self-evident, as it must first answer the question, "Why model?" In other words, a mathematical model must address pre-defined objectives. Common sense suggests that by increasing the number of objectives, the model's reliability improves [43, 52].

## 1.5 Types of Mathematical Models in Epidemiology

### 1.5.1 Deterministic Models

The first proposals for epidemiological models date back to the early twentieth century, each influenced by the assumptions that predetermine them. Two fundamental models are proposed: the deterministic model and the stochastic model. The first of these models laid the foundation for what is now called "mathematical modeling in epidemiology".

The work of Hamer [53] established the basis for deterministic models in epidemiology [46]. The hypothesis underlying this model is as follows: the number of new cases reported for an infectious disease depends on the number of susceptible individuals, the number of already infected individuals, and a proportionality coefficient representing the rate of contagion. This coefficient includes the virulence of the infectious agent and, more importantly, its ability to spread, as well as the frequency of contact between healthy and infected individuals. Later, this simplified model proved insufficient to account for the complexity of an epidemiological phenomenon. Therefore, new hypotheses were added, making the model more complex and also more realistic. This led to the proposal of compartmental models or class-based epidemic models [52].

### 1.5.2 Compartmental Infectious Models

In epidemiology, the methodology for managing nonuniform flows of individuals who do not all meet the same classification criteria (such as healthy, potentially infected, recovering, etc.) has been a key concern. Therefore, it is natural to consider their distribution in epidemiological classes or compartments. Through their identification and arrangement, these compartments form the basic units for the mathematical modeling of an epidemic. It should be noted that these compartments are not isolated, but open and closely interconnected, as the concept of an epidemic would otherwise lose its meaning. An obvious analogy can be drawn with the dynamics of systems, a highly important topic in physics [43, 49, 52].

The population within a compartment is assumed to be homogeneous, which means that individuals in the compartment share the same status regarding infection and retain this status as long as they remain in the compartment. An individual who changes state (due to immunity or death) must move to another compartment. This results in inter-compartmental flows, leading to variations in the respective population sizes. This type of model is called deterministic.

The dynamics generated by the epidemic phenomenon are similar to those described in dynamic systems, which requires the definition of initial conditions that will determine the evolution of the system. The flows between compartments depend only on the population sizes of the compartments and the proportionality coefficients, such as the rates of contagion, recovery, etc. A deterministic model excludes probabilistic criteria. Therefore, the initial conditions set will always lead to the same system evolution. These are continuous-time deterministic systems. The variable "time  $t$ " is thus fundamental in the equations governing this category of systems. Compared to a time step  $\Delta t$ , the number of new cases is evaluated only within this time interval,

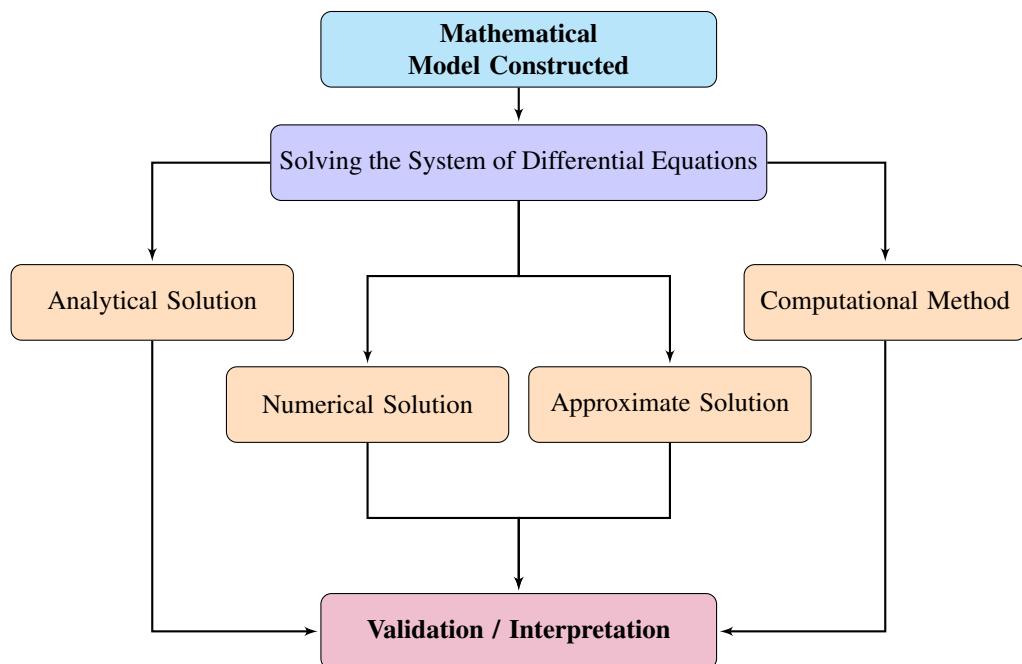
not the number of individuals recorded after  $\Delta t$ . The evolution is therefore continuous, but not instantaneous [43, 52, 53].

### 1.5.3 Formulation of a Model

In principle, the more complex a mathematical model is, the more representative it becomes of the phenomenon to be depicted. It is worth noting that such a model must meet objectives that do not unnecessarily delve into complexity. A mathematical model designed to understand the spread of a pandemic is merely a tool to help with its health management, without claiming to address all the questions raised by its propagation.

By dismissing the notion of an absolute standard model, we limit ourselves to considering the mathematical model as a reflection of assumptions or hypotheses. Throughout its development, the criteria for these assumptions will predetermine its reliability. It is essential that they are chosen carefully and, above all, objectively, as will be the case in this study. However, an established mathematical model, such as one for a pandemic, must be approached with humility and relativity, allowing for extrapolation or time-based projections to provide another perspective on the phenomenon of pandemic spread. The flip side of this lies in the complexity that such systems demand, with the challenging task of solving mathematical models typically based on systems of nonlinear differential equations. Determining an analytical solution is often impossible [53, 54].

The calculation of solutions can be performed using specific algorithms, such as the finite difference approximation method. For this study, we used the ordinary differential equation (ODE) solving library available in Python programming language. Specifically, we used the `solve_ivp` function from the `scipy` library, which offers the advantage of providing graphical solutions to nonlinear differential equation systems, which makes it perfectly suited for the interpretation phase. The diagram shown in Figure 1.6 is an example of a procedure used in the modeling process.



**Figure 1.6.** A typical flowchart in a modeling process.

## 1.6 Population Dynamics and Mathematical Formulation

The translation of population dynamics into mathematical language was initially met with skepticism, if not curiosity, as the descriptive approach was prevalent at the time. Applying a mathematical model to a biological phenomenon, for example, seemed to transgress the established methodologies of observation, experimentation, and interpretation that had been widely accepted until then. The assignment of a mathematical equation to a biological fact or to a public health issue was considered almost heretical. This apparent antagonism might suggest competition between approaches, implying a reversal of primacy and hierarchy. However, this is not the case. Instead, it is more accurate to speak of complementarity, as observations or descriptions in biology are materialized through posed questions, and mathematical models, rich in their concepts, contribute to the proposal of scientifically validated solutions [55].

Mathematical models aimed at studying population dynamics date back to the 17th century, and the first applications of differential calculus in epidemiology were attributed to Bernoulli [56]. The work of P. D. Enko laid the foundations for what could be called “mathematical epidemiology,” extended to infectious diseases (1873) [Wikipedia John Snow \(1847\)](#) by Thomas Jones Barker.

In terms of studying dynamics focused on population evolution, it is undoubtedly the mathematical model of Alfred Lotka (1925) [57] and Vito Volterra [58] (1926) that captures attention with the "predator-prey" model.

## 1.7 A Dynamic Model of Population Evolution

The simplest model to describe and understand the demographic evolution of a population, in terms of its growth or decline, is to relate these changes to a parameter that quantifies this evolution. This parameter is defined as the proportional size of the population, linked to its increase in terms of regeneration or its decrease in terms of species extinction. Another simplifying assumption would be to consider that a population evolves in an isolated system, that is, without external influence but regenerates internally. Mathematically, the evolution  $y$  over time of the population as a function of  $t$  follows the differential equation:

$$y'(t) = ky(t).$$

The factor  $k$  represents the rate of evolution of the species mentioned above. An elementary integration of this separable-variable ODE leads to:

$$y(t) = C \exp(kt).$$

This equation describes the dynamics of the population over time [49, 55, 59].

However, this overly simplistic mathematical model applies only to the early stages of population evolution. A more realistic model is described by the logistic function, which follows the differential equation.

$$y'(t) = ky(t) \left(1 - \frac{y(t)}{\mu}\right).$$

The new parameter  $\mu$  is called the carrying capacity parameter. We solve the logistic equation by rewriting it in the following form:

$$\frac{y'}{y \left(1 - \frac{y}{\mu}\right)} = k.$$

We consider the fraction  $\frac{1}{y(1 - \frac{y}{\mu})}$  and decompose it as follows:

$$\frac{1}{y\left(1 - \frac{y}{\mu}\right)} = \frac{A}{y} + \frac{B}{1 - \frac{y}{\mu}}.$$

After reducing to the same denominator, we proceed by identifying coefficients and obtain the following:

$$\begin{cases} A = 1 \\ B = \frac{1}{\mu}. \end{cases}$$

This allows us to write

$$\frac{1}{y\left(1 - \frac{y}{\mu}\right)} = \frac{1}{y} + \frac{1}{\mu\left(1 - \frac{y}{\mu}\right)} = \frac{1}{y} + \frac{1}{\mu - y}.$$

We can now integrate the logistic differential equation by writing:

$$\int \left( \frac{1}{y} + \frac{1}{\mu - y} \right) dy = k dt \implies \ln \left| \frac{y}{\mu - y} \right| = kt + c.$$

This also allows us to write

$$\frac{y}{\mu - y} = e^{kt+c} \implies y(t) = \frac{\mu e^{kt}}{1 + e^{kt+c}} = \frac{\mu e^{kt}}{1 + e^{kt} e^c}.$$

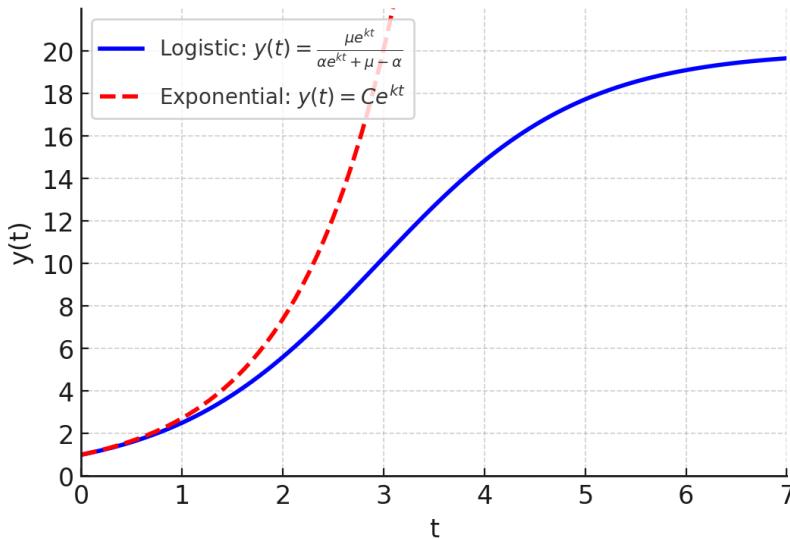
Taking into account the initial condition  $y(0) = \alpha$ , we have the following

$$\alpha = \frac{\mu}{1 + e^c} \implies e^c = \frac{\mu}{\alpha} - 1.$$

By substituting this value of  $e^c$  into the expression for the unknown function  $y(t)$ , we obtain:

$$y(t) = \frac{\mu e^{\kappa t}}{\alpha e^{\kappa t} + \mu - \alpha}.$$

The code PYTHON below solves both the differential equation leading to the exponential law of  $y(t)$  and the logistic differential equation, as well as the respective graphical representations of  $y(t)$  (1.7).



**Figure 1.7.** Exponential law and logistic function for population growth with environmental limits

## 1.8 Dynamic Model of a Predator-Prey System

Most often, elements of a population do not live in isolation, but multiple species can co-exist, with one species representing the prey and the other the predator. We then have two populations  $y_1$  and  $y_2$  evolving simultaneously. The rate of change of the species  $y_1$  is a linear function of  $y_2$ , and vice versa, leading to the following system of differential equations.

$$\begin{cases} y'_1 = \left(1 - \frac{y_2}{\mu_2}\right) y_1, \\ y'_2 = \left(1 - \frac{y_1}{\mu_1}\right) y_2. \end{cases}$$

The integration of the first equation yields.

$$y_1(t) = C_1 \exp \left[ \left(1 - \frac{y_2}{\mu_2}\right) t \right].$$

The integration of the second equation yields.

$$y_2(t) = C_2 \exp \left[ \left(1 - \frac{y_1}{\mu_1}\right) t \right].$$

The functions  $y_1(t)$  and  $y_2(t)$  represent the populations of prey and predators, respectively. Let us express the ratio of their derivatives as follows:

$$\frac{y'_1}{y'_2} = \frac{\left(1 - \frac{y_2}{\mu_2}\right) y_1}{\left(1 - \frac{y_1}{\mu_1}\right) y_2}.$$

Applying the method of separation of variables, we obtain the following.

$$\int_{y_0^1}^{y_1} \frac{d\alpha_1}{\left(1 - \frac{\alpha_2}{\mu_2}\right) \alpha_1} = \int_{y_0^2}^{y_2} \frac{d\alpha_2}{\left(1 - \frac{\alpha_1}{\mu_1}\right) \alpha_2}.$$

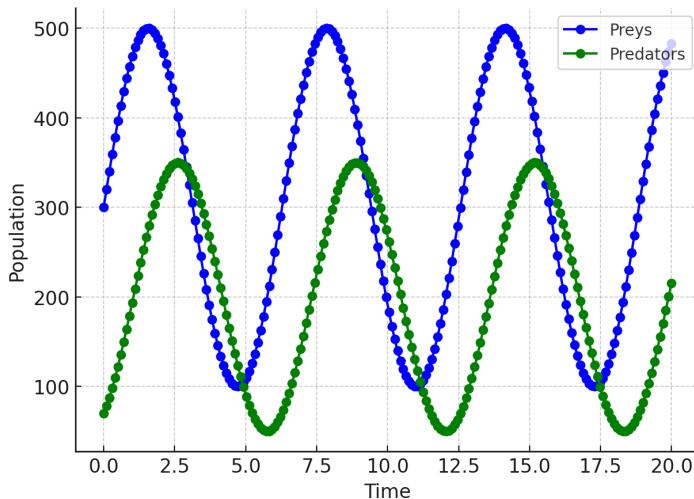
The calculation of the integrals leads to the following:

$$\mu_2 \log \left( \frac{\mu_2 - y_0^1}{\mu_2 - y_1} \right) = \mu_1 \log \left( \frac{\mu_1 - y_0^2}{\mu_1 - y_2} \right).$$

This can also be written as:

$$\left( \frac{\mu_2 - y_0^1}{\mu_2 - y_1} \right)^{\frac{\mu_2}{\mu_1}} = \frac{\mu_1 - y_0^2}{\mu_1 - y_2}.$$

Therefore, it is possible to track the dynamics of predator-prey populations based on their initial numbers. The following PYTHON code generates a graphical representation of the “prey” population and the “predator” population (Figure 1.8), obtained using Code A.5.



**Figure 1.8.** Predator-prey model evolution laws

## 1.9 Problematic of Mathematical Modeling of a Pandemic

Mathematical models dedicated to biology in general and epidemiology in particular are similar to phenomenological models, also called pattern models or "mechanistic" models [42, 44]. These models characterize evolutionary phenomena such as those observed during the spread of an epidemic. The concept of a model is inherently ambiguous as it refers to various interpretations that can define these models. However, they aspire to the same objectives: providing a rational and scientific explanation of the observed facts [44].

The mathematical modeling of a pandemic is unique in that it is based on both pattern and mechanistic approaches. The pattern aspect is related to the phenomenological nature of the pandemic, while the mechanistic aspect aims to understand the causes of the pandemic phenomenon. Therefore, it is important to note that a reliable mathematical model of a pandemic, such as COVID-19, cannot be developed without understanding the mechanisms of widespread contamination within a population.

The epidemiological phenomenon, in its simplest approach, begins with a primary infection during which the identified virus causes a health crisis within a healthy population by colonizing at least one host. This is the infection phase. The evolutionary phenomenon of the pandemic

is driven by a dynamic of virus multiplication or reproduction not only within the infected host but also through the “exportation” of the virus’s offspring to colonize other hosts: this is the contamination phase, which, when limited in space and time, is an epidemic. When generalized, it becomes a pandemic.

## 1.10 Models - Epidemic Curve and Plateau

During the expansion of a pandemic, a curve known as the "epidemic curve" or *epicurve* is regularly established to reflect the progression of the epidemic [42, 44]. Given the induced health concerns, this epi-curve is updated daily and reports the number of infected individuals, that is, the number of people who fall ill each day.

The epidemic curve is plotted in a reference frame where the  $x$  axis represents the dates of evaluation of the pandemic and the  $y$  axis represents the number of individuals infected with the virus on a specific date. The epidemic curve is regularly updated based on available data. More than just a graph, the epidemic curve contains relevant information on the outbreak of a pandemic. However, it is essential to extract, analyze, and interpret this information.

An epidemic curve can be seen as a juxtaposition of time series of daily infections with stages of unequal lengths. The study of an epidemic curve can be carried out through analysis windows and correlations.

In general, an epidemic curve reflects the dynamics and scale of the pandemic. Initially, this curve can only increase, in which case we can speak of a pandemic. Recurring questions about the pandemic are regularly and legitimately asked. When will the epidemic peak? Will the epidemic curve decline? Will it show a peak or plateau over time? The paradox in a pandemic situation is that the exact start date of the epidemic is unknown, and predicting the precise end of the epidemic is challenging.

In epidemiological statistics, the concepts of peak and plateau are often referenced. However, it is crucial to distinguish between these two notions. When an epidemic reaches its peak, the growth of the epidemic curve is rapid, and it is assumed that its decline will be equally rapid, marked by a point called the “peak.” In the case of an epidemic plateau, the decline is slower and a more or less extended flattening is observed before the curve levels off along the time axis. This suggests the end of the pandemic, far from its virulent phase. Flattening of the epidemic curve can be achieved through total or partial population lockdowns or by implementing barrier measures such as mask wear and social distancing.

## 1.11 Simplicity and Complexity of an Epidemiological Model

Epidemiological models are not immutable; they can be enhanced by introducing new compartments and adjusting them according to a specific epidemiological context. This leads to a multitude of combinations of different compartments, resulting in an increased model complexity. However, the goal remains to analyze and interpret the evolution of the same epidemic. Figure 1.9 illustrates a potential scenario of the various phases that can govern an epidemiological situation. The idea is to divide the global model into interconnected subcompartments that can address real-world situations. However, this "architecture" is incomplete, as it does not account for all possible scenarios, such as demographic factors, incubation periods, loss of immunity, or vaccination phases, all of which deserve to be included in the model [42, 44].  $S$ :Susceptible,  $E$ :Exposed,  $I_p$ :Infected presymptomatic,  $A$ :Asymptomatic,  $I_m$ :Infected with mild symptoms,  $I_s$ :Infected with severe symptoms,  $H$ :Hospitalized, Rea:Admitted to intensive care,  $R$ :Recovered, and  $D$ :Deceased;  $\beta$ :Disease transmission rate,  $\sigma$ :Incubation rate ( $\frac{1}{\text{incubation period}}$ ),  $\gamma$ :Recovery rate ( $\frac{1}{\text{duration of the illness}}$ )

UATION OF SCIENTIFIC AND TECHNOLOGICAL CHOICES.

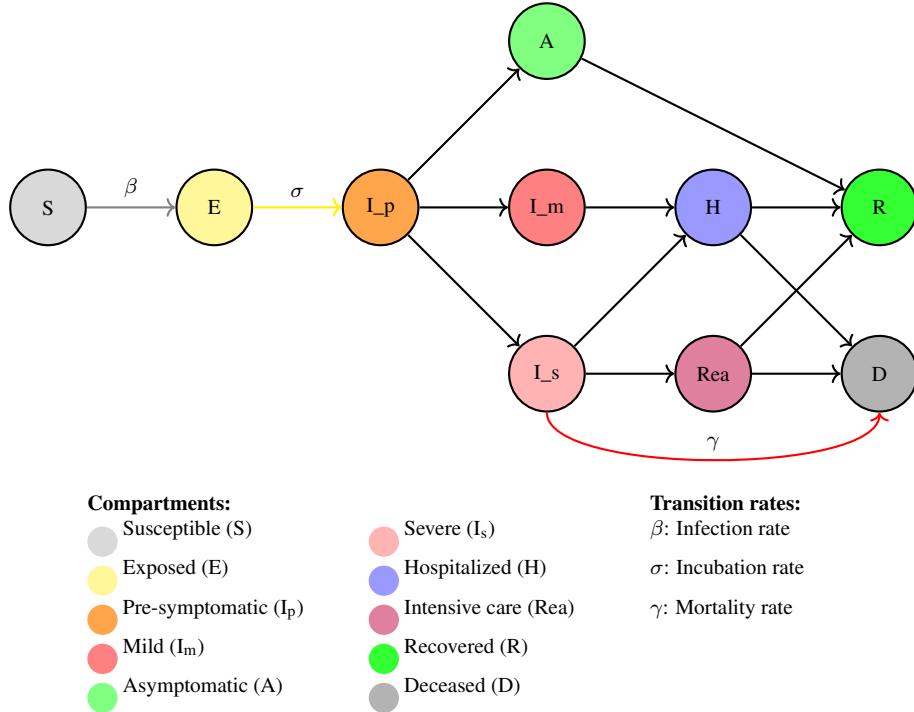


Figure 1.9. Epidemiological model with compartment definitions

As part of another complexification of the SEIR model adapted to COVID-19, we describe below the model proposed by [50, 51], based on an alternative compartmentalization of the SEIR model by adding compartments relevant to this pandemic. The flows governing the "exchanges" of individuals between the different compartments are quantified by parameters set a priori. This model includes the basic compartments of epidemiological modeling along with other compartments specific to the model proposed by the authors.

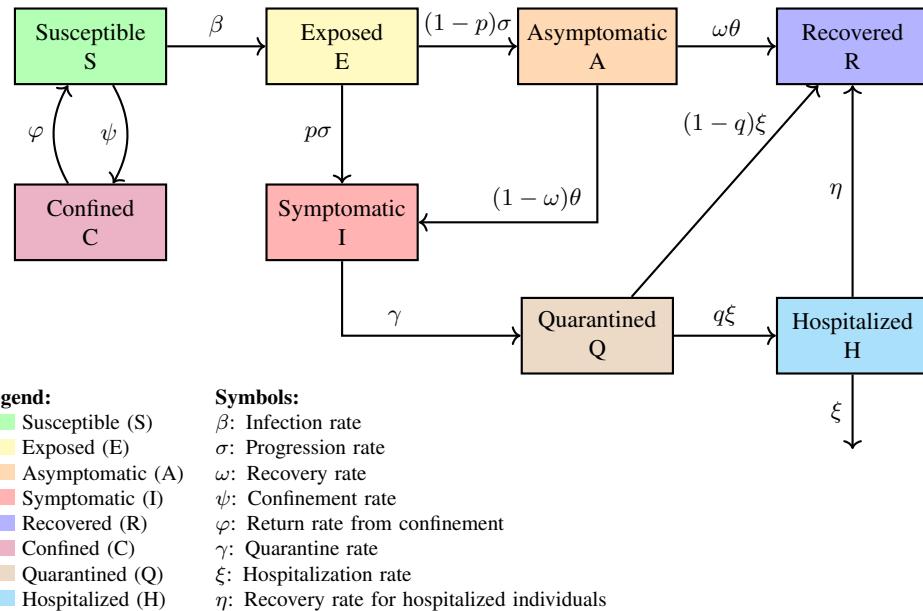
A susceptible population  $S$  exposed to contact with infected individuals will see its number reduced by a fraction or infection rate  $\beta$ , also called the "force of infection." This fraction of individuals loses its susceptible status and enters the exposed compartment ( $E$ ), predisposed to contamination. A fraction  $p$  of these individuals becomes infected by contact with infectious individuals at a contamination rate  $\sigma$  and enters the infected compartment ( $I$ ). The remaining fraction  $(1 - p)$ , relatively healthy, forms the class of asymptomatic individuals ( $A$ ). The fate of asymptomatic individuals can take two forms:

- A fraction  $w$  of asymptomatic individuals can be declared noninfectious at a rate  $\theta$ , in which case they join the recovered compartment ( $R$ ).
- The remaining fraction  $(1 - w)$  will have been contaminated and will join the symptomatic compartment ( $I$ ).

Among the health measures in the presence of an epidemic is the management of infected individuals, i.e., those in the compartment  $I$ . Among the possible measures is quarantine at a rate  $\gamma$ , creating a "quarantine" compartment ( $Q$ ). A fraction  $q$  of quarantined individuals may be directed to a hospital facility at a rate  $\xi$ , hence the compartment ( $H$ ). The remaining fraction of individuals, i.e.,  $(1 - q)\xi$ , will join the recovered compartment  $R$ , which, to remember, also includes the deceased. The occupants of compartment  $H$  can, in turn, join the recovered

compartment  $R$  at a rate  $\eta$ .

The model described above incorporates a confinement operation by creating a compartment ( $C$ ) adjacent to the susceptible compartment ( $S$ ). Confinement involves removing a subset of individuals from the compartment ( $S$ ) at a rate  $\psi$  and temporarily isolating them from the epidemic dynamics. Deconfinement or reverse confinement involves the extraction of individuals from the compartment ( $C$ ) at a rate  $\varphi$  and the reintroduction of them into the susceptible compartment ( $S$ ) with their status restored. See Figure 1.10.



**Figure 1.10. Complex SEASIR Model: Infection, Recovery & Containment**

The authors of the model have proposed the following mathematical model.

$$\left\{ \begin{array}{l} \frac{dS}{dt} = -\beta S - \psi S + \varphi C, \\ \frac{dC}{dt} = \psi S - \varphi C, \\ \frac{dE}{dt} = \beta S - \sigma E, \\ \frac{dA}{dt} = (1-p)\sigma - \theta A, \\ \frac{dI}{dt} = p\sigma E - \gamma I + (1-\omega)\theta A, \\ \frac{dQ}{dt} = \gamma I - \varsigma Q, \\ \frac{dH}{dt} = q\zeta Q - \eta H, \\ \frac{dR}{dt} = \omega\theta A + (1-q)\zeta Q + \eta H. \end{array} \right. \quad (1.2)$$

How, for example, can one quantify in an equation the wearing of masks or social distancing? Epidemiological models are thus subject to multiple sources of uncertainty. This complexification, through the introduction of a large number of parameters such as interactions between individuals or probabilities of contamination, is highlighted in the equations. These parameters can be difficult to set or estimate.

### 1.11.1 Another Example: The EHESP Model

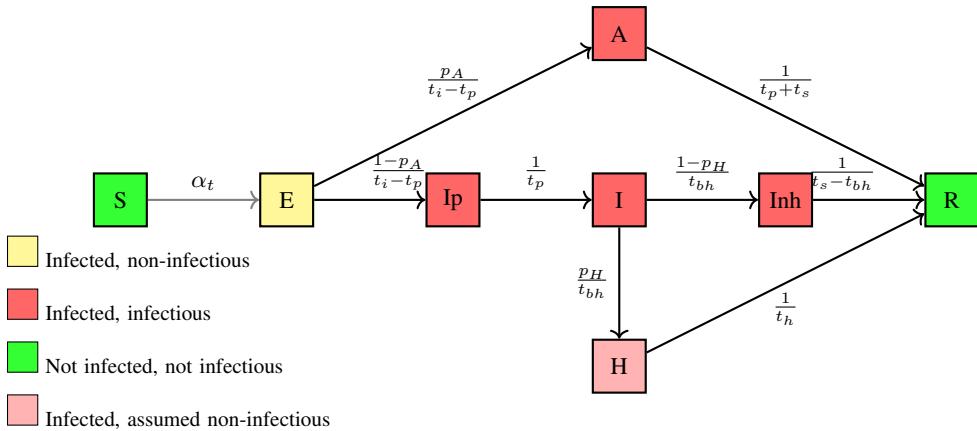


Figure 1.11. The EHESP Model

At the beginning of the pandemic, the majority of the population is in the  $S$  (susceptible) compartment. At a rate  $\alpha_t$  (infection rate), which depends on:

- The number of infectious individuals at time  $t$ ,
- The number of daily contacts,
- The number of immunized individuals,

a portion of the population is transferred to the  $E$  (exposed) compartment.

This compartment  $E$  is assumed to be non-contagious, which is an approximation: the incubation phase of the original strain actually consists of two phases:

- A non-infectious phase (approximately 3 days),
- An infectious phase (approximately 2 days).

In this model, individuals are assumed to remain in this compartment for an average time  $t_i$  (incubation time). With probability  $p(A)$  (probability of becoming asymptomatic), these individuals become asymptomatic and are transferred to the  $A$  compartment. Otherwise, they are transferred to the  $I$  (infectious) compartment.

In the absence of hospitalization, the individuals remain infectious for an average time  $t_s$  (infectious period). However, with probability  $p(H)$  (probability of hospitalization), people can be hospitalized (compartment  $H$ ), which occurs after an average time  $t_{bh}$  (time before hospitalization). The affected individuals remain in the hospital for an average time  $t_h$  (hospitalization time) before being transferred to the  $R$  (recovered) compartment, after their recovery or death. The period of immunity after infection was unknown at the beginning of the epidemic, so it was systematically assumed that reinfection was impossible.

Due to the precautions taken by healthcare workers and the low number of contacts in the hospital, individuals in the  $H$  compartment are assumed to be non-contagious. This compartment  $H$  has a medical reality, but is also added for practical reasons. In fact, the data reported by hospitals is more reliable and well-documented. They provide data of higher quality than the number of infectious individuals, which is biased by the number of daily tests performed. For this reason, many epidemiological models have chosen to base their analysis on hospitalization data<sup>1</sup>. The proposed model adds two compartments:

<sup>1</sup>This was not the case for the original SEIRAH model, but it has been modified.

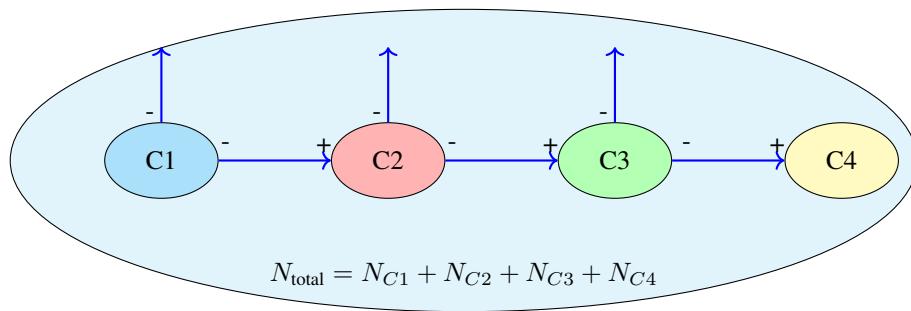
- Divides the incubation phase into two compartments:
  - ▶ A non-infectious incubation phase (compartment  $E$ ), with an average duration of  $t_i - t_p$ ,
  - ▶ An infectious incubation phase  $I_p$  with an average duration of  $t_p$  (pre-symptomatic infectious period).
- It also adds a compartment  $I_{nh}$  (infectious non-hospitalized), which groups individuals who are still infectious but are not known to be hospitalized.

Intuitively, one can think of this model as being stratified by age.

## 1.12 Introduction to SIR Models

It has been mentioned previously that the spread of an infectious agent within a population resembles a dynamic phenomenon, implying the use of a set of primarily mathematical tools in its theoretical approach and analysis [43, 52]. From an epidemiological perspective, two main classes of individuals co-exist during an epidemic: the class of healthy individuals that do not host the infectious agent (at least temporarily) and the class of sick or infected individuals. The numbers in these two classes evolve over time, influenced by the presence and frequency of contacts between these two categories of individuals. To remain in the spirit of the dynamics of physical systems, such an epidemiological phenomenon can be approached using a mathematical model based on differential equations, the solution of which will account for the evolution of the epidemic and, to some extent, contribute to the management of the health crisis. The population sizes mentioned above, which vary during an epidemic, will be represented by a function of the variable  $t$  for each class of individuals involved in the pandemic [42, 44, 46].

The first relatively simple models were proposed by Kermack and McKendrick [8]. However, their efficiency has been verified in the case of several epidemics whose evolution closely follows that initiated by these models. The basic principle is relatively simple: decompose a global epidemiological phenomenon affecting a population into epidemiological classes or interconnected compartments [60–62]. The number of compartments to consider depends on the classes of individuals who experience epidemiological effects Figure 1.12.



**Figure 1.12.** Epidemiological compartmentalization structure

Beyond the interconnection between different compartments, these compartments can also be influenced by external factors (demographic input, deaths, etc.). However, one of the assumptions of the compartmental model (an adjective accepted in epidemiology) is the stability of the total population size:

$$N_{\text{population}} = N_{C1} + N_{C2} + N_{C3} + N_{C4}.$$

This model is used to describe the temporal evolution of a disease within a population. The

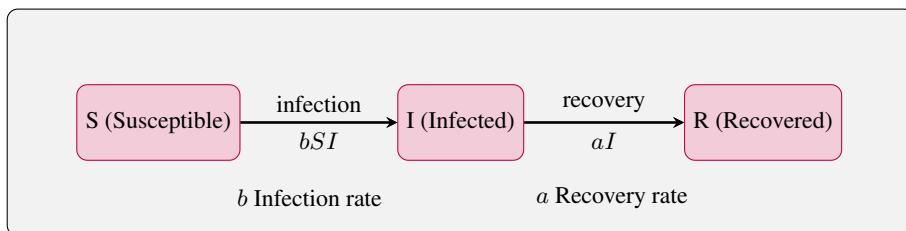
Kermack and McKendrick model is based on simple assumptions about the flow of individuals from one compartment to another according to the indicated epidemiological laws or rules [8]. Theoretically, the number of compartments is not fixed in advance but depends on the nature of the study to be conducted. The present study will be based on three and then four compartments. The mechanisms of infection transmission are now well understood: they respond to a dynamic, in the physical sense, of propagation and the mode of contamination. Generally, epidemics are transmitted through infectious agents. An epidemic, which operates on a very short time scale, is defined as a sudden manifestation of a disease that affects a substantial part of a population in a given region, followed by its gradual extinction [52].

The most cited and studied basic model in the literature is the **SIR** model, which refers to the distribution of the population into three compartments. Susceptible, Infected and Recovered or Removed (immunized or deceased) [46]. The SIR model can extend from the most elementary model that integrates a minimal number of parameters and a fairly simple propagation scenario to the most elaborate schemes. Other classes that fit the logic of epidemic propagation can be incorporated into the S, I, and R compartments. The components S, I, and R then become time-dependent variables:

$$\begin{cases} S & \rightarrow S(t), \\ I & \rightarrow I(t), \\ R & \rightarrow R(t). \end{cases}$$

- **Susceptible (S):** Healthy individuals who are susceptible to infection and do not have immunity against the infectious agent.
- **Infected (I):** Individuals who are infected and can transmit the disease upon contact with susceptible individuals.
- **Recovered (R):** Individuals who have recovered from the disease (or died) and are assumed to have acquired permanent immunity.

These compartments are illustrated in 1.13.



**Figure 1.13. Basic diagram of the SIR model**

## 1.13 Heuristic Approach to the Simplified SIR Model

### 1.13.1 Model Construction

The main objective is to develop the differential equation system that models the epidemiological evolution relative to the figure above by integrating the epidemiological parameters  $b$  and  $a$ . The functions of Susceptibility, Infection, and Recovery or Death depend on the time variable  $t$  (in days) and will be denoted respectively as  $S(t)$ ,  $I(t)$ , and  $R(t)$ . We denote  $\Delta t$  as the increment in time (also in days). The epidemiological parameters are two in number:

- The probability that a susceptible individual becomes infected, denoted  $b$  (infection rate).
- The probability that an infected individual recovers (removed) or dies, denoted  $a$  [46, 52].

The time increment is performed according to the classical scheme:

$$\begin{cases} t & \rightarrow t + \Delta t, \\ S(t) & \rightarrow S(t + \Delta t), \\ I(t) & \rightarrow I(t + \Delta t), \\ R(t) & \rightarrow R(t + \Delta t). \end{cases}$$

It is necessary to relate the epidemiological parameters to time. Here, we opt for the linear variation of these parameters (the simplest case). The following model is then adopted:

$b \rightarrow b\Delta t$  (probability that a susceptible individual becomes infected during  $\Delta t$ ),

$a \rightarrow a\Delta t$  (probability that an infected individual recovers or dies during  $\Delta t$ ).

### 1.13.2 Modeling Epidemics Under Population Conservation

The evolution equations are given by:

$$\begin{cases} S(t + \Delta t) & = S(t) - (b\Delta t)S(t)I(t), \\ I(t + \Delta t) & = I(t) + (b\Delta t)S(t)I(t) - (a\Delta t)I(t), \\ R(t + \Delta t) & = R(t) + (a\Delta t)I(t). \end{cases}$$

This can also be written in difference-quotient form:

$$\begin{cases} \frac{S(t+\Delta t)-S(t)}{\Delta t} & = -bS(t)I(t), \\ \frac{I(t+\Delta t)-I(t)}{\Delta t} & = bS(t)I(t) - aI(t), \\ \frac{R(t+\Delta t)-R(t)}{\Delta t} & = aI(t). \end{cases}$$

Taking the limit as  $\Delta t \rightarrow 0$ , we obtain the system of differential equations that describes the SIR model for epidemic propagation:

$$\begin{cases} \frac{dS}{dt} & = -bS(t)I(t), \\ \frac{dI}{dt} & = bS(t)I(t) - aI(t), \\ \frac{dR}{dt} & = aI(t). \end{cases}$$

Often, the total population size  $N$  involved in the SIR model is introduced. In this case, the system becomes:

$$\begin{cases} \frac{dS}{dt} & = -bNS(t)I(t) \Rightarrow \frac{1}{N} \frac{dS}{dt} = -bSI, \\ \frac{dI}{dt} & = bNS(t)I(t) - aNI(t) \Rightarrow \frac{1}{N} \frac{dI}{dt} = bSI - aI, \\ \frac{dR}{dt} & = aNI(t) \Rightarrow \frac{1}{N} \frac{dR}{dt} = aI. \end{cases}$$

It is therefore possible to admit the first system of differential equations as representing the mathematical model, except in cases where one wishes to include the population size in the calculations. It should also be noted that if the number  $N$  is very large, normalization of the population size is carried out by dividing it by the total population, resulting in a fractional population size in the form of a decimal number.

By adding the three differential equations of the model, we obtain:

$$\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \frac{d}{dt}(S + I + R) = 0.$$

The sum  $(S + I + R)$  is therefore a constant that does not depend on the variable  $t$ . It precisely represents the total population size in all compartments:  $S + I + R = N$ .

## 1.14 Theoretical Foundations and Simulations of SIR Models

It should be recalled that the SIR model is a simplified version among epidemiological models. Nevertheless, this model continues to be the subject of numerous studies, primarily in the mathematical domain. This simplicity originates from the a priori hypotheses imposed on the model, the most important of which are listed below.

- The population size remains constant across all compartments and during the period of the epidemic, which means that there is no update on the population size.
- Demographic variations (birth and natural death) are not included in the basic SIR model. However, more complex models are developed.
- Recovered individuals (cured or deceased) acquire permanent immunity and cannot be reinfected. These hypotheses may seem restrictive, but within certain limits, they are satisfied.

We have the following property:

$$\begin{cases} S(t) \geq 0, \\ I(t) \geq 0, \end{cases} \quad \forall t \geq 0.$$

**Proof.** Suppose that for  $t' \geq 0$ ,  $S(t') < 0$ . In the interval  $[0, t']$ , we consider  $t_1$  so that  $S(t_1) < 0$ . Knowing that  $S(0) \geq 0$  and that the functions  $S(t)$  and  $I(t)$  are continuous and differentiable, particularly  $S(0)S(t') < 0$ , the conditions of the intermediate value theorem are satisfied. Applying this theorem, we can write:

$$\exists \tau \in [0, t'] \quad \text{such that} \quad S(\tau) = 0.$$

From the differential equation:

$$\frac{dS}{dt} = -bIS \implies \frac{dS}{S} = -bIdt.$$

Integration of this separable differential equation leads to:

$$S(t) = S(\tau) \exp\left(-b \int_{\tau}^t I(x)dx\right) \quad \text{with} \quad t > \tau.$$

Since  $S(\tau) = 0$ , then  $\forall t, S(t) = 0$ , which is not compatible with the model; therefore, for all  $t, S(t) > 0$ .

The model is characterized by its simplicity and is not predictive. It is oriented towards the graphical aspect of solutions  $S(t)$ ,  $I(t)$ , and  $R(t)$  relative to the same reference frame. The modeling requires fixing the parameters infection rate  $b$  and remission rate  $a$ . It is also necessary to set the initial conditions at  $t = 0$  (beginning of the epidemic) for the three functions, namely  $S(0)$ ,  $I(0)$ , and  $R(0) = 0$ . The algorithmic procedure is given in Figure 1.14.

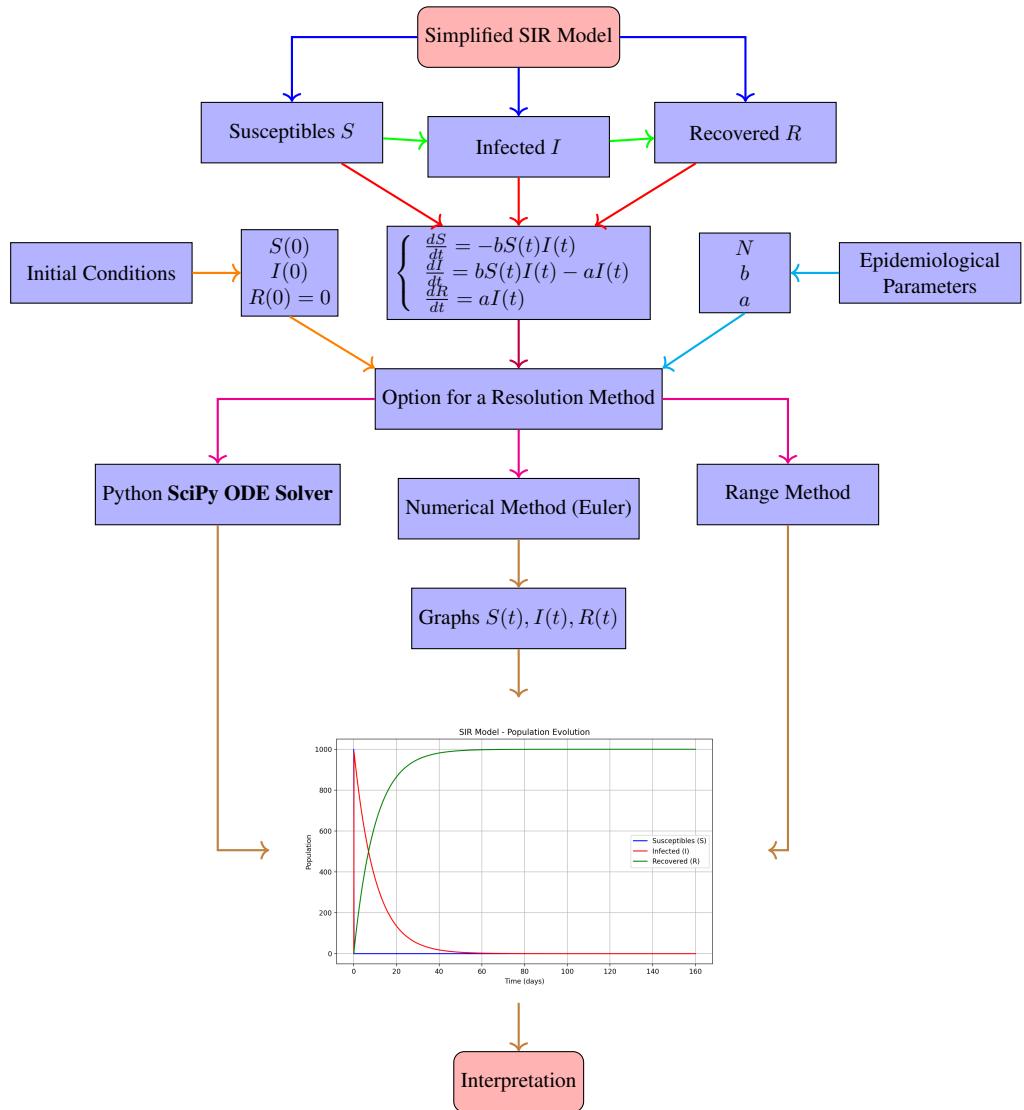


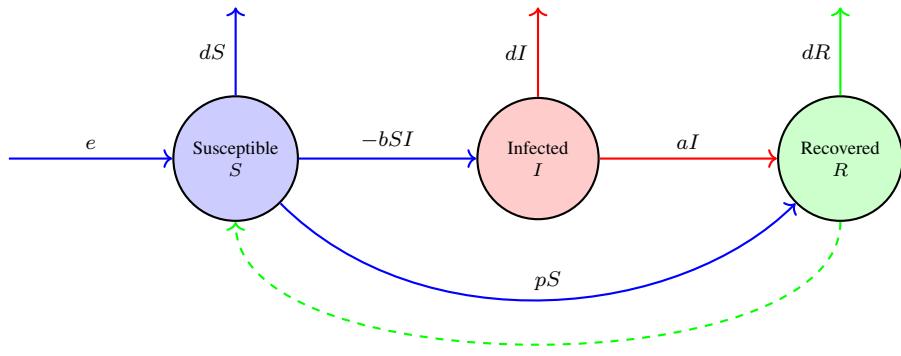
Figure 1.14. Flowchart for solving the SIR mathematical model

**Remark 1.1.** It is clear that the choice of epidemiological parameters incorporated into the mathematical model will influence the shape and spatial positioning of the  $S$ ,  $I$ ,  $R$  epidemic curves.

## 1.15 Generalized SIR Model

### 1.15.1 Schematic Design and Mathematical Modeling

The model presented above is one of the simplest forms of the SIR model. It evolves in isolation, which means that it has no exchanges with its environment. More elaborate and perhaps slightly more complex models are proposed in mathematical modeling for the spread of an epidemic. The model in Figure 1.15 is an example that may take other, more complex forms.



**Figure 1.15.** A Generalized SIR Model

This model incorporates three components compared to the simplified SIR system:

- A **feeding component** that supplies the compartment  $I$  with individuals outside the system, which can be of demographic origin, thus with a rate denoted as  $b$ .
- A **death component** with a rate denoted as  $d$  that affects the three compartments.
- A component related to vaccination, with a ratio  $p$ , allowing an individual from compartment  $I$  to be immunized against the disease and directly join the compartment  $R$ .
- An input component denoted by  $e$ , representing a constant influx of healthy individuals into the  $S$  compartment, for instance through births or immigration.

The SIR model mentioned describes a more global model compared to the one generally described in the literature. It is represented by the following system of differential equations:

$$\begin{cases} \frac{dS}{dt} &= e - dS - bSI + pS, \\ \frac{dI}{dt} &= bSI - (d + a)I, \\ \frac{dR}{dt} &= aI - dR + pS. \end{cases}$$

The basic reproduction number  $R_0$  is calculated as follows:

$$R_0 = \frac{eb}{(d+a)(d+p)}.$$

### 1.15.2 Stability Analysis and Equilibrium Points in Generalized SIR Systems

The equilibrium point is obtained by solving the following homogeneous system of equations:

$$\begin{aligned} \frac{dS}{dt} &= 0 \Rightarrow e - bSI - dS + pS = 0, \\ \frac{dI}{dt} &= 0 \Rightarrow bSI - (d + a)I = 0, \\ \frac{dR}{dt} &= 0 \Rightarrow aI - dR + pS = 0. \end{aligned}$$

By setting  $I = 0$ , the solutions of the system provide the equilibrium point  $E$ :

$$E = \left( \frac{e}{d+p}, 0, \frac{pe}{d(d+p)} \right).$$

## 1.15. GENERALIZED SIR MODEL

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To study the stability of the SIR system at the equilibrium point, consider the following system of equations:

$$\begin{cases} E_1 &= e - dS - bSI - pS, \\ E_2 &= bSI - (d + a)I, \\ E_3 &= aI - dR + pS. \end{cases}$$

We construct the Jacobian matrix  $J$  of the vector  $(E_1, E_2, E_3)$ :

$$J = \begin{pmatrix} \frac{\partial E_1}{\partial S} & \frac{\partial E_1}{\partial I} & \frac{\partial E_1}{\partial R} \\ \frac{\partial E_2}{\partial S} & \frac{\partial E_2}{\partial I} & \frac{\partial E_2}{\partial R} \\ \frac{\partial E_3}{\partial S} & \frac{\partial E_3}{\partial I} & \frac{\partial E_3}{\partial R} \end{pmatrix}.$$

The calculation of the partial derivatives provides the following:

$$J = \begin{pmatrix} -d - bI + p & -bS & 0 \\ bI & bS - (d + a) & 0 \\ p & a & -d \end{pmatrix}.$$

Evaluating the Jacobian at the equilibrium point  $E$ :

$$E = \begin{cases} S &= \frac{e}{d+p}, \\ I &= 0, \\ R &= \frac{pe}{d(d+p)}, \end{cases}$$

gives

$$J_E = \begin{pmatrix} -d - p & -\frac{be}{d+p} & 0 \\ 0 & \frac{be}{d+p} - (d + a) & 0 \\ p & a & -d \end{pmatrix}.$$

The eigenvalues of the matrix  $J_E$  are:

$$\begin{cases} \alpha_1 &= -d - p, \\ \alpha_2 &= \frac{be}{d+p} - (d + a) = (d + a)(R_0 - 1), \\ \alpha_3 &= -d. \end{cases}$$

For asymptotic stability, all eigenvalues must be negative. This leads to two important cases:

- |   |  |
|---|--|
| <ul style="list-style-type: none"> <li>■ When <math>R_0 &lt; 1</math>:           <ul style="list-style-type: none"> <li>► All eigenvalues are negative.</li> <li>► The system is asymptotically stable.</li> <li>► No epidemic occurs.</li> </ul> </li> </ul> | <ul style="list-style-type: none"> <li>■ When <math>R_0 &gt; 1</math>:           <ul style="list-style-type: none"> <li>► The eigenvalue <math>\alpha_2</math> becomes positive.</li> <li>► The system becomes unstable.</li> <li>► An epidemic occurs.</li> </ul> </li> </ul> |
|---|--|

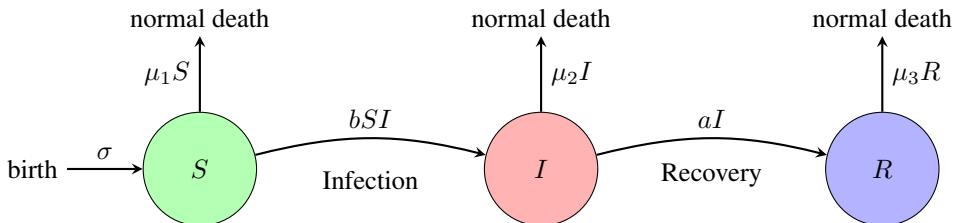
The basic reproduction number  $R_0$  thus serves as an epidemic threshold, determining whether the disease will die out ( $R_0 < 1$ ) or spread ( $R_0 > 1$ ).

## 1.16 SIR Model Including Demographics

The SIR model presented so far is a simplified version that does not account for all the circumstances surrounding the epidemiological phenomenon. In reality, this model is only applicable for pandemics that are limited in time. For longer periods or a succession of epidemics, it is necessary to complicate the model by including additional scenarios and epidemiological parameters. Certain conditions must be specified for this type of model:

- It is assumed that there is no vertical transmission of the disease, for example in a hereditary form. A newborn is considered susceptible and thus joins the **S** compartment.
- It is also assumed that birth-and-death rates balance each other.

In our case, ambient demography is included through two parameters: a parameter  $\sigma$  defining the birth rate or the immigration rate of healthy individuals in the **S** compartment and a series of three parameters  $\mu_1$ ,  $\mu_2$ , and  $\mu_3$  defining the mortality rates relative to each compartment. The corresponding SIR model is represented by the following figure (see Figure 1.16).



**Figure 1.16. SIR Model Dynamics**

The mathematical model corresponding to this is as follows.

$$\begin{cases} \frac{dS}{dt} = (\mu_1 S + \mu_2 I + \mu_3 R) - bSI - \mu_1 S, \\ \frac{dI}{dt} = bSI - aI - \mu_2 I, \\ \frac{dR}{dt} = aI - \mu_3 R. \end{cases}$$

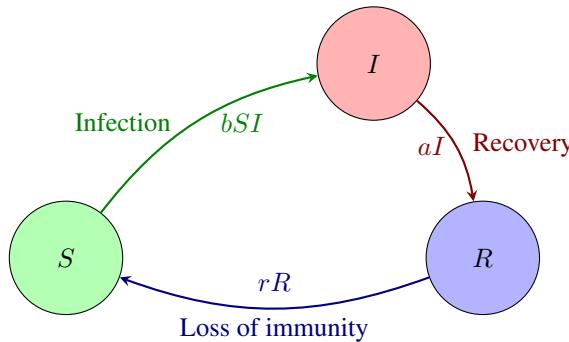
## 1.17 Immunity Loss Dynamics in the SIRS Model

### 1.17.1 Recurrent Epidemics Without Birth or Death

The SIRS model is another version of the SIR model, but removes the assumption of permanent immunity for individuals in the recovered compartment **R**. A proportion, denoted as  $r$ , may lose this immunity and thus reenter the susceptible compartment **S**. This will "feed" the population of the **S** compartment, leading to oscillations in the **S** and **R** compartments (see Figure 1.17). It is possible to add a demographic component to the SIRS model shown in the figure above, affecting susceptible and infected compartments through birth-and-death rates. For example, in the case of an SIRS mathematical model without mortality, the model can be represented by the following system of differential equations.

$$\begin{cases} \frac{dS}{dt} = -bIS + rR, \\ \frac{dI}{dt} = bIS - aI, \\ \frac{dR}{dt} = aI - rR. \end{cases}$$

The parameter  $r$  included in this mathematical model represents the rate of loss of immunity and the average immunity period. Note that if  $r \rightarrow 0$ , the SIRS model tends to transform into a classic SIR model.



**Figure 1.17.** Diagram of an SIRS Model (Loss of Immunity)

## 1.18 SI Model

The SI model is a rudimentary epidemiological model with limited impact on mathematical modeling of epidemic spread. It is based on simple logic: the only observable event is the infection of a susceptible individual. Let  $S(t)$  and  $I(t)$  be the numbers of susceptible and infected individuals, respectively, as functions of time  $t$ . At any given time, the total population is the following:

$$N(t) = S(t) + I(t).$$

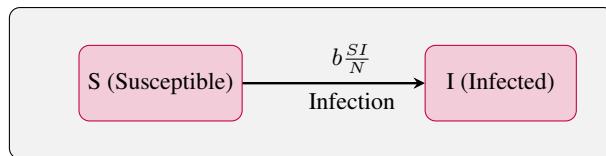
The assumptions associated with the SI model are those stated in the standard SIR model. Establishing a relationship between susceptibility and infection is generally not straightforward. Some authors have adopted the so-called mass incidence function, denoted as  $f(S, I)$ , such that:

$$f(S, I) = bSI \quad \text{where } b : \text{infection rate.}$$

In the presence of a large population, it is preferable to normalize the right-hand side of the expression  $f(S, I)$  by writing:

$$f(S, I) = b \frac{SI}{N}.$$

This form of writing implies that an infected individual can only contaminate a proportion of susceptible individuals. This gives the first diagram of the following model:

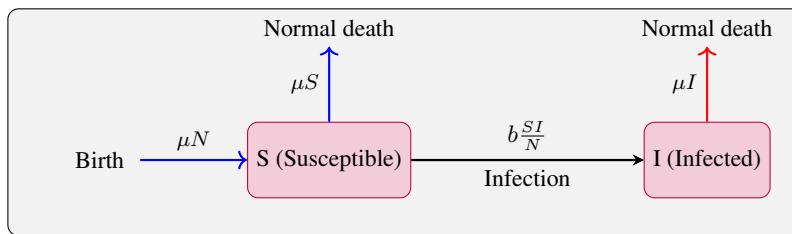


**Figure 1.18.** SI Model Dynamics

To obtain the system of differential equations that mathematically represents the model  $SI$ , we reason based on the flow of individuals entering or leaving each compartment  $S$  and  $I$ :

1. For the susceptible compartment  $S$ , there can be three types of flows:
  - (a) An incoming flow that can represent an external "input" of individuals, for example, from birth, in a proportionality ratio  $\mu$  relative to the total population  $N$ . This incoming flow is therefore  $+\mu N$ .

- (b) Regarding outgoing flows, there are two types:
- An outgoing flow due to death in a proportionality ratio  $d$  relative to the total population. We have kept the same coefficient  $\mu$  to simplify the study and ensure a demographically stable model. This first outgoing flow from the compartment  $S$  is of the form  $-\mu S$ .
  - The flow related to contaminations leaves compartment  $S$  proportionally to the susceptible population  $S$  and the infected population  $I$  with an infection or contamination rate  $b$ . This flow is thus quantified by the product  $(-b \frac{IS}{N})$ .
2. The flow following the infected compartment  $I$  is decomposed as follows:
- A flow representing mortality with the expression  $(-\mu I)$ .
  - The flow in the compartment  $I$  has the same absolute value as the one leaving the compartment  $S$ , that is, with an expression of the form  $(+b \frac{IS}{N})$ .



**Figure 1.19.** SI Model with Demography

The variations over time of the populations in compartments  $S$  and  $I$  are given, respectively, by the derivatives  $\frac{dS}{dt}$  and  $\frac{dI}{dt}$  such that:

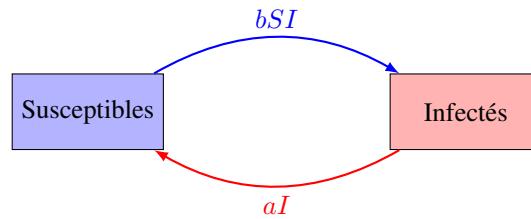
$$\begin{cases} \frac{dS}{dt} = \mu N - \mu S - b \frac{IS}{N}, \\ \frac{dI}{dt} = -\mu I + b \frac{IS}{N}. \end{cases}$$

Often, in the presence of a large population ( $N$  sufficiently large), it is preferable to perform calculations by normalization and replace  $\frac{S}{N}$  by  $S$ :

$$\begin{cases} \frac{dS}{dt} = \mu N - \mu S - bIS, \\ \frac{dI}{dt} = -\mu I + bIS. \end{cases}$$

## 1.19 Modeling Disease Dynamics with the SIS Framework

We aim to relativize the assumed permanent immunity of individuals who reside in the  $R$  compartment by asserting that they can become "contagious" again. We start from the scenario where the recovered individuals (compartment  $R$ ) reenter directly into the susceptible compartment  $S$ . They will then enjoy the status of susceptibles, which means that they can become infected or infectious again. This is in a sense a "relapse" due to the absence of immunity. More simply, one could say "you get sick, you recover, but you do not benefit from immunity". The most frequently cited case is the common cold. The model thus obtained is called the "SIS model", schematized as follows:



**Figure 1.20.** Diagram of the SIS Model

The SIS model is described by the following system of differential equations:

$$\begin{cases} \frac{dS}{dt} = -bSI + aI, \\ \frac{dI}{dt} = bSI - aI. \end{cases}$$

The assumption of conservation of the total population  $N$  during the epidemic leads to the following:

$$N = S + I \implies S = N - I.$$

The system of differential equations then becomes:

$$\begin{cases} \frac{dS}{dt} = bI(N - I) + aI, \\ \frac{dI}{dt} = bI(N - I) - aI. \end{cases}$$

We can observe that  $\frac{dS}{dt} = -\frac{dI}{dt}$ . Therefore, it is sufficient to consider only one of the differential equations, and in our case it will be the one corresponding to  $\frac{dI}{dt}$ . We rewrite the derivative as follows.

$$\frac{dI}{dt} = bNI - bI^2 - aI = (bN - a)I - bI^2.$$

We proceed to factor out the common factor as follows:

$$I'(t) = (bN - a)I \left(1 - \frac{b}{bN - a}I\right),$$

or equivalently,

$$I'(t) = (bN - a)I \left(1 - \frac{I}{\frac{bN-a}{b}}\right),$$

$$I'(t) = (bN - a)I \left(1 - \frac{I}{N - \frac{a}{b}}\right).$$

Now we introduce:

$$r = bN - a \quad \text{and} \quad k = N - \frac{a}{b}.$$

We obtain

$$I'(t) = rI \left(1 - \frac{I}{k}\right) = \frac{r}{k}I(k - I).$$

The expression in the second member defines what is commonly referred to as the *logistic function*.

## 1.20 Hidden Depths of SIR/SEIR Models

By designing models dedicated to epidemiological phenomena, a more precise description of the population has been sought. However, the SIR model, despite its simplicity, has nonetheless paved the way for more refined models that better represent epidemic contexts. This involves first the addition of supplementary compartments in accordance with the dynamics of the epidemic. In other words, increasing the complexity of the SIR model. However, there are three compartments that are fundamental to all such models:

- the susceptible population compartment  $S$ ,
- the infected or infectious population compartment  $I$ ,
- and the recovered (removed) compartment  $R$ .

The complexity arises first from the addition of new compartments and then from the possible connections between them. The final objective is a more precise description of the population with regard to its infection and recovery characteristics. This could also serve as a mechanism for health management and prevention.

Many diseases have a latent phase during which an infected individual may not yet be aware of their infectious status. This phase, situated between susceptibility to the disease and confirmed infection, is represented by an intermediate compartment “Exposed” ( $E$ ) inserted between the compartments  $S$  and  $I$ .

## 1.21 SEIR Model Parameters

In the SEIR model, the following parameters are defined as shown in Figure 1.21:

- $\beta$ : the transmission rate of the disease, representing the probability of infection through contact between susceptible and infected individuals.
- $\sigma$ : the incubation rate that governs the transition from exposed to infected. The inverse,  $1/\sigma$ , defines the average duration of the latent period.
- $\gamma$ : the recovery rate. The inverse,  $D = 1/\gamma$ , represents the average duration of infection.
- $\mu$ : the natural mortality rate, representing the probability of death from natural causes. This applies to all compartments.
- $\nu$ : the birth rate, representing the rate at which new individuals enter the population.
- $N$ : the total population size, where  $N = S + E + I + R$ .

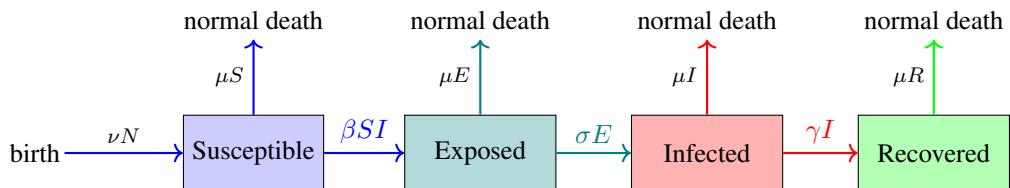


Figure 1.21. SEIR Model Configuration

The system of differential equations governing the SEIR model is given by:

$$\begin{cases} \frac{dS}{dt} = \nu N - \beta SI - \mu S, \\ \frac{dE}{dt} = \beta SI - \sigma E - \mu E, \\ \frac{dI}{dt} = \sigma E - \gamma I - \mu I, \\ \frac{dR}{dt} = \gamma I - \mu R. \end{cases}$$

## 1.22 Stochastic Compartmental Models

Ecological systems are intricate networks of interactions between organisms and their environments, where mathematical models serve as essential tools to understand and predict dynamic behaviors. For example, the Lotka-Volterra model captures the oscillatory dynamics of predator-prey relationships, providing insights into population fluctuations. This model has been widely applied, from studying predator-prey dynamics in natural ecosystems to analyzing the transmission of infectious diseases within human populations.

In contrast, the Gilpin-Ayala model extends its scope beyond predator-prey interactions to explore broader ecological phenomena, such as species co-existence, competition, and community structure. Provides a deeper understanding of the dynamics within microbial communities, vertebrate populations, and other ecological systems, shedding light on the mechanisms that underpin ecosystem stability and resilience [63–66].

These models, along with many others, are indispensable in ecological research. They provide frameworks for analyzing empirical data through sensitivity analyzes, parameter estimation, and simulation studies. In doing so, researchers can uncover the fundamental principles that govern population dynamics and ecosystem behavior. Such models not only improve our understanding of the resilience, adaptability, and interconnectedness of life on Earth, but also inform conservation strategies and management practices aimed at preserving biodiversity and ecosystem services [67, 68].

In natural ecosystems, perturbations are common and can significantly influence ecological dynamics. Perturbations refer to external or internal factors that cause deviations from a system's expected behavior. Examples include natural disasters such as earthquakes, volcanic eruptions, and hurricanes, which can drastically alter landscapes and ecosystems. In biological systems, perturbations can involve sudden climatic changes, the introduction of invasive species, or disease outbreaks. These events can lead to changes in population dynamics, species distributions, and community structures [69–71].

In epidemiological models, stochastic effects are particularly important to understand the spread of infectious diseases. Perturbations in this context may include random variations in contact rates between individuals, pathogen mutations, or changes in public health interventions. For example, the emergence of a new virus strain or unexpected behavioral changes during an epidemic can significantly alter disease transmission patterns. Stochastic models in epidemiology incorporate these random effects, offering more realistic predictions and information on disease spread and control strategies [72–74].

In summary, mathematical modeling in ecology bridges empirical observation and theoretical understanding, allowing the exploration of complex ecological systems and their governing dynamics. Whether through the Lotka-Volterra model, the Gilpin-Ayala model, or other frameworks, these tools provide unique perspectives on the intricate web of life and its proliferation within ecological systems [75].

### 1.22.1 White Noise

In the field of modeling infectious disease, compartmental deterministic models, such as the Susceptible-Infectious-Recovered (SIR) model, have traditionally been used to study the dynamics of disease. However, these models often fail to capture the inherent randomness and variability observed in real-world epidemiological processes. To address this limitation, stochastic compartmental models are employed, incorporating sources of randomness such as **white noise**, which is analogous to **Brownian motion**.

White noise is characterized by its constant variance and uncorrelated fluctuations, making it an ideal representation of the uncertainty and variability in disease transmission and progression.

When integrated into epidemiological models, white noise introduces a dynamic element that captures random events capable of significantly altering the dynamics of the disease. For example, fluctuations in human behavior, seasonal variations, or unexpected superspreading events can all be modeled using white noise.

One common approach to incorporating white noise is to modify the transmission rate, denoted as  $\beta$ , which represents the probability of disease transmission from an infected individual to a susceptible individual. By introducing fluctuations in  $\beta$  through white noise, the model can reflect changes in contact patterns, the effectiveness of preventive measures, or other stochastic factors that influence transmission dynamics. This stochastic approach results in more realistic simulations that capture the unpredictability of infectious disease outbreaks.

In addition, white noise can be applied to other parameters in epidemiological models, such as the recovery rate ( $\gamma$ ) or the duration of infectiousness. Introducing randomness into these parameters allows the model to account for variations in disease progression, treatment efficacy, and population susceptibility over time. This stochastic framework better reflects the complexities of real-world epidemiological dynamics, allowing researchers to explore a broader range of scenarios and assess the effectiveness of various intervention strategies.

In summary, white noise plays a pivotal role in enhancing the realism and predictive accuracy of stochastic compartmental models in infectious disease epidemiology. By simulating the inherent randomness and variability of disease transmission and progression, white noise facilitates more accurate predictions and a deeper understanding of the factors driving epidemic outbreaks. Consider the standard SIR model equations:

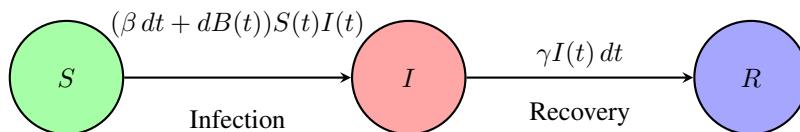
$$\begin{cases} \frac{dS(t)}{dt} = -\beta S(t)I(t), \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - \gamma I(t), \\ \frac{dR(t)}{dt} = \gamma I(t), \end{cases} \quad (1.3)$$

where

- $S$ : Number of susceptible individuals,
- $I$ : Number of infectious individuals,
- $R$ : Number of individuals who have recovered and gained immunity,
- $\beta$ : Transmission rate,
- $\gamma$ : Recovery rate.

To incorporate the effect of white noise on the transmission rate  $\beta$ , we express this as  $\beta + B(t)$ , where  $B(t)$  represents white noise or Brownian motion at time  $t$ . Integrating this modified transmission rate into the SIR model yields the following stochastic differential equations:

$$\begin{cases} dS(t) = -(\beta S(t)I(t)) dt - S(t)I(t)dB(t), \\ dI(t) = (\beta S(t)I(t) - \gamma I(t)) dt + S(t)I(t)dB(t), \\ dR(t) = \gamma I(t)dt. \end{cases}$$



**Figure 1.22. SIR Model with white noise.**

This extension introduces a time-varying transmission rate influenced by the random fluctuations captured by the white noise term  $B(t)$ . The inclusion of white noise allows the model to

## 1.22. STOCHASTIC COMPARTMENTAL MODELS

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account for the unpredictability and variability inherent in disease transmission, making it more representative of real-world scenarios.

In the following sections, we will explore the mathematical properties of stochastic SIR models with white noise, analyze their behavior through simulations, and investigate their implications for understanding infectious disease dynamics in a stochastic context.

### 1.22.2 Telegraph Noise

In the study of infectious disease dynamics, deterministic compartmental models such as the SIR model have been widely used. However, these models often fail to account for the inherent randomness and unpredictability observed in real-world epidemiological processes. To address this limitation, stochastic compartmental models are employed, incorporating sources of randomness such as **telegraph noise**, which can be modeled using a **Markov chain**  $r(t)$ . Telegraph noise is characterized by abrupt, discrete changes in system parameters, simulating sudden events or shifts in behavior that can significantly influence disease dynamics.

Telegraph noise mimics the behavior of a telegraph signal, switching between distinct states. In the context of infectious disease modeling, it can represent sudden changes in public health interventions, such as the implementation or relaxation of lockdown measures, variations in testing strategies, or fluctuations in public compliance with preventive measures. These abrupt changes can lead to significant variations in disease transmission rates, epidemic peaks, and the effectiveness of control strategies.

By incorporating telegraph noise into epidemiological models, researchers can better capture the dynamic nature of real-world interventions and behavioral changes. This stochastic approach enhances the realism of compartmental models, providing deeper insights into the complex dynamics of infectious disease outbreaks. For example, telegraph noise can simulate the impact of sudden policy changes on disease transmission, allowing the evaluation of intervention strategies under realistic and unpredictable conditions.

In addition, telegraph noise can model other stochastic processes in epidemiology, such as the emergence of drug-resistant strains, changes in pathogen virulence, or fluctuations in population susceptibility. By capturing the discontinuous and unpredictable nature of these events, telegraph noise enriches the predictive capabilities of epidemiological models, enabling researchers to better anticipate and respond to emerging infectious disease threats.

In summary, telegraph noise is a powerful tool for enhancing the realism and predictive accuracy of stochastic compartmental models in infectious disease epidemiology. By simulating sudden and discrete changes in real-world epidemiological processes, telegraph noise enables researchers to better understand the dynamics of disease spread and evaluate the effectiveness of control measures in mitigating outbreaks. Consider the standard SIR model equations:

$$\begin{cases} \frac{dS}{dt} &= -\beta SI, \\ \frac{dI}{dt} &= \beta SI - \gamma I, \\ \frac{dR}{dt} &= \gamma I, \end{cases} \quad (1.4)$$

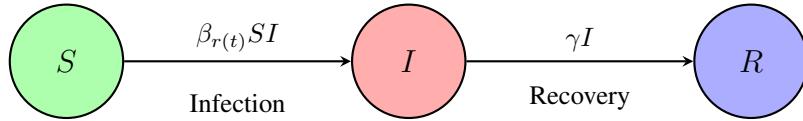
where

- $S$ : Number of susceptible individuals
- $I$ : Number of infectious individuals
- $R$ : Number of recovered individuals
- $\beta$ : Transmission rate
- $\gamma$ : Recovery rate

To incorporate telegraph noise into the transmission rate  $\beta$ , we denote it as  $\beta_{r(t)}$ , where  $r(t)$  is a Markov chain representing telegraph noise. The transmission rate  $\beta_{r(t)}$  switches between

discrete values  $\beta_1, \beta_2, \dots, \beta_n$  based on the state of the Markov chain. Each  $\beta_i$  corresponds to a distinct epidemiological condition, such as different levels of public health interventions or behavioral changes. Incorporating this modified transmission rate into the SIR model yields the following equations:

$$\begin{cases} \frac{dS(t)}{dt} = -\beta_{r(t)}S(t)I(t), \\ \frac{dI(t)}{dt} = \beta_{r(t)}S(t)I(t) - \gamma I(t), \\ \frac{dR(t)}{dt} = \gamma I(t). \end{cases} \quad (1.5)$$



**Figure 1.23.** SIR Model with telegraph noise.

Here,  $\beta_{r(t)}$  introduces variability in the transmission rate, reflecting the dynamic changes in disease transmission due to telegraph noise. The Markov chain  $r(t)$  governs the switching behavior between different values  $\beta_i$ , capturing the unpredictable nature of the dynamics of infectious disease.

In the following sections, we will explore the mathematical properties of stochastic SIR models with telegraph noise, analyze their behavior through simulations, and investigate the implications of this stochasticity for understanding infectious disease spread in real-world scenarios.

### 1.22.3 White Noise and Telegraph Noise in Epidemiological Models

In the scientific literature, the importance of vaccines in epidemiology is well documented, particularly their critical role in preventing the transmission of infectious diseases [66]. Vaccination programs targeting diseases such as tuberculosis, pertussis, tetanus, diphtheria, polio, and measles are essential to achieve herd immunity, a key threshold for preventing outbreaks [67]. Researchers also use epidemiological models, such as **SIS**, **SIR**, **SIRS**, **SEIR**, and **SVIS**, to analyze the impact of vaccination on disease dynamics [73]. More specifically, this study explores how different types of noise can be incorporated into these models. Detailed examples and in-depth analyzes will be provided in the following sections. To illustrate the impact of vaccination in a deterministic framework, consider the following **SIR** model extended to include vaccination:

$$\begin{cases} \frac{dS(t)}{dt} = -(1-q)\vartheta S(t) - (\vartheta + p)S(t) - \beta S(t)I(t) + \gamma I(t) + \delta V(t), \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - (\vartheta + \gamma)I(t), \\ \frac{dV(t)}{dt} = q\vartheta + pS(t) - (\vartheta + \delta)V(t), \end{cases} \quad (1.6)$$

where

- $S$ : Proportion of susceptible individuals
- $I$ : Proportion of infected individuals
- $V$ : Proportion of vaccinated individuals
- $\beta$ : Transmission rate
- $\gamma$ : Recovery rate
- $\vartheta$ : Birth/death rate
- $p$ : Vaccination rate of susceptible individuals
- $q$ : Fraction of newborns vaccinated
- $\delta$ : Rate of immunity loss in vaccinated individuals

This deterministic model assumes constant parameters and does not account for random fluctuations or abrupt changes [65].

To incorporate the inherent randomness in disease transmission, we introduce stochastic elements such as **white noise** and **telegraph noise**. White noise represents continuous random fluctuations, while telegraph noise models sudden changes [69].

First, consider the inclusion of white noise, which introduces continuous stochastic fluctuations in the transmission rate.

$$\begin{cases} dS(t) = [ - (1-q)\vartheta S(t) - (\vartheta + p)S(t) - \beta S(t)I(t) + \gamma I(t) + \delta V(t) ] dt \\ \quad - \sigma S(t)I(t)dB(t), \\ dI(t) = [\beta S(t)I(t) - (\vartheta + \gamma)I(t) ] dt + \sigma S(t)I(t)dB(t), \\ dV(t) = [q\vartheta S(t) + pS(t) - (\vartheta + \delta)V(t) ] dt, \end{cases} \quad (1.7)$$

where  $\sigma$  represents the intensity of the white noise and  $B(t)$  is a standard **Brownian motion**.

Next, to incorporate telegraph noise, we assume that the transmission rate  $\beta$  switches between discrete values  $\beta_1, \beta_2, \dots, \beta_n$  according to a **Markov chain**  $r(t)$  [76]:

$$\begin{cases} dS(t) = [ - (1 - q_{r(t)})\vartheta_{r(t)}S(t) - (\vartheta_{r(t)} + p_{r(t)})S(t) - \beta_{r(t)}S(t)I(t) \\ \quad + \gamma_{r(t)}I(t) + \delta_{r(t)}V(t) ] dt, \\ dI(t) = [\beta_{r(t)}S(t)I(t) - (\vartheta_{r(t)} + \gamma_{r(t)})I(t) ] dt, \\ dV(t) = [q_{r(t)}\vartheta_{r(t)} + p_{r(t)}S(t) - (\vartheta_{r(t)} + \delta_{r(t)})V(t) ] dt. \end{cases} \quad (1.8)$$

Finally, we combine both white noise and telegraph noise into a single stochastic model.

$$\begin{cases} dS(t) = [ - (1 - q_{r(t)})\vartheta_{r(t)}S(t) - (\vartheta_{r(t)} + p_{r(t)})S(t) - \beta_{r(t)}S(t)I(t) \\ \quad + \gamma_{r(t)}I(t) + \delta_{r(t)}V(t) ] dt - \sigma_{r(t)}S(t)I(t)dB(t), \\ dI(t) = [\beta_{r(t)}S(t)I(t) - (\vartheta_{r(t)} + \gamma_{r(t)})I(t) ] dt + \sigma_{r(t)}S(t)I(t)dB(t), \\ dV(t) = [q_{r(t)}\vartheta_{r(t)} + p_{r(t)}S(t) - (\vartheta_{r(t)} + \delta_{r(t)})V(t) ] dt. \end{cases} \quad (1.9)$$

In this combined model:

- $r(t)$  is a Markov chain representing telegraph noise, with states corresponding to different epidemiological conditions.
- $\sigma_{r(t)}$  represents the intensity of the white noise, varying with the state of the Markov chain.
- The parameters  $\beta_{r(t)}$ ,  $\vartheta_{r(t)}$ ,  $p_{r(t)}$ ,  $\gamma_{r(t)}$ , and  $\delta_{r(t)}$  depend on the current state of the Markov chain  $r(t)$ .

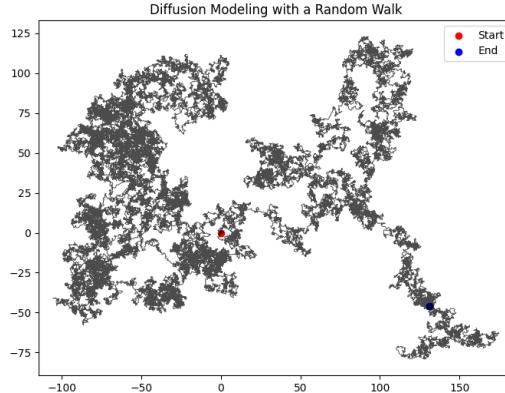
By integrating both white noise and telegraph noise, we can analyze the effects of continuous fluctuations and sudden changes on disease transmission and vaccination dynamics. This comprehensive approach provides a more realistic representation of randomness in epidemiological processes, offering valuable insight into the effectiveness of vaccination strategies and the thresholds for herd immunity under stochastic influences.

Diffusion refers to the process by which an expanding agent (such as a particle, molecule, information, or disease) spreads through a suitable medium to achieve a more homogeneous distribution. This phenomenon is observed in various fields, including chemistry, information science, telecommunications, biology, and epidemiology.

## 1.23 Random Walk and Diffusion

The Figure 1.24 illustrates a two-dimensional random walk, a fundamental model used to represent particle diffusion in various physical and biological systems. Each discrete step represents

stochastic movement, effectively modeling dispersion phenomena ranging from atomic motion to epidemic spread. This visualization was generated by the algorithm presented in the list A.6.



**Figure 1.24.** Two-dimensional Brownian motion simulation demonstrating diffusion through a random walk process. The path shows  $N = 10,000$  steps with uniform step length.

## 1.24 Key Characteristics of Diffusion

The diffusion process exhibits several fundamental properties:

- **Stochastic Dynamics:**

$$X_{t+1} = X_t + \xi_t, \quad \xi_t \sim \mathcal{N}(0, \sigma^2)$$

. where  $\xi_t$  represents random increments independently identically distributed (i.i.d.).

- **Scale Invariance:** The phenomenon manifests itself similarly across multiple orders of magnitude:

- ▶ Microscopic scale ( $\mu\text{m}$ ): Molecular diffusion in fluids
- ▶ Mesoscopic scale (mm-cm): Nutrient dispersion in tissues
- ▶ Macroscopic scale (km): Epidemic spread between populations.

- **Asymptotic Behavior:** The mean squared displacement grows linearly with time,

$$\mathbb{E}[X_t^2] \propto t,$$

which is characteristic of normal diffusion processes.

In epidemiological modeling, spatial diffusion provides crucial insight into disease propagation dynamics. The random walk framework helps quantify:

- Transmission rates between adjacent regions
- Effects of population density on spread velocity
- Impact of mobility restrictions on diffusion coefficients

The visualization demonstrates several key features:

- **Path Irregularity:** Non-differentiable trajectory characteristic of Wiener processes
- **Exploratory Behavior:** Progressive coverage of the plane through ergodic motion
- **Scale-free Properties:** Self-similar patterns at different resolution levels

This mathematical representation bridges microscopic stochastic processes with macroscopic observable phenomena, providing a unified framework for analysis across scientific disciplines.

# CHAPTER 2

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## Mathematical and Stochastic Tools for Dynamic Epidemiological Models

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Stochastic calculus provides a rigorous framework for analyzing systems affected by randomness and noise, especially those modeled by stochastic processes such as Brownian motion. This chapter offers a progressive and in-depth exploration of the key concepts, techniques, and mathematical tools required to understand and solve stochastic differential equations, starting from foundational principles and building up to advanced topics including stability and ergodicity.

The discussion begins with a general overview of Brownian motion and related constructs in probability theory. A review of the foundational notation and the theory of stochastic processes sets the stage for a deeper exploration. We then introduce processes such as the Lévy, Poisson, and compound Poisson processes, which enrich the classical framework by incorporating jumps and discontinuities. Brownian motion, with its continuous, yet nowhere differentiable paths, remains the cornerstone of stochastic modeling, and its properties serve as a natural gateway to the study of stochastic integration.

The next focus is on the construction and properties of Itô's stochastic integral. This begins with the necessary preliminaries and definitions and proceeds to the multidimensional case and its extension to local integrands. A key result in this context is Itô's formula, which generalizes the classical chain rule to stochastic processes. The theory is further solidified through the study of moment inequalities and Gronwall-type estimates, which provide crucial bounds for stochastic integrals and lay the groundwork for the analysis of stochastic differential equations. With the foundation of stochastic integration established, we turn our attention to stochastic differential equations. These equations, typically expressed in the form

$$X_t = X_0 + \int_0^t \sigma(X_s) dB_s + \int_0^t b(X_s) ds,$$

describe systems in which the evolution of a variable over time is influenced by both deterministic trends and random fluctuations. Here,  $B_t$  denotes Brownian motion, and the functions  $\sigma$  and  $b$  capture the diffusion and drift coefficients, respectively. The formulation and solution of such equations rely on the theory of Itô integrals and require conditions such as local Lipschitz continuity to ensure the existence and uniqueness. Approximate solutions, particularly the Euler–Maruyama scheme, provide practical methods for numerical simulations and contribute to the broader study of convergence and error analysis.

An essential part of the theory concerns the stability of solutions. Various notions of sta-

bility are considered, including stability in probability, almost sure exponential stability, and moment-exponential stability. These concepts describe how solutions behave over time under small perturbations or in the presence of noise. In addition, we analyze conditions under which stochastic effects can stabilize or destabilize a system, highlighting the intricate interplay between deterministic structure and random fluctuations.

The chapter concludes with a thorough investigation of the long-term behavior of stochastic systems. This involves the study of recurrence and transience, providing a probabilistic framework for determining whether a process returns to a given state or drifts away. Particular attention is paid to the distinction between positive and null recurrence, along with the criteria for their occurrence. Further refinements explore path excursions and recurrence under linearization. Finally, we address the concept of ergodicity, which concerns the convergence of time averages to ensemble averages and reflects the statistical equilibrium properties of stochastic systems. By establishing when and how ergodic behavior arises, we gain insight into the reliability and predictability of long-term observations.

This chapter offers both theoretical insights and practical tools for understanding stochastic dynamics. The material covered is fundamental to applications in fields such as quantitative finance, physics, biology, engineering, and data science, where systems are often influenced by randomness. The development of stochastic calculus is largely due to the pioneering work of Kolmogorov, Lévy, Doob, and Itô, whose contributions continue to guide contemporary research and applications in probability theory and stochastic modeling. These notes draw inspiration from several sources. The works of Laurence Craig Evans [77] and Bernt Øksendal [78] provide an accessible introduction. The books by Richard Durrett [79], Philip Protter [80], and Hui-Hsiung Kuo [81] are also approachable. More advanced references include the books by Michel Métivier [82], Chris Rogers and David Williams [83, 84], Daniel Stroock and Srinivasa Varadhan [85], Ioannis Karatzas and Steven Shreve [86], Daniel Revuz and Marc Yor [87], Jean Jacod [88], Iosif Gikhman and Anatoli Skorokhod [89], A. V. Skorokhod [90], Rafail Khasminskii [91], G. George Yin and Chao Zhu [92], and Claude Dellacherie and Paul-André Meyer [93, 94]. Finally, accessible references with exercises include the book by Francis Comets and Thierry Meyre [95] (in French) and Paolo Baldi [96], among others.

## 2.1 Brownian Motion and Generalities

### 2.1.1 Generalities and Notations in Probability Theory

- **Sample space:**  $(\Omega)$  The set of all possible outcomes (elementary events).
- **Observable events:**  $(\mathcal{F})$  A family of subsets of  $\Omega$  representing measurable events.
- **A  $\sigma$ -algebra:**  $\mathcal{F}$  on  $\Omega$  must satisfy
  1.  $\emptyset \in \mathcal{F}$  (contains the empty set).
  2. Closed under complements: If  $A \in \mathcal{F}$ , then  $A^C = \Omega \setminus A \in \mathcal{F}$ .
  3. Closed under countable unions: If  $\{A_i\}_{i \geq 1} \subset \mathcal{F}$ , then  $\bigcup_{i=1}^{\infty} A_i \in \mathcal{F}$ .
- **Measurable space:** The pair  $(\Omega, \mathcal{F})$ .
- **$\sigma$ -algebra generated by  $\mathcal{C}$ :** The smallest  $\sigma$ -algebra  $\sigma(\mathcal{C})$  containing  $\mathcal{C}$ .
- **Borel  $\sigma$ -algebra on  $\mathbb{R}^d$ :**  $\mathcal{B}^d = \sigma(\text{open sets in } \mathbb{R}^d)$ .
- **Real-valued random variable:** A function  $X : \Omega \rightarrow \mathbb{R}$  is  $\mathcal{F}$ -measurable, if

$$\{\omega \mid X(\omega) \leq a\} \in \mathcal{F}, \quad \forall a \in \mathbb{R}.$$

- **Random vector/matrix:** Measurable if all their components are measurable.

- **Indicator function** of a set  $A \subset \Omega$

$$\mathbb{1}_A(\omega) = \begin{cases} 1 & \text{if } \omega \in A, \\ 0 & \text{otherwise.} \end{cases}$$

$\mathbb{1}_A$  is  $\mathcal{F}$ -measurable if and only if  $A \in \mathcal{F}$ .

- **Random variable taking values in  $(\Omega', \mathcal{F}')$**  A mapping  $X : \Omega \rightarrow \Omega'$  is  $(\mathcal{F}, \mathcal{F}')$ -measurable if

$$\{\omega \mid X(\omega) \in A'\} \in \mathcal{F}, \quad \forall A' \in \mathcal{F}'.$$

- For  $X : \Omega \rightarrow \mathbb{R}^d$ , the  $\sigma$ -algebra generated by  $X$  is

$$\sigma(X) = \sigma(\{\omega \in \Omega \mid X(\omega) \in U\} \mid U \subset \mathbb{R}^d \text{ open}).$$

- For a family  $\{X_i; i \in I\}$ :

$$\sigma(X_i; i \in I) = \sigma\left(\bigcup_{i \in I} \sigma(X_i)\right).$$

- The  $\sigma$ -algebra  $\sigma(X)$  characterizes the measurability of functions with respect to  $X$ .

**Lemma 2.1 (Measurability condition).** *If  $X, Y : \Omega \rightarrow \mathbb{R}^d$  are two given functions, then  $Y$  is  $\sigma(X)$ -measurable if and only if there exists a Borel measurable function  $g : \mathbb{R}^d \rightarrow \mathbb{R}^d$  such that  $Y = g(X)$ .*

**Definition 2.2 (Probability measure).** *A probability measure  $\mathbf{P}$  on a measurable space  $(\Omega, \mathcal{F})$  is a function  $\mathbf{P} : \mathcal{F} \rightarrow [0, 1]$  satisfying*

- $\mathbf{P}(\Omega) = 1$  (normalization).
- For any disjoint sequence  $\{A_i\}_{i \geq 1} \subset \mathcal{F}$  (i.e.,  $A_i \cap A_j = \emptyset$  for  $i \neq j$ ),

$$\mathbf{P}\left(\bigcup_{i=1}^{\infty} A_i\right) = \sum_{i=1}^{\infty} \mathbf{P}(A_i) \quad (\text{countable additivity}).$$

**Definition 2.3 (Probability space).** *The triple  $(\Omega, \mathcal{F}, \mathbf{P})$  is called a probability space.*

**Definition 2.4 (Completion of a  $\sigma$ -algebra).** *For a probability space  $(\Omega, \mathcal{F}, \mathbf{P})$ , define*

$$\overline{\mathcal{F}} = \{A \subset \Omega : \exists B, C \in \mathcal{F} \text{ with } B \subset A \subset C \text{ and } \mathbf{P}(B) = \mathbf{P}(C)\}.$$

- $\overline{\mathcal{F}}$  is a  $\sigma$ -algebra called the completion of  $\mathcal{F}$ .
- If  $\mathcal{F} = \overline{\mathcal{F}}$ , the space is called complete.
- Otherwise,  $\mathbf{P}$  can be extended to  $\overline{\mathcal{F}}$  by setting  $\mathbf{P}(A) = \mathbf{P}(B) = \mathbf{P}(C)$ , making  $(\Omega, \overline{\mathcal{F}}, \mathbf{P})$  complete.

**Definition 2.5 (Expectation and moments).** *Let  $X$  be a real-valued random variable defined on a probability space  $(\Omega, \mathcal{F}, \mathbf{P})$ . The expectation of  $X$  is defined as  $\mathbb{E}(X) = \int_{\Omega} X d\mathbf{P}$ , its variance as  $V(X) = \mathbb{E}[(X - \mathbb{E}(X))^2]$ , and for  $p > 0$ , the  $p$ -th moment is given by  $\mathbb{E}(|X|^p)$ .*

**Definition 2.6 (Covariance).** *For two real-valued random variables  $X$  and  $Y$ ,*

$$\text{Cov}(X, Y) = \mathbb{E}[(X - \mathbb{E}(X))(Y - \mathbb{E}(Y))].$$

- $X$  and  $Y$  are uncorrelated if  $\text{Cov}(X, Y) = 0$ .

For  $\mathbb{R}^d$ -valued random variables:

- $X = (X_1, \dots, X_d)^T$  has expectation  $\mathbb{E}(X) = (\mathbb{E}(X_1), \dots, \mathbb{E}(X_d))^T$ .
- For matrix-valued  $X = (X_{ij})_{d \times m}$ ,  $\mathbb{E}(X) = (\mathbb{E}(X_{ij}))_{d \times m}$ .
- The covariance matrix is

$$\text{Cov}(X, Y) = \mathbb{E} [(X - \mathbb{E}(X))(Y - \mathbb{E}(Y))^T],$$

which is symmetric and non-negative definite.

**Definition 2.7 (Distribution of a random variable).** Let  $X$  be an  $\mathbb{R}^d$ -valued random variable. Then  $X$  induces a probability measure  $\mu_X$  on  $(\mathbb{R}^d, \mathcal{B}^d)$ , called its distribution, defined by

$$\mu_X(B) = \mathbf{P}\{\omega \mid X(\omega) \in B\} \quad \text{for } B \in \mathcal{B}^d.$$

The expectation can then be expressed as

$$\mathbb{E}(X) = \int_{\mathbb{R}^d} x d\mu_X(x).$$

**Proposition 2.8 (Transformation formula).** If  $g : \mathbb{R}^d \rightarrow \mathbb{R}^m$  is Borel measurable, then

$$\mathbb{E}(g(X)) = \int_{\mathbb{R}^d} g(x) d\mu_X(x).$$

**Definition 2.9 ( $L^p$  spaces).** For  $p \in (0, \infty)$ , define

$$L^p = L^p(\Omega, \mathbb{R}^d) = \{X : \mathbb{R}^d\text{-valued random variables with } \mathbb{E}(|X|^p) < \infty\}.$$

In  $L^1$ , we have the fundamental inequality

$$|\mathbb{E}(X)| \leq \mathbb{E}(|X|).$$

**Theorem 2.10 (Fundamental inequalities).**

1. **Hölder's inequality:** For  $p > 1$ , where  $\frac{1}{p} + \frac{1}{q} = 1$ ,  $X \in L^p$ , and  $Y \in L^q$ ,

$$|\mathbb{E}(X^T Y)| \leq (\mathbb{E}(|X|^p))^{1/p} (\mathbb{E}(|Y|^q))^{1/q}.$$

2. **Minkowski's inequality:** For  $p \geq 1$  and  $X, Y \in L^p$ ,

$$(\mathbb{E}(|X + Y|^p))^{1/p} \leq (\mathbb{E}(|X|^p))^{1/p} + (\mathbb{E}(|Y|^p))^{1/p}.$$

3. **Chebyshev's inequality:** For  $c > 0$ ,  $p > 0$ , and  $X \in L^p$ ,

$$\mathbf{P}\{|X| \geq c\} \leq c^{-p} \mathbb{E}(|X|^p).$$

**Corollary 2.11 (Moment comparison).** For  $0 < r < p < \infty$  and  $X \in L^p$ , we have

$$(\mathbb{E}(|X|^r))^{1/r} \leq (\mathbb{E}(|X|^p))^{1/p}.$$

**Definition 2.12 (Modes of convergence).** Let  $X$  and  $\{X_k\}_{k \geq 1}$  be  $\mathbb{R}^d$ -valued random variables.

- **Almost sure convergence:**  $X_k \rightarrow X$  a.s. if there exists a  $\mathbf{P}$ -null set  $\Omega_0 \in \mathcal{F}$  such that for every  $\omega \notin \Omega_0$ ,

$$\lim_{k \rightarrow \infty} X_k(\omega) = X(\omega) \quad (\text{in the usual sense in } \mathbb{R}^d).$$

Denoted by  $\lim_{k \rightarrow \infty} X_k = X$  a.s.

- **Convergence in probability:**  $X_k \xrightarrow{\mathbf{P}} X$  if for all  $\varepsilon > 0$ ,

$$\lim_{k \rightarrow \infty} \mathbf{P}\{|X_k - X| > \varepsilon\} = 0.$$

- **Convergence in  $\mathbf{L}^p$ :** For  $X_k, X \in \mathbf{L}^p$ ,  $X_k \xrightarrow{\mathbf{L}^p} X$  if

$$\lim_{k \rightarrow \infty} \mathbb{E}(|X_k - X|^p) = 0.$$

- **Convergence in distribution:**  $X_k \xrightarrow{d} X$  if for all continuous and bounded  $g : \mathbb{R}^d \rightarrow \mathbb{R}$ ,

$$\lim_{k \rightarrow \infty} \mathbb{E}(g(X_k)) = \mathbb{E}(g(X)).$$

**Theorem 2.13 (Characterizations of convergence).** Let  $\{X_k\}_{k \in \mathbb{N}}$  be a sequence of random variables and  $X$  a random variable. Then:

- $X_k \xrightarrow{\mathbf{P}} X$  if and only if every subsequence  $\{X_{k_n}\}_{n \in \mathbb{N}}$  contains a further subsequence  $\{X_{k_{n_m}}\}_{m \in \mathbb{N}}$  that converges almost surely to  $X$ .
- If  $\sum_{k=1}^{\infty} \mathbb{E}[|X_k - X|^p] < \infty$  for some  $p > 0$ , then  $X_k \xrightarrow{\text{a.s.}} X$ .

**Theorem 2.14 (Monotone Convergence Theorem).** If  $\{X_k\}$  is an increasing sequence of nonnegative random variables, then

$$\lim_{k \rightarrow \infty} \mathbb{E}(X_k) = \mathbb{E}\left(\lim_{k \rightarrow \infty} X_k\right).$$

**Theorem 2.15 (Dominated Convergence Theorem).** Let  $p \geq 1$ ,  $\{X_k\} \subset \mathbf{L}^p(\Omega, \mathbb{R}^d)$ , and  $Y \in \mathbf{L}^p(\Omega, \mathbb{R})$ . If

$$|X_k| \leq Y \quad \text{almost surely},$$

and  $X_k \xrightarrow{\mathbf{P}} X$ , then

$$X \in \mathbf{L}^p(\Omega, \mathbb{R}^d),$$

$$X_k \xrightarrow{\mathbf{L}^p} X,$$

and

$$\lim_{k \rightarrow \infty} \mathbb{E}(X_k) = \mathbb{E}(X).$$

When  $Y$  is bounded, this is called the Bounded Convergence Theorem.

**Definition 2.16 (Independence).**

- A collection  $\{A_i \mid i \in I\} \subset \mathcal{F}$  is independent if for all finite subsets  $\{i_1, \dots, i_k\} \subset I$ ,

$$\mathbf{P}\left(\bigcap_{j=1}^k A_{i_j}\right) = \prod_{j=1}^k \mathbf{P}(A_{i_j}).$$

- Two sub- $\sigma$ -algebras  $\mathcal{F}_1, \mathcal{F}_2 \subset \mathcal{F}$  are independent if

$$\mathbf{P}(A_1 \cap A_2) = \mathbf{P}(A_1)\mathbf{P}(A_2) \quad \forall A_1 \in \mathcal{F}_1, A_2 \in \mathcal{F}_2.$$

- A collection  $\{\mathcal{F}_i \mid i \in I\}$  is independent if for all finite subsets  $\{i_1, \dots, i_k\} \subset I$  and  $A_{i_j} \in \mathcal{F}_{i_j}$ ,

$$\mathbf{P}\left(\bigcap_{j=1}^k A_{i_j}\right) = \prod_{j=1}^k \mathbf{P}(A_{i_j}).$$

**Definition 2.17 (Independent random variables).** A family  $\{X_i \mid i \in I\}$  is independent if the  $\sigma$ -algebras  $\{\sigma(X_i) \mid i \in I\}$  are independent. Two random variables  $X : \Omega \rightarrow \mathbb{R}^d$  and  $Y : \Omega \rightarrow \mathbb{R}^m$  are independent if and only if

$$\mathbf{P}(X \in A, Y \in B) = \mathbf{P}(X \in A)\mathbf{P}(Y \in B)$$

, for all  $A \in \mathcal{B}^d$  and  $B \in \mathcal{B}^m$ .

**Theorem 2.18 (Properties of independent variables).** For independent integrable random variables:

- If  $X, Y$  are independent and integrable, then  $XY$  is integrable and

$$\mathbb{E}(XY) = \mathbb{E}(X) \cdot \mathbb{E}(Y).$$

- For  $X, Y \in \mathbf{L}^2(\Omega, \mathbb{R})$ :

- ▶ If  $X$  and  $Y$  are uncorrelated, then  $V(X + Y) = V(X) + V(Y)$ ,
- ▶ If  $X$  and  $Y$  are independent, then they are uncorrelated,
- ▶ For jointly normal independent random variables,  $X$  and  $Y$  are uncorrelated if and only if they are independent.

**Definition 2.19 (Limit superior of sets).** For a sequence  $\{A_k\} \subset \mathcal{F}$ , the limit superior is defined as

$$\limsup_{k \rightarrow \infty} A_k = \{\omega \in \Omega \mid \omega \in A_k \text{ for infinitely many } k\} = \bigcap_{i=1}^{\infty} \bigcup_{k=i}^{\infty} A_k \in \mathcal{F}.$$

**Lemma 2.20 (Borel-Cantelli Lemma).**

1. If  $\sum_{k=1}^{\infty} \mathbf{P}(A_k) < \infty$ , then  $\mathbf{P}(\limsup_{k \rightarrow \infty} A_k) = 0$ .
2. If  $\{A_k\} \subset \mathcal{F}$  is independent and  $\sum_{k=1}^{\infty} \mathbf{P}(A_k) = \infty$ , then  $\mathbf{P}(\limsup_{k \rightarrow \infty} A_k) = 1$ . That is, there exists a set  $\Omega_{\theta} \in \mathcal{F}$  with  $\mathbf{P}(\Omega_{\theta}) = 1$  such that for all  $\omega \in \Omega_{\theta}$ ,  $\omega$  belongs to infinitely many  $A_k$ .

**Definition 2.21 (Conditional probability).** For  $A, B \in \mathcal{F}$  with  $\mathbf{P}(B) > 0$ , the conditional probability is given by

$$\mathbf{P}(A \mid B) = \frac{\mathbf{P}(A \cap B)}{\mathbf{P}(B)}.$$

**Definition 2.22 (Conditional expectation).** Let  $X \in \mathbf{L}^1(\Omega, \mathbb{R})$  and  $\mathcal{G} \subset \mathcal{F}$  be a sub- $\sigma$ -algebra. The conditional expectation  $\mathbb{E}(X | \mathcal{G})$  is the almost surely unique  $\mathcal{G}$ -measurable random variable satisfying

$$\int_G X d\mathbf{P} = \int_G \mathbb{E}(X | \mathcal{G}) d\mathbf{P}, \quad \forall G \in \mathcal{G}.$$

When  $\mathcal{G} = \sigma(Y)$ , we write  $\mathbb{E}(X | Y)$ .

**Example 2.23 (Discrete case).** Consider a countable partition  $\{A_k\} \subset \mathcal{F}$  that satisfies  $\bigcup_k A_k = \Omega$  with  $\mathbf{P}(A_k) > 0$  and  $A_k \cap A_j = \emptyset$  for  $k \neq j$ . For  $\mathcal{G} = \sigma(\{A_k\})$ , the conditional expectation becomes

$$\mathbb{E}(X | \mathcal{G}) = \sum_k \frac{\mathbb{E}(X \mathbb{1}_{A_k})}{\mathbf{P}(A_k)} \mathbb{1}_{A_k}.$$

For any  $\omega \in A_k$ , this simplifies to

$$\mathbb{E}(X | \mathcal{G})(\omega) = \frac{\mathbb{E}(X \mathbb{1}_{A_k})}{\mathbf{P}(A_k)} = \mathbb{E}(X | A_k).$$

**Proposition 2.24 (Properties of Conditional Expectation).** For  $X \in \mathbf{L}^1(\Omega, \mathbb{R})$  and a sub- $\sigma$ -algebra  $\mathcal{G} \subset \mathcal{F}$ , the following properties hold almost surely:

■ **Tower property:**

$$\mathbb{E}[\mathbb{E}(X | \mathcal{G})] = \mathbb{E}(X).$$

■ **Jensen's inequality:**

$$|\mathbb{E}(X | \mathcal{G})| \leq \mathbb{E}(|X| | \mathcal{G}).$$

■ **Trivial  $\sigma$ -algebra:** If  $\mathcal{G} = \{\emptyset, \Omega\}$ , then

$$\mathbb{E}(X | \mathcal{G}) = \mathbb{E}(X).$$

■ **Positivity:** If  $X \geq 0$ , then

$$\mathbb{E}(X | \mathcal{G}) \geq 0.$$

■ **Measurability:** If  $X$  is  $\mathcal{G}$ -measurable, then

$$\mathbb{E}(X | \mathcal{G}) = X.$$

■ **Constants:** If  $X = c$  for some constant  $c \in \mathbb{R}$ , then

$$\mathbb{E}(X | \mathcal{G}) = c.$$

■ **Linearity:** For  $a, b \in \mathbb{R}$ , we have

$$\mathbb{E}(aX + bY | \mathcal{G}) = a\mathbb{E}(X | \mathcal{G}) + b\mathbb{E}(Y | \mathcal{G}).$$

■ **Monotonicity:** If  $X \leq Y$ , then

$$\mathbb{E}(X | \mathcal{G}) \leq \mathbb{E}(Y | \mathcal{G}).$$

■ **Pull-out property:** If  $X$  is  $\mathcal{G}$ -measurable, then

$$\mathbb{E}(XY | \mathcal{G}) = X\mathbb{E}(Y | \mathcal{G}).$$

- **Independence:** If  $\sigma(X)$  and  $\mathcal{G}$  are independent, then

$$\mathbb{E}(X \mid \mathcal{G}) = \mathbb{E}(X).$$

- **Nested conditioning:** If  $\mathcal{G}_1 \subset \mathcal{G}_2$ , then

$$\mathbb{E}[\mathbb{E}(X \mid \mathcal{G}_2) \mid \mathcal{G}_1] = \mathbb{E}(X \mid \mathcal{G}_1).$$

**Definition 2.25 (Vector conditional expectation).** For  $X = (X_1, \dots, X_d)^T \in \mathbf{L}^1(\Omega, \mathbb{R}^d)$ , the vector-valued conditional expectation is given by

$$\mathbb{E}(X \mid \mathcal{G}) = (\mathbb{E}(X_1 \mid \mathcal{G}), \dots, \mathbb{E}(X_d \mid \mathcal{G}))^T.$$

## 2.1.2 Stochastic Processes

**Definition 2.26 (Filtration).** A filtration  $\{\mathcal{F}_t\}_{t \geq 0}$  is defined as

- A family of increasing sub- $\sigma$ -algebras ( $\mathcal{F}_t \subset \mathcal{F}_s$  for  $t < s$ ),
- Right-continuous if  $\mathcal{F}_t = \bigcap_{s > t} \mathcal{F}_s$ ,
- Said to satisfy the usual conditions if it is right-continuous and  $\mathcal{F}_0$  contains all  $\mathbf{P}$ -null sets.

**Definition 2.27 (Stochastic process).** A stochastic process is a family  $\{X_t\}_{t \in I}$  of  $\mathbb{R}^d$ -valued random variables where

- $I$  is typically  $\mathbb{R}_+ = [0, \infty)$ , an interval  $[a, b]$ , or  $\mathbb{N}$ ,
- For each  $t \in I$ ,  $X_t : \Omega \rightarrow \mathbb{R}^d$  is a random variable.

**Definition 2.28 (Complete filtration).** For  $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbf{P})$ , a complete filtered probability space, we define

$$\mathcal{F}_\infty = \sigma \left( \bigcup_{t \geq 0} \mathcal{F}_t \right),$$

where  $\sigma \left( \bigcup_{t \geq 0} \mathcal{F}_t \right)$  denotes the smallest  $\sigma$ -algebra containing all  $\mathcal{F}_t$ -measurable sets for  $t \geq 0$ .

**Definition 2.29 (Sample paths).** For a stochastic process  $\{X_t\}_{t \in I}$ ,

- For a fixed  $\omega \in \Omega$ , the mapping  $t \mapsto X_t(\omega)$  is called a sample path,
- An alternative notation  $X(t, \omega)$  emphasizes the two-parameter nature.

**Definition 2.30 (Process regularity).** An  $\mathbb{R}^d$ -valued process  $\{X_t\}_{t \geq 0}$  is said to be

- **Continuous:** if almost all sample paths are continuous,
- **Càdlàg:** (right-continuous with left limits) if it is right-continuous and  $\lim_{s \uparrow t} X_s(\omega)$  exists and is finite for all  $t > 0$ , almost surely,
- **Integrable:** if  $X_t \in \mathbf{L}^1$  for all  $t \geq 0$ .

**Definition 2.31 (Measurability properties).** A process  $\{X_t\}_{t \geq 0}$  is said to be

- **Adapted:** if  $X_t$  is  $\mathcal{F}_t$ -measurable for all  $t$ ,

- **Measurable:** if  $(t, \omega) \mapsto X_t(\omega)$  is  $\mathcal{B}(\mathbb{R}_+) \times \mathcal{F}$ -measurable,
- **Progressively measurable:** if for all  $T > 0$ , the process  $\{X_t\}_{0 \leq t \leq T}$  is  $\mathcal{B}([0, T]) \otimes \mathcal{F}_T$ -measurable.

**Definition 2.32 (Special process classes).** The following are special process classes:

- **Optional:** ( $\mathcal{O}$ -measurable) The smallest  $\sigma$ -algebra making all càdlàg adapted processes measurable,
- **Predictable:** ( $\mathcal{P}$ -measurable) The smallest  $\sigma$ -algebra making all left-continuous adapted processes measurable,
- **Increasing process:** A process with almost surely nonnegative, nondecreasing, right-continuous paths,
- **Finite variation:** A process that can be expressed as  $A_t = \bar{A}_t - \hat{A}_t$  where  $\bar{A}_t$  and  $\hat{A}_t$  are increasing processes.

**Definition 2.33 (Modifications and Indistinguishability of Processes).** Two stochastic processes  $\{X_t\}_{t \geq 0}$  and  $\{Y_t\}_{t \geq 0}$  are called

- **Modifications (or versions):** of each other if for all  $t \geq 0$ ,  $\mathbf{P}(X_t = Y_t) = 1$ ,
- **Indistinguishable:** if  $\mathbf{P}(\forall t \geq 0, X_t = Y_t) = 1$ .

**Definition 2.34 (Stopping time).** A random variable  $\tau : \Omega \rightarrow [0, \infty]$  is an  $\{\mathcal{F}_t\}$ -stopping time if

$$\{\tau \leq t\} \in \mathcal{F}_t \quad \text{for all } t \geq 0.$$

**Definition 2.35 (Stochastic Intervals).** For stopping times  $\tau \leq \rho$ , define the interval

$$[[\tau, \rho]] = \{(t, \omega) ; \tau(\omega) \leq t \leq \rho(\omega)\}.$$

Other intervals, such as  $((\tau, \rho]]$ ,  $[[\tau, \rho))$ , and  $((\tau, \rho))$ , are defined similarly.

**Definition 2.36 (Stopped  $\sigma$ -algebra).** For a stopping time  $\tau$ , the stopped  $\sigma$ -algebra is defined as

$$\mathcal{F}_\tau = \{A \in \mathcal{F} ; A \cap \{\tau \leq t\} \in \mathcal{F}_t \text{ for all } t \geq 0\}.$$

If  $\tau \leq \rho$ , then  $\mathcal{F}_\tau \subseteq \mathcal{F}_\rho$ .

**Theorem 2.37 (Measurability of stopped processes).** For any progressively measurable process  $\{X_t\}$  and stopping time  $\tau$ , the random variable  $X_\tau \mathbb{1}_{\{\tau < \infty\}}$  is  $\mathcal{F}_\tau$ -measurable. Moreover, if  $\tau < \infty$  almost surely, then  $X_\tau$  is  $\mathcal{F}_\tau$ -measurable.

**Theorem 2.38 (Exit times).** Let  $\{X_t\}$  be a càdlàg  $\{\mathcal{F}_t\}$ -adapted process, and let  $D \subset \mathbb{R}^d$  be open. Then

- $\tau = \inf\{t \geq 0 ; X_t \notin D\}$  is a stopping time.
- For any stopping time  $\rho$ ,  $\theta = \inf\{t \geq \rho ; X_t \notin D\}$  is a stopping time.

**Definition 2.39 (Martingale).** An  $\mathbb{R}^d$ -valued  $\{\mathcal{F}_t\}$ -adapted integrable process  $\{M_t\}$  is a martingale if

$$\mathbb{E}(M_t | \mathcal{F}_s) = M_s \quad \text{almost surely for all } 0 \leq s < t.$$

- Every martingale has a càdlàg modification.
- The stopped process is defined as  $M^\tau = \{M_{\tau \wedge t}\}_{t \geq 0}$ .

**Theorem 2.40 (Doob's stopping theorem).** For a martingale  $\{M_t\}$  and finite stopping times  $\theta, \rho$ , we have

$$\mathbb{E}(M_\theta \mid \mathcal{F}_\rho) = M_{\theta \wedge \rho} \quad \text{almost surely.}$$

**Theorem 2.41 (Stopped Martingale Property).** For any martingale  $\{M_t\}$  and stopping time  $\tau$ , the stopped process  $M_{\tau \wedge t}$  is a martingale satisfying

$$\mathbb{E}(M_{\tau \wedge t} \mid \mathcal{F}_s) = M_{\tau \wedge s} \quad \text{almost surely for all } 0 \leq s < t.$$

**Definition 2.42 (Square-Integrable).** A process  $\{X_t\}$  is square-integrable if  $\mathbb{E}(|X_t|^2) < \infty$  for all  $t \geq 0$ .

**Definition 2.43 (Quadratic variation of a stochastic process).** Let  $X = (X_t)_{t \geq 0}$  be a càdlàg adapted stochastic process on a complete filtered space  $(\Omega, \mathcal{F}, (\mathcal{F}_t)_{t \geq 0}, \mathbb{P})$ . The quadratic variation of  $X$  is the process  $[X] = ([X]_t)_{t \geq 0}$  defined as the following limit in probability:

$$[X]_t \stackrel{\Delta}{=} \lim_{n \rightarrow +\infty} \sum_{k=1}^{2^n} (X_{t \wedge k2^{-n}} - X_{t \wedge (k-1)2^{-n}})^2, \quad (2.1)$$

where the convergence is uniform on compact time intervals. More generally, for any sequence of partitions  $(\pi_n)_{n \in \mathbb{N}}$  of  $\mathbb{R}^+$  with mesh tending to zero, i.e.,  $\|\pi_n\| \rightarrow 0$ , we have:

$$[X]_t = \lim_{n \rightarrow +\infty} \sum_{t_i \in \pi_n} (X_{t_{i+1} \wedge t} - X_{t_i \wedge t})^2 \quad \text{in probability.} \quad (2.2)$$

This limit always exists for semimartingales and defines an increasing, adapted, càdlàg process.

**Definition 2.44 (Angle bracket of a continuous local martingale).** Let  $M \in \mathcal{M}_{\text{loc},c}$  be a continuous local martingale. There exists a unique process  $\langle M \rangle = (\langle M \rangle_t)_{t \geq 0}$  satisfying the following properties:

1.  $\langle M \rangle$  is predictable, i.e.,  $\mathcal{P}$ -measurable, where  $\mathcal{P}$  denotes the predictable  $\sigma$ -algebra,
2.  $t \mapsto \langle M \rangle_t$  is almost surely continuous, increasing, and satisfies  $\langle M \rangle_0 = 0$ ,
3. the process  $M^2 - \langle M \rangle$  is a local martingale.

For a general local martingale  $M \in \mathcal{M}_{\text{loc}}$ , the angle bracket is defined as the predictable projection of its quadratic variation, namely:

$$\langle M \rangle \stackrel{\Delta}{=} [M]^p, \quad (2.3)$$

where  ${}^p$  denotes the predictable projection (dual predictable projection in the Doob-Meyer decomposition).

**Theorem 2.45 (Canonical decomposition of quadratic variation).** Let  $M$  be a local martingale. Its quadratic variation admits the following decomposition, valid for all  $t \geq 0$ :

$$[M]_t = \langle M^c \rangle_t + \sum_{0 < s \leq t} (\Delta M_s)^2, \quad (2.4)$$

where  $M^c$  denotes the continuous part of  $M$  and  $\Delta M_s := M_s - M_{s-}$  represents the jumps of  $M$ . More precisely:

1. if  $M$  is continuous, then  $[M] = \langle M \rangle$  is a continuous process,
2. if  $M$  is purely discontinuous, then  $[M]_t = \sum_{s \leq t} (\Delta M_s)^2$ ,
3. in the general case,  $\langle M \rangle = \langle M^c \rangle$  corresponds to the compensated (predictable) part of  $[M]$ .

This decomposition represents the stochastic analogue of the classical formula  $d(x^2) = 2x dx + d\langle x \rangle$  in ordinary differential calculus.

**Theorem 2.46 (Quadratic Variation).** For a real-valued continuous square-integrable martingale  $\{M_t\}_{t \geq 0}$ , the following hold:

- There exists a unique continuous, increasing, integrable, and adapted process  $\langle M \rangle_t$  such that  $\{M_t^2 - \langle M \rangle_t\}$  is a continuous martingale vanishing at  $t = 0$ .
- For any finite stopping time  $\tau$ , we have

$$\mathbb{E}[M_\tau^2] = \mathbb{E}[\langle M \rangle_\tau].$$

**Definition 2.47 (Joint Quadratic Variation).** For continuous square-integrable martingales  $M$  and  $N$ , the joint quadratic variation is defined by

$$\langle M, N \rangle_t = \frac{1}{4} (\langle M + N \rangle_t - \langle M - N \rangle_t).$$

- The process  $\{M_t N_t - \langle M, N \rangle_t\}$  is a martingale.
- For a finite stopping time  $\tau$ , we have

$$\mathbb{E}(M_\tau N_\tau) = \mathbb{E}(\langle M, N \rangle_\tau).$$

**Definition 2.48 (Local Martingale).** A right-continuous adapted process  $\{M_t\}$  is called a local martingale if there exist stopping times  $\tau_k \uparrow \infty$  almost surely such that processes  $\{M_{\tau_k \wedge t} - M_0\}$  are martingales.

**Theorem 2.49 (Quadratic Variation for Local Martingales).** For continuous local martingales  $M$  and  $N$ , the following hold:

- There exists a unique finite variation process  $\langle M, N \rangle_t$  making  $\{M_t N_t - \langle M, N \rangle_t\}$  a local martingale.
- When  $M = N$ ,  $\langle M \rangle_t$  is the quadratic variation.

**Theorem 2.50 (Strong Law for Martingales).** For a continuous local martingale  $\{M_t\}$  with  $M_0 = 0$ , the following results hold:

1. If  $\lim_{t \rightarrow \infty} \langle M \rangle_t = \infty$  almost surely, then

$$\lim_{t \rightarrow \infty} \frac{M_t}{\langle M \rangle_t} = 0 \quad \text{almost surely.}$$

2. If  $\limsup_{t \rightarrow \infty} \frac{\langle M \rangle_t}{t} < \infty$  almost surely, then

$$\lim_{t \rightarrow \infty} \frac{M_t}{t} = 0 \quad \text{almost surely.}$$

3. For a continuous adapted increasing process  $\{A_t\}$  with

$$\lim_{t \rightarrow \infty} A_t = \infty \quad \text{almost surely, and} \quad \int_0^\infty \frac{d\langle M \rangle_t}{(1 + A_t)^2} < \infty \quad \text{almost surely,}$$

we have

$$\lim_{t \rightarrow \infty} \frac{M_t}{A_t} = 0 \quad \text{almost surely.}$$

**Definition 2.51 (Supermartingales and Submartingales).** An adapted integrable process  $\{M_t\}$  is

- A **supermartingale** if  $\mathbb{E}(M_t | \mathcal{F}_s) \leq M_s$  almost surely for all  $s \leq t$ ,
- A **submartingale** if  $\mathbb{E}(M_t | \mathcal{F}_s) \geq M_s$  almost surely for all  $s \leq t$ .

**Properties:**

- If  $\{M_t\}$  is a martingale, then  $\{|M_t|^p\}$  is a submartingale for  $p \geq 1$ ,
- $\mathbb{E}(M_t)$  decreases for supermartingales and increases for submartingales,
- Doob's stopping theorem extends to supermartingales and submartingales.

**Theorem 2.52 (Doob's Martingale Convergence Theorem).** For a right-continuous supermartingale satisfying  $\sup_t \mathbb{E}[M_t^-] < \infty$ , the following results hold:

1. The limit  $M_t \rightarrow M_\infty$  exists almost surely, with  $M_\infty \in L^1$  (this property always holds for nonnegative supermartingales).
2. A right-continuous supermartingale is uniformly integrable if and only if  $M_t \rightarrow M_\infty$  both almost surely and in  $L^1$ , for some  $M_\infty \in L^1$ .
3. For any  $X \in L^1$ , the conditional expectations satisfy

$$\mathbb{E}(X | \mathcal{F}_t) \rightarrow \mathbb{E}(X | \mathcal{F}_\infty)$$

, with convergence both almost surely and in  $L^1$ .

**Theorem 2.53 (Doob's Supermartingale Inequalities).** For a real-valued supermartingale  $\{M_t\}$  and an interval  $[a, b] \subset \mathbb{R}_+$ , the following inequalities hold for all  $c > 0$ :

$$\begin{aligned} c\mathbf{P}\left(\sup_{a \leq t \leq b} M_t \geq c\right) &\leq \mathbb{E}[M_a] + \mathbb{E}[M_b^-], \\ c\mathbf{P}\left(\inf_{a \leq t \leq b} M_t \leq -c\right) &\leq \mathbb{E}[M_b^-]. \end{aligned}$$

**Theorem 2.54 (Doob's Submartingale Inequalities).** For  $p > 1$  and a nonnegative submartingale  $\{M_t\} \subset \mathbf{L}^p(\Omega, \mathbb{R})$ , the following inequality holds for any bounded interval  $[a, b] \subset \mathbb{R}_+$ :

$$\mathbb{E}\left[\sup_{a \leq t \leq b} M_t^p\right] \leq \left(\frac{p}{p-1}\right)^p \mathbb{E}[M_b^p].$$

**Theorem 2.55 (Doob's Martingale Inequalities).** For an  $\mathbb{R}^d$ -valued martingale  $\{M_t\}$  and  $[a, b] \subset \mathbb{R}_+$ , the following inequalities hold:

1. If  $p \geq 1$  and  $M_t \in \mathbf{L}^p(\Omega, \mathbb{R}^d)$ , then

$$\mathbf{P}\left(\sup_{a \leq t \leq b} |M_t| \geq c\right) \leq \frac{\mathbb{E}[|M_b|^p]}{c^p} \quad \text{for all } c > 0.$$

2. If  $p > 1$  and  $M_t \in \mathbf{L}^p(\Omega, \mathbb{R}^d)$ , then

$$\mathbb{E}\left[\sup_{a \leq t \leq b} |M_t|^p\right] \leq \left(\frac{p}{p-1}\right)^p \mathbb{E}[|M_b|^p].$$

**Theorem 2.56 (Decomposition Theorem).** Let  $\{A_t\}_{t \geq 0}$  and  $\{U_t\}_{t \geq 0}$  be two continuous adapted increasing processes with  $A_0 = U_0 = 0$  almost surely. Let  $\{M_t\}_{t \geq 0}$  be a real-valued continuous local martingale with  $M_0 = 0$  almost surely. Let  $\xi$  be a nonnegative  $\mathcal{F}_0$ -measurable random variable. Define

$$X_t = \xi + A_t - U_t + M_t \quad \text{for } t \geq 0.$$

If  $X_t$  is nonnegative, then

$$\left\{ \lim_{t \rightarrow \infty} A_t < \infty \right\} \cap \left\{ \lim_{t \rightarrow \infty} X_t \text{ exists and is finite} \right\} \cap \left\{ \lim_{t \rightarrow \infty} U_t < \infty \right\} \quad \text{almost surely,}$$

where  $B \subset D$  almost surely means  $\mathbf{P}(B \cap D^c) = 0$ . In particular, if  $\lim_{t \rightarrow \infty} A_t < \infty$  almost surely, then for almost every  $\omega \in \Omega$ ,

$$\lim_{t \rightarrow \infty} X_t(\omega) \text{ exists and is finite, and } \lim_{t \rightarrow \infty} U_t(\omega) < \infty.$$

### 2.1.3 Lévy, Poisson, and Compound Poisson Processes

**Definition 2.57 (Lévy Process).** A stochastic process  $X = (X_t)_{t \geq 0}$  defined on a probability space  $(\Omega, \mathcal{F}, \mathbf{P})$  is called a Lévy process if it satisfies the following properties

1. **Null initial value:**  $X_0 = 0$  almost surely.
2. **Independent increments:** For any  $0 \leq t_1 < t_2 < \dots < t_n$ , the random variables  $X_{t_2} - X_{t_1}, \dots, X_{t_n} - X_{t_{n-1}}$  are independent.
3. **Stationary increments:**  $X_t - X_s \stackrel{d}{=} X_{t-s}$  for all  $0 \leq s < t$ .
4. **Càdlàg paths:** The sample paths  $t \mapsto X_t(\omega)$  are right-continuous with left limits almost surely.
5. **Stochastic continuity:**  $\lim_{s \rightarrow t} \mathbf{P}(|X_t - X_s| > \varepsilon) = 0$  for all  $\varepsilon > 0$  and  $t \geq 0$ .

**Theorem 2.58 (Lévy-Khintchine Representation).** The characteristic function of a Lévy process  $X_t$  can be expressed as  $\mathbb{E}[e^{iuX_t}] = e^{t\eta(u)}$ , where the Lévy exponent  $\eta: \mathbb{R} \rightarrow \mathbb{C}$  has the form

$$\eta(u) = ibu - \frac{1}{2}\sigma^2 u^2 + \int_{\mathbb{R} \setminus \{0\}} (e^{iux} - 1 - iux \mathbb{1}_{\{|x|<1\}}) \nu(dx),$$

where  $b \in \mathbb{R}$  is the drift coefficient,  $\sigma \geq 0$  is the diffusion coefficient and  $\nu$  is the Lévy measure satisfying  $\int_{\mathbb{R}} (1 \wedge x^2) \nu(dx) < \infty$ .

**Definition 2.59 (Poisson Process).** A Poisson process with intensity  $\lambda > 0$  is a counting process  $(N_t)_{t \geq 0}$  satisfying

1.  $N_0 = 0$  almost surely,
2. Independent increments,
3. For  $0 \leq s < t$ , the increment  $N_t - N_s$  follows a Poisson distribution

$$\mathbf{P}(N_t - N_s = k) = \frac{(\lambda(t-s))^k}{k!} e^{-\lambda(t-s)}, \quad k \in \mathbb{N}_0.$$

For a time-dependent intensity  $\lambda_t > 0$ , the probability mass function becomes

$$\mathbf{P}(N_t - N_s = k) = \frac{\left( \int_s^t \lambda_u du \right)^k}{k!} \exp \left( - \int_s^t \lambda_u du \right).$$

**Definition 2.60 (Compound Poisson Process).** A stochastic process  $(Y_t)_{t \geq 0}$  is called a compound Poisson process if it satisfies

1. **Poisson counting basis:**  $(N_t)_{t \geq 0}$  is a Poisson process with intensity  $\lambda > 0$ ,
2. **Independent jumps:**  $(\xi_k)_{k \geq 1}$  are i.i.d. random variables with common distribution  $F$ ,
3. **Independence:**  $(\xi_k)$  and  $(N_t)$  are independent,
4. **Compound structure:**  $Y_t = \sum_{k=1}^{N_t} \xi_k$ ,  $Y_0 = 0$  a.s.,
5. **Jump times:**  $\tau_k = \inf\{t \geq 0, N_t \geq k\}$  are the arrival times.

**Remark 2.61 (Relationships).** The compound Poisson process connects several fundamental concepts

- **Link to Poisson process:** When  $\xi_k \equiv 1$ ,  $Y_t$  reduces to  $N_t$ ,
- **Moment generating function:**  $\mathbb{E}[e^{uY_t}] = \exp(\lambda t(\mathbb{E}[e^{u\xi_1}] - 1))$ ,
- **Lévy measure connection:**  $\nu(dx) = \lambda F(dx)$ , where  $\nu$  is the Lévy measure,
- **Increments:**  $Y_t - Y_s \stackrel{d}{=} \sum_{k=1}^{N_{t-s}} \xi_k$  for  $0 \leq s < t$ ,
- **Martingale property:**  $Y_t - \lambda t \mathbb{E}[\xi_1]$  is a martingale.

**Definition 2.62 (Poisson Random Measure).** For a given Lévy process  $X$ , the Poisson random measure on  $\mathbb{R}_+ \times \mathbb{R} \setminus \{0\}$  is defined as

$$N(\omega; dt, dx) = \sum_{s>0} \mathbb{1}_{\{\Delta X_s(\omega) \neq 0\}} \delta_{(s, \Delta X_s(\omega))}(dt, dx),$$

where  $\Delta X_s = X_s - X_{s-}$  denotes the jump at time  $s$ . Its compensated version is given by

$$\tilde{N}(dt, dx) = N(dt, dx) - \nu(dx)dt,$$

where  $\nu$  represents the Lévy measure of the process.

**Theorem 2.63 (Lévy-Itô Decomposition).** Every Lévy process  $X_t$  admits the following decomposition:

$$X_t = bt + \sigma B_t + Z_t + M_t,$$

where

$$\begin{aligned} Z_t &= \int_{[0,t] \times \{|x|>1\}} x N(ds, dx), \\ M_t &= \lim_{\epsilon \downarrow 0} \int_{[0,t] \times \{\epsilon < |x| \leq 1\}} x \tilde{N}(ds, dx). \end{aligned}$$

Here,  $(B_t)_{t \geq 0}$  is a standard Brownian motion,  $Z_t$  captures all large jumps (with  $|x| > 1$ ), and  $M_t$  arises as the limit of compensated small jumps (with  $0 < |x| \leq 1$ ).

**Proposition 2.64 (Martingale Property).** The small jump component

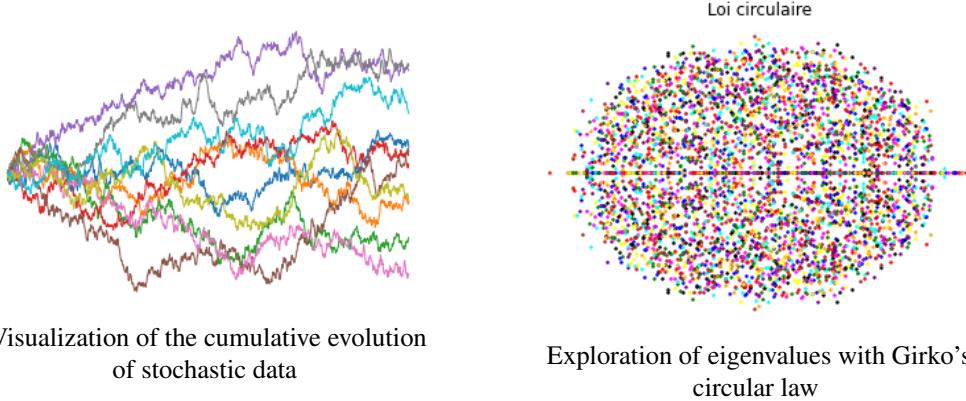
$$M_t^\epsilon = \int_{[0,t] \times \{\epsilon < |x| \leq 1\}} x \tilde{N}(ds, dx),$$

satisfies

$$\mathbb{E}[(M_t^\epsilon - M_t^{\epsilon'})^2] = t \int_{\epsilon'}^{\epsilon} x^2 \nu(dx), \quad 0 < \epsilon' < \epsilon \leq 1,$$

and converges in  $\mathbb{M}^2(\mathbf{P})$  as  $\epsilon \downarrow 0$  to  $M_t$ .

- For any Lévy process  $\sum_{s \leq t} |\Delta X_s|^2 < \infty$  almost surely, but  $\sum_{s \leq t} |\Delta X_s|$  may be infinite.
- The Lévy triplet  $(b, \sigma^2, \nu)$  completely characterizes the law of the process.
- Poisson random measures satisfy  $N(A) \sim \text{Pois}(\nu \otimes \lambda(A))$  for  $A \in \mathcal{B}(\mathbb{R}_+ \times \mathbb{R} \setminus \{0\})$ .
- The stable processes  $\alpha$  satisfy the scaling property  $X_t \sim t^{1/\alpha} X_1$  for  $\alpha \in (0, 2]$ .



**Figure 2.1.** The two plots were generated using Python scripts (see listings A.7 and A.8 in the appendix).

## 2.1.4 Brownian Motion

Brownian motion describes the random motion of particles in a fluid, first observed by Robert Brown in 1827. Mathematically, a standard Brownian motion  $\{B_t\}_{t \geq 0}$  is a stochastic process with independent increments and continuous sample paths almost surely. This fundamental process appears throughout stochastic analysis and applied mathematics.

**Definition 2.65 (Standard One-Dimensional Brownian Motion).** Let  $(\Omega, \mathcal{F}, \mathbf{P})$  be a probability space with filtration  $\{\mathcal{F}_t\}_{t \geq 0}$ . A standard one-dimensional Brownian motion is a real-valued stochastic process  $\{B_t\}_{t \geq 0}$  satisfying the following properties:

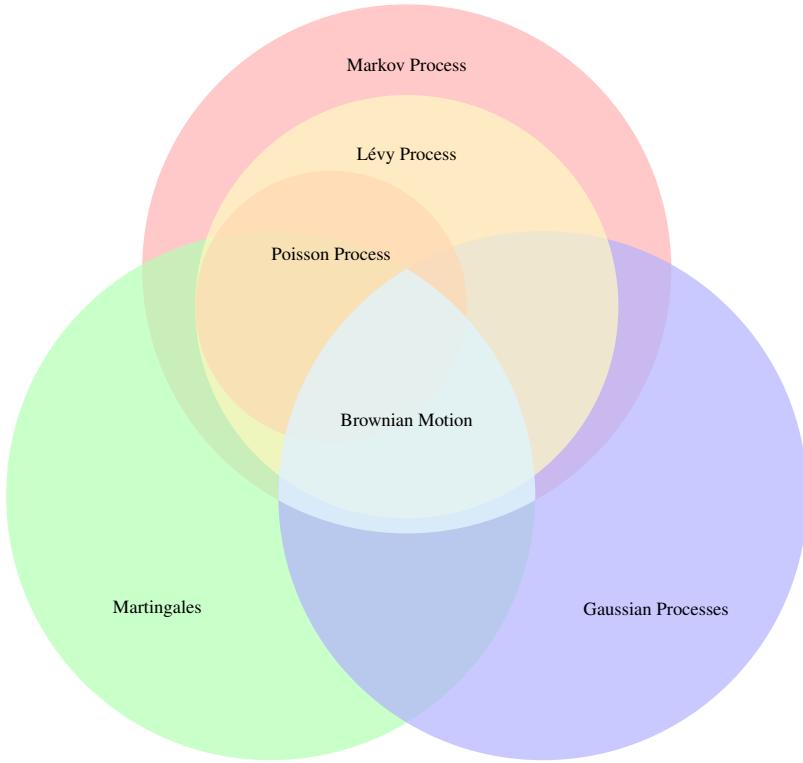
- $B_0 = 0$  almost surely,
- $B_t$  has continuous sample paths almost surely,
- For  $0 \leq s < t$ , the increment  $B_t - B_s$  is normally distributed with mean 0 and variance  $t - s$ , i.e.,  $B_t - B_s \sim \mathcal{N}(0, t - s)$ ,
- For all  $s < t$ , the increment  $B_t - B_s$  is independent of  $\mathcal{F}_s$ .

**Remark 2.66.** The definition extends naturally to finite time intervals  $[0, T]$  for any  $T > 0$  by restricting the process to  $t \in [0, T]$ .

**Proposition 2.67 (Increment Properties of Brownian Motion).** For a standard one-dimensional Brownian motion  $\{B_t\}_{t \geq 0}$  and any  $0 \leq t_0 < t_1 < \dots < t_k$ , the following properties hold:

- The increments  $B_{t_i} - B_{t_{i-1}}$  for  $i = 1, \dots, k$  are independent (independent increments property),
- Each increment  $B_{t_i} - B_{t_{i-1}}$  follows a normal distribution with mean 0 and variance  $t_i - t_{i-1}$ , i.e.,  $B_{t_i} - B_{t_{i-1}} \sim \mathcal{N}(0, t_i - t_{i-1})$  (stationary increments property).

**Definition 2.68 (Natural Filtration).** The **natural filtration** of  $\{B_t\}$  is  $\mathcal{F}_t^B = \sigma(B_s ; 0 \leq s \leq t)$ .



**Figure 2.2.** Diagram showing the hierarchical relationships between different classes of stochastic processes. The Venn diagram illustrates how Brownian motion sits at the intersection of Markov processes, martingales, and Gaussian processes, while also being a special case of Lévy processes. Poisson processes are shown as a subset of both Lévy and Markov processes.

**Lemma 2.69 (Filtration Extension).** If  $\{\mathcal{F}_t\}$  satisfies  $\mathcal{F}_t^B \subset \mathcal{F}_t$  and  $B_t - B_s \perp\!\!\!\perp \mathcal{F}_s$  for  $s < t$ , then  $\{B_t\}$  remains a Brownian motion with respect to  $\{\mathcal{F}_t\}$ .

**Theorem 2.70 (Completion of Space).** The completion  $(\Omega, \overline{\mathcal{F}}, \mathbf{P})$  preserves Brownian motion properties, where

$$\overline{\mathcal{F}}_t = \sigma(\mathcal{F}_t^B \cup \mathcal{N}), \quad \mathcal{N} = \{A \in \overline{\mathcal{F}} ; \mathbf{P}(A) = 0\}.$$

**Definition 2.71 (Augmented Filtration).** The augmented filtration  $\{\overline{\mathcal{F}}_t\}$  satisfies

- The usual conditions (right-continuous, complete).
- $\{B_t\}$  remains Brownian with respect to  $\{\overline{\mathcal{F}}_t\}$ .

**Remark 2.72.** Unless specified otherwise, we work with complete probability spaces and filtrations that satisfy the usual conditions.

**Proposition 2.73 (Fundamental Properties).** For a 1D Brownian motion  $\{B_t\}$

1.  $\{-B_t\}$  is a Brownian motion (symmetry),
2.  $X_t = c^{-1/2}B_{ct}$  is Brownian for  $c > 0$  (scaling property),
3.  $\{B_t\}$  is a square-integrable martingale with  $\langle B \rangle_t = t$ ,

4. Strong Law  $\lim_{t \rightarrow \infty} \frac{B_t}{t} = 0$  a.s.,
5. Sample paths are nowhere differentiable a.s.,
6. Path regularity
  - (a) Hölder continuous for  $\delta \in (0, 1/2)$ ,
  - (b) Nowhere Hölder for  $\delta > 1/2$ .

**Theorem 2.74 (Law of the Iterated Logarithm).** Almost surely, the following limits hold:

$$\begin{aligned} \blacksquare \quad & \limsup_{t \downarrow 0} \frac{B_t}{\sqrt{2t \log \log(1/t)}} = 1, & \blacksquare \quad & \limsup_{t \rightarrow \infty} \frac{B_t}{\sqrt{2t \log \log t}} = 1, \\ \blacksquare \quad & \liminf_{t \downarrow 0} \frac{B_t}{\sqrt{2t \log \log(1/t)}} = -1, & \blacksquare \quad & \liminf_{t \rightarrow \infty} \frac{B_t}{\sqrt{2t \log \log t}} = -1. \end{aligned}$$

**Corollary 2.75 (Path Bounds).** For any  $\varepsilon > 0$ , there exists a random variable  $\rho_\varepsilon > 0$  such that, almost surely,

$$-(1 + \varepsilon) \sqrt{2t \log \log t} \leq B_t \leq (1 + \varepsilon) \sqrt{2t \log \log t}, \quad \forall t \geq \rho_\varepsilon.$$

Moreover, for any  $0 < \epsilon < 1$ , almost every sample path exceeds the bounds

$$-(1 - \epsilon) \sqrt{2t \log \log t} \quad \text{and} \quad (1 - \epsilon) \sqrt{2t \log \log t},$$

infinitely often as  $t \rightarrow \infty$ .

**Definition 2.76 ( $d$ -Dimensional Brownian Motion).** A stochastic process  $B_t = (B_t^1, \dots, B_t^d)_{t \geq 0}$  is called a  $d$ -dimensional Brownian motion if:

- Each component  $\{B_t^i\}_{t \geq 0}$  is a standard one-dimensional Brownian motion,
- The components  $\{B_t^1\}, \dots, \{B_t^d\}$  are mutually independent.

**Theorem 2.77 (Asymptotic Behavior).** For a  $d$ -dimensional Brownian motion  $B_t$ , the following holds:

$$\limsup_{t \rightarrow \infty} \frac{\|B_t\|}{\sqrt{2t \log \log t}} = 1 \quad \text{almost surely,}$$

where  $\|\cdot\|$  denotes the Euclidean norm.

**Remark 2.78.** This result shows that the components cannot simultaneously achieve their maximal growth rate, as this would yield  $\sqrt{d}$  instead of 1 in the limit.

**Proposition 2.79 (Martingale Properties of Brownian Motion).** A  $d$ -dimensional Brownian motion  $B_t = (B_t^1, \dots, B_t^d)_{t \geq 0}$  is a continuous martingale with quadratic variation given by

$$\langle B^i, B^j \rangle_t = \delta_{ij} t \quad \text{for } 1 \leq i, j \leq d,$$

where  $\delta_{ij}$  is the Kronecker delta, defined as

$$\delta_{ij} = \begin{cases} 1 & \text{if } i = j, \\ 0 & \text{otherwise.} \end{cases}$$

**Theorem 2.80 (Lévy's Characterization Theorem).** Let  $M_t = (M_t^1, \dots, M_t^d)_{t \geq 0}$  be a continuous  $d$ -dimensional local martingale with  $M_0 = \mathbf{0}$  almost surely. If the quadratic variation satisfies

$$\langle M^i, M^j \rangle_t = \delta_{ij} t \quad \text{for } 1 \leq i, j \leq d,$$

then  $M_t$  is a  $d$ -dimensional Brownian motion.

**Theorem 2.81 (Time-Change of Martingales).** Let  $M = \{M_t\}_{t \geq 0}$  be a continuous real-valued local martingale with  $M_0 = 0$  and  $\lim_{t \rightarrow \infty} \langle M \rangle_t = \infty$  almost surely. Define the stopping time

$$\tau_t = \inf\{s > 0 : \langle M \rangle_s > t\}.$$

Then the time-changed process  $\{M_{\tau_t}\}_{t \geq 0}$  is a standard one-dimensional Brownian motion.

## 2.2 Stochastic Integral

### 2.2.1 Preliminaries and Definitions

The stochastic integral with respect to Brownian motion cannot be defined pathwise since Brownian paths are almost surely nowhere differentiable. Kiyoshi Itô first rigorously defined this integral in 1949, establishing what is now known as the Itô stochastic integral. We develop this theory systematically below. Let  $(\Omega, \mathcal{F}, \mathbf{P})$  be a complete probability space with filtration  $\{\mathcal{F}_t\}_{t \geq 0}$  satisfying the usual conditions, and let  $\{B_t\}_{t \geq 0}$  be a one-dimensional  $\{\mathcal{F}_t\}$ -adapted Brownian motion.

**Definition 2.82 (Space of Integrable Processes).** For  $0 \leq a < b < \infty$ , define  $\mathbb{M}^2([a, b], \mathbb{R})$  as the space of all real-valued  $\{\mathcal{F}_t\}$ -adapted measurable processes  $f = \{f(t)\}_{a \leq t \leq b}$  satisfying

$$\|f\|_{a,b}^2 = \mathbb{E} \left( \int_a^b |f(t)|^2 dt \right) < \infty. \quad (2.5)$$

We identify processes  $f$  and  $\tilde{f}$  in  $\mathbb{M}^2([a, b], \mathbb{R})$  when  $\|f - \tilde{f}\|_{a,b}^2 = 0$ , writing  $f \equiv \tilde{f}$ .

**Remark 2.83.** The space  $(\mathbb{M}^2([a, b], \mathbb{R}), \|\cdot\|_{a,b})$  is complete. Every  $f \in \mathbb{M}^2([a, b], \mathbb{R})$  has a predictable modification  $\tilde{f}$  with  $f \equiv \tilde{f}$ , given by

$$\tilde{f}(t) = \limsup_{h \downarrow 0} \frac{1}{h} \int_{t-h}^t \hat{f}(s) ds,$$

where  $\hat{f}$  is a progressively measurable modification.

**Definition 2.84 (Simple Processes).** A process  $g = \{g(t)\}_{a \leq t \leq b}$  is simple if there exists a partition  $a = t_0 < t_1 < \dots < t_k = b$  and bounded  $\mathcal{F}_{t_i}$ -measurable random variables  $\xi_i$  ( $0 \leq i \leq k-1$ ) such that

$$g(t) = \xi_0 \mathbb{1}_{[t_0, t_1]}(t) + \sum_{i=1}^{k-1} \xi_i \mathbb{1}_{(t_i, t_{i+1}]}(t). \quad (2.6)$$

Denote by  $\mathbb{M}_0([a, b], \mathbb{R})$  the space of all simple processes.

**Definition 2.85 (Itô Integral for Simple Processes).** For  $g \in \mathbb{M}_0([a, b], \mathbb{R})$  given by (2.6), define its Itô integral as

$$\int_a^b g(t) dB_t = \sum_{i=0}^{k-1} \xi_i (B_{t_{i+1}} - B_{t_i}).$$

**Lemma 2.86 (Properties of Itô Integral).** *For any  $g \in \mathbb{M}_0([a, b], \mathbb{R})$ , we have*

$$\mathbb{E} \left( \int_a^b g(t) dB_t \right) = 0 \quad \text{and} \quad \mathbb{E} \left( \left| \int_a^b g(t) dB_t \right|^2 \right) = \mathbb{E} \left( \int_a^b |g(t)|^2 dt \right). \quad (2.7)$$

**Lemma 2.87 (Linearity of Itô Integral).** *For any  $g_1, g_2 \in \mathbb{M}_0([a, b], \mathbb{R})$  and real constants  $c_1, c_2$ :*

- $c_1 g_1 + c_2 g_2 \in \mathbb{M}_0([a, b], \mathbb{R})$ .
- The integral satisfies

$$\int_a^b [c_1 g_1(t) + c_2 g_2(t)] dB_t = c_1 \int_a^b g_1(t) dB_t + c_2 \int_a^b g_2(t) dB_t.$$

**Lemma 2.88 (Density of Simple Processes).** *For any  $f \in \mathbb{M}^2([a, b], \mathbb{R})$ , there exists a sequence  $\{g_n\}_{n \geq 1}$  in  $\mathbb{M}_0([a, b], \mathbb{R})$  such that*

$$\lim_{n \rightarrow \infty} \mathbb{E} \left( \int_a^b |f(t) - g_n(t)|^2 dt \right) = 0.$$

Using Lemma 2.88, for any  $f \in \mathbb{M}^2([a, b], \mathbb{R})$ , we can construct the Itô integral by approximation. Let  $\{g_n\}$  be a sequence of simple processes approximating  $f$  in the sense that

$$\lim_{n \rightarrow \infty} \mathbb{E} \left( \int_a^b |f(t) - g_n(t)|^2 dt \right) = 0.$$

By the Itô isometry (Lemma 2.86), the sequence  $\left\{ \int_a^b g_n(t) dB_t \right\}$  is Cauchy in  $\mathbf{L}^2(\Omega, \mathbb{R})$  since

$$\begin{aligned} \mathbb{E} \left( \left| \int_a^b g_n(t) dB_t - \int_a^b g_m(t) dB_t \right|^2 \right) &= \mathbb{E} \left( \int_a^b |g_n(t) - g_m(t)|^2 dt \right) \\ &\rightarrow 0 \quad \text{as } n, m \rightarrow \infty. \end{aligned}$$

**Definition 2.89 (Itô Integral).** *For  $f \in \mathbb{M}^2([a, b], \mathbb{R})$ , the Itô integral is defined as*

$$\int_a^b f(t) dB_t = \mathbf{L}^2\text{-} \lim_{n \rightarrow \infty} \int_a^b g_n(t) dB_t,$$

where  $\{g_n\}$  is any approximating sequence of simple processes.

**Theorem 2.90 (Properties of Itô Integral).** *For  $f, g \in \mathbb{M}^2([a, b], \mathbb{R})$  and  $\alpha, \beta \in \mathbb{R}$ :*

1.  $\int_a^b f(t) dB_t$  is  $\mathcal{F}_b$ -measurable,
2.  $\mathbb{E} \left( \int_a^b f(t) dB_t \right) = 0$ ,
3. *Itô Isometry:*  $\mathbb{E} \left( \left| \int_a^b f(t) dB_t \right|^2 \right) = \mathbb{E} \left( \int_a^b |f(t)|^2 dt \right)$ ,
4. *Linearity:*  $\int_a^b [\alpha f(t) + \beta g(t)] dB_t = \alpha \int_a^b f(t) dB_t + \beta \int_a^b g(t) dB_t$ .

**Theorem 2.91 (Conditional Properties).** For  $f \in \mathbb{M}^2([a, b], \mathbb{R})$ :

$$\begin{aligned}\mathbb{E}\left(\int_a^b f(t)dB_t \middle| \mathcal{F}_a\right) &= 0, \\ \mathbb{E}\left(\left|\int_a^b f(t)dB_t\right|^2 \middle| \mathcal{F}_a\right) &= \mathbb{E}\left(\int_a^b |f(t)|^2 dt \middle| \mathcal{F}_a\right) \\ &= \int_a^b \mathbb{E}(|f(t)|^2 | \mathcal{F}_a) dt.\end{aligned}$$

**Lemma 2.92 (Multiplication by  $\mathcal{F}_a$ -measurable RV).** For  $f \in \mathbb{M}^2([a, b], \mathbb{R})$  and  $\xi$  bounded  $\mathcal{F}_a$ -measurable:

$$\int_a^b \xi f(t)dB_t = \xi \int_a^b f(t)dB_t.$$

**Proposition 2.93 (Additive Property).** Let  $T > 0$  and  $f \in \mathbb{M}^2([0, T], \mathbb{R})$ . For any  $0 \leq a < b \leq T$ , the restriction  $\{f(t)\}_{a \leq t \leq b}$  belongs to  $\mathbb{M}^2([a, b], \mathbb{R})$ , making  $\int_a^b f(t)dB_t$  well-defined. The integral satisfies

$$\int_a^b f(t)dB_t + \int_b^c f(t)dB_t = \int_a^c f(t)dB_t \quad \text{for } 0 \leq a < b < c \leq T.$$

**Definition 2.94 (Indefinite Itô Integral).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$ , the indefinite Itô integral is the process

$$I(t) = \int_0^t f(s)dB_s \quad \text{for } 0 \leq t \leq T,$$

with  $I(0) = 0$  by convention.

**Theorem 2.95 (Martingale Property).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$ , the indefinite integral  $\{I(t)\}$  is:

- A square-integrable  $\{\mathcal{F}_t\}$ -martingale,
- Satisfies the maximal inequality:

$$\mathbb{E}\left[\sup_{0 \leq t \leq T} \left|\int_0^t f(s)dB_s\right|^2\right] \leq 4\mathbb{E}\left(\int_0^T |f(s)|^2 ds\right).$$

**Theorem 2.96 (Continuity).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$ , the indefinite integral  $\{I(t)\}$  has a continuous version.

**Theorem 2.97 (Quadratic Variation).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$ , the indefinite integral  $I(t)$  has quadratic variation

$$\langle I \rangle_t = \int_0^t |f(s)|^2 ds \quad \text{for } 0 \leq t \leq T.$$

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For stopping time applications, note that the indicator process  $\mathbb{1}_{[0,\tau]}(t)$  is  $\{\mathcal{F}_t\}$ -adapted, bounded, right-continuous, and predictable since

$$\{\omega; \mathbb{1}_{[0,\tau]}(t, \omega) \leq r\} \in \mathcal{F}_t \quad \text{for all } r \in \mathbb{R}.$$

**Definition 2.98 (Stopped Itô Integral).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$  and a stopping time  $\tau \leq T$ ,

$$\int_0^\tau f(s) dB_s = \int_0^T \mathbb{1}_{[0,\tau]}(s) f(s) dB_s.$$

For stopping times  $\rho \leq \tau \leq T$ ,

$$\int_\rho^\tau f(s) dB_s = \int_0^\tau f(s) dB_s - \int_0^\rho f(s) dB_s = \int_0^T \mathbb{1}_{(\rho, \tau]}(s) f(s) dB_s.$$

**Theorem 2.99 (Stopped Integral Properties).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$  and  $\rho \leq \tau \leq T$ ,

$$\begin{aligned} \mathbb{E} \left( \int_\rho^\tau f(s) dB_s \right) &= 0, \\ \mathbb{E} \left( \left| \int_\rho^\tau f(s) dB_s \right|^2 \right) &= \mathbb{E} \left( \int_\rho^\tau |f(s)|^2 ds \right). \end{aligned}$$

**Theorem 2.100 (Conditional Properties).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$  and  $\rho \leq \tau \leq T$ ,

$$\begin{aligned} \mathbb{E} \left( \int_\rho^\tau f(s) dB_s \middle| \mathcal{F}_\rho \right) &= 0, \\ \mathbb{E} \left( \left| \int_\rho^\tau f(s) dB_s \right|^2 \middle| \mathcal{F}_\rho \right) &= \mathbb{E} \left( \int_\rho^\tau |f(s)|^2 ds \middle| \mathcal{F}_\rho \right). \end{aligned}$$

**Lemma 2.101 (Stopped Integral Representation).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R})$  and a stopping time  $\tau \leq T$ ,

$$\int_0^\tau f(s) dB_s = I(\tau),$$

where  $I(t)$  is the indefinite integral from Definition 2.94.

**Corollary 2.102 (Covariance Property).** For  $f, g \in \mathbb{M}^2([0, T], \mathbb{R})$  and  $\rho \leq \tau \leq T$ ,

$$\mathbb{E} \left( \left( \int_\rho^\tau f(s) dB_s \right) \left( \int_\rho^\tau g(s) dB_s \right) \middle| \mathcal{F}_\rho \right) = \mathbb{E} \left( \int_\rho^\tau f(s)g(s) ds \middle| \mathcal{F}_\rho \right).$$

### 2.2.2 Multidimensional Itô Integral

Let  $B_t = (B_t^1, \dots, B_t^m)^\top$  be an  $m$ -dimensional Brownian motion. Define  $\mathbb{M}^2([0, T], \mathbb{R}^{d \times m})$  as the space of  $d \times m$  matrix-valued processes  $f = (f_{ij})$  with

$$\mathbb{E} \left( \int_0^T \|f(s)\|^2 ds \right) < \infty, \quad \|f\| = \sqrt{\text{trace}(f^\top f)}.$$

**Definition 2.103 (Multidimensional Itô Integral).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R}^{d \times m})$ , the integral is the  $\mathbb{R}^d$ -valued process

$$\int_0^t f(s) dB_s = \left( \sum_{j=1}^m \int_0^t f_{1j}(s) dB_s^j, \dots, \sum_{j=1}^m \int_0^t f_{dj}(s) dB_s^j \right)^\top.$$

**Theorem 2.104 (Multidimensional Properties).** For  $f \in \mathbb{M}^2([0, T], \mathbb{R}^{d \times m})$  and  $\rho \leq \tau \leq T$ ,

$$\begin{aligned} \mathbb{E} \left( \int_\rho^\tau f(s) dB_s \middle| \mathcal{F}_\rho \right) &= 0, \\ \mathbb{E} \left( \left\| \int_\rho^\tau f(s) dB_s \right\|^2 \middle| \mathcal{F}_\rho \right) &= \mathbb{E} \left( \int_\rho^\tau \|f(s)\|^2 ds \middle| \mathcal{F}_\rho \right). \end{aligned}$$

The assertions follow from

- the definition of the multidimensional Itô integral and Theorem 2.100,
- Theorem 2.100 combined with the following lemma.

**Lemma 2.105 (Orthogonality of Independent Brownian Integrals).** For two independent Brownian motions  $\{B_t^1\}$  and  $\{B_t^2\}$ , and  $f, g \in \mathbb{M}^2([0, T], \mathbb{R})$  with stopping times  $\rho \leq \tau \leq T$ ,

$$\mathbb{E} \left( \int_\rho^\tau f(s) dB_s^1 \int_\rho^\tau g(s) dB_s^2 \middle| \mathcal{F}_\rho \right) = 0.$$

### 2.2.3 Extension to Local Integrands

Let  $\mathbb{M}_{loc}^2(\mathbb{R}_+, \mathbb{R}^{d \times m})$  denote the space of all  $d \times m$  matrix-valued  $\{\mathcal{F}_t\}$ -adapted processes  $f$  satisfying

$$\int_0^T \|f(t)\|^2 dt < \infty \quad \text{a.s., } \forall T > 0,$$

where  $\|f(t)\| = \sqrt{\text{trace}(f(t)^\top f(t))}$ .

**Definition 2.106 (Local Itô Integral).** For  $f \in \mathbb{M}_{loc}^2(\mathbb{R}_+, \mathbb{R}^{d \times m})$ , define stopping times

$$\tau_n = n \wedge \inf \left\{ t \geq 0 ; \int_0^t \|f(s)\|^2 ds \geq n \right\}.$$

Then the **local Itô integral** is the continuous  $\mathbb{R}^d$ -valued process

$$I(t) = \lim_{n \rightarrow \infty} \int_0^{t \wedge \tau_n} f(s) dB_s,$$

which satisfies  $I(t \wedge \tau_n) = \int_0^{t \wedge \tau_n} f(s) dB_s$  for each  $n$ .

**Remark 2.107.** The local Itô integral is

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- A continuous local martingale.
- Independent of the choice of localizing sequence  $\{\tau_n\}$ .
- Coincidences with the standard Itô integral when  $f \in \mathbb{M}^2$ .

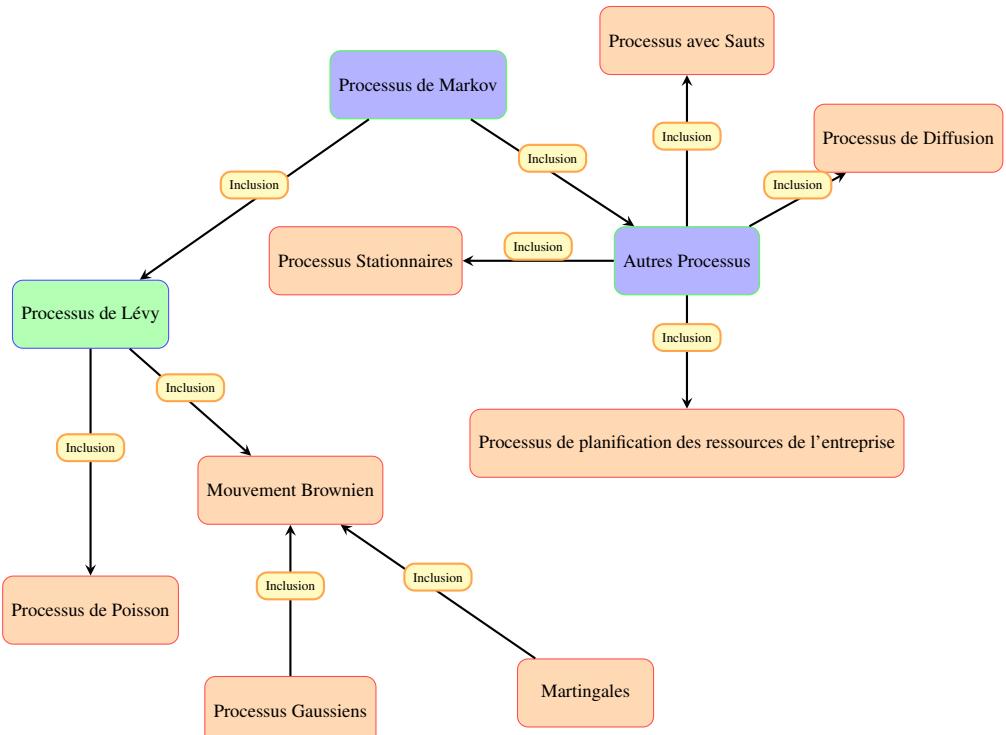
**Theorem 2.108 (Properties of Local Itô Integral).** For  $f \in \mathbb{M}_{loc}^2(\mathbb{R}_+, \mathbb{R}^{d \times m})$

1. The quadratic variation process is

$$\left\langle \int_0^\cdot f(s) dB_s \right\rangle_t = \int_0^t \|f(s)\|^2 ds.$$

2. For stopping times  $\rho \leq \tau$

$$\begin{aligned} \mathbb{E} \left( \int_\rho^\tau f(s) dB_s \middle| \mathcal{F}_\rho \right) &= 0, \\ \mathbb{E} \left( \left\| \int_\rho^\tau f(s) dB_s \right\|^2 \middle| \mathcal{F}_\rho \right) &= \mathbb{E} \left( \int_\rho^\tau \|f(s)\|^2 ds \middle| \mathcal{F}_\rho \right). \end{aligned}$$



**Figure 2.3.** Hierarchical diagram showing the inclusion relationships between different types of stochastic processes. The arrows indicate the inclusion relationships between process classes, with the Markov process as the general class encompassing Lévy processes and other types. Brownian motion appears as an important special case at the intersection of several classes.

## 2.2.4 Itô's Formula

Itô's formula serves as the fundamental theorem of stochastic calculus, analogous to the chain rule in classical calculus. While Lebesgue integration relies on the fundamental theorem of calculus for computations, stochastic integration requires Itô's formula due to the non-differentiable nature of Brownian motion.

**Definition 2.109 (Itô Process).** A one-dimensional Itô process is a continuous, adapted stochastic process  $\{x(t)\}_{t \geq 0}$  of the form

$$x(t) = x(0) + \int_0^t f(s) ds + \int_0^t g(s) dB_s,$$

where  $f \in \mathbf{L}^1(\mathbb{R}_+, \mathbb{R})$  (drift coefficient) and  $g \in \mathbf{L}^2(\mathbb{R}_+, \mathbb{R})$  (diffusion coefficient). We express this in differential form as

$$dx(t) = f(t) dt + g(t) dB_t.$$

**Definition 2.110 (Function Space).** Let  $\mathcal{C}^{2,1}(\mathbb{R}^d \times \mathbb{R}_+)$  denote the space of functions  $V(x, t)$  that are twice continuously differentiable in  $x \in \mathbb{R}^d$  and once continuously differentiable in  $t \in \mathbb{R}_+$ . For such functions, we define

$$\begin{aligned} V_t &= \frac{\partial V}{\partial t}, \quad V_x = \left( \frac{\partial V}{\partial x_1}, \dots, \frac{\partial V}{\partial x_d} \right), \\ V_{xx} &= \left( \frac{\partial^2 V}{\partial x_i \partial x_j} \right)_{d \times d}. \end{aligned}$$

**Theorem 2.111 (One-dimensional Itô's Formula).** Let  $x(t)$  be an Itô process with  $dx(t) = f(t) dt + g(t) dB_t$ . For  $V \in \mathcal{C}^{2,1}(\mathbb{R} \times \mathbb{R}_+)$ ,

$$\begin{aligned} dV(x(t), t) &= \underbrace{\left[ V_t(x(t), t) + V_x(x(t), t)f(t) + \frac{1}{2}V_{xx}(x(t), t)g^2(t) \right] dt}_{\text{Temporal derivative and diffusion terms}} \\ &\quad + \underbrace{V_x(x(t), t)g(t) dB_t}_{\text{Stochastic term}} \quad a.s.. \end{aligned}$$

**Definition 2.112 (Multidimensional Itô Process).** A  $d$ -dimensional Itô process  $\{x(t)\}_{t \geq 0}$  in  $\mathbb{R}^d$  has the form

$$x(t) = x(0) + \int_0^t f(s) ds + \int_0^t g(s) dB(s),$$

where  $f \in \mathbf{L}^1(\mathbb{R}_+, \mathbb{R}^d)$  and  $g \in \mathbf{L}^2(\mathbb{R}_+, \mathbb{R}^{d \times m})$ , with differential form

$$dx(t) = f(t) dt + g(t) dB(t).$$

**Theorem 2.113 (Multidimensional Itô's Formula).** For a  $d$ -dimensional Itô process  $x(t)$  and

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$V \in \mathcal{C}^{2,1}(\mathbb{R}^d \times \mathbb{R}_+)$ ,

$$\begin{aligned} dV(x(t), t) &= \underbrace{\left[ V_t(x(t), t) + V_x(x(t), t)f(t) + \frac{1}{2} \text{trace} (g^T(t)V_{xx}(x(t), t)g(t)) \right] dt}_{\text{Temporal derivative, drift, and diffusion terms (multivariate form)}} \\ &\quad + \underbrace{V_x(x(t), t)g(t) dB(t)}_{\text{Stochastic term (Brownian motion)}} \quad a.s.. \end{aligned}$$

**Remark 2.114 (Itô's Multiplication Table).** *The stochastic differential calculus follows these rules for  $(i \neq j)$*

$\times$	$dt$	$dB_i$	$dB_j$
$dt$	0	0	0
$dB_i$	0	$dt$	0
$dB_j$	0	0	$dt$

**Table 2.1.** Multiplication Rules for Stochastic Differentials

- For  $i = j$   $dB_i \cdot dB_i = dt$  (Itô's quadratic variation).
- For  $i \neq j$   $dB_i \cdot dB_j = 0$  (independent Brownian motions).
- All products involving  $dt$  vanish  $dt^2 = 0$  and  $dt \cdot dB_i = 0$ .

This leads to the cross-variation formula

$$dx_i(t)dx_j(t) = \sum_{k=1}^m g_{ik}(t)g_{jk}(t)dt.$$

**Theorem 2.115 (Stochastic Integration by Parts).** *Let  $x(t)$  be an Itô process and  $y(t)$  be a finite variation process. Then, the following stochastic integration by parts formula holds:*

$$d[x(t)y(t)] = y(t)dx(t) + x(t)dy(t).$$

In integral form, we have

$$x(t)y(t) - x(0)y(0) = \int_0^t y(s)dx(s) + \int_0^t x(s)dy(s),$$

where the second integral is understood in the Lebesgue-Stieltjes sense.

Process	Dynamics	Itô's Formula
General case with jumps	$dX_t = \mu_t dt + \sigma_t dB_t + dJ_t$ $J_t$ jump process	$df(t, X_t) = \left( \partial_t f + \mu_t \partial_x f + \frac{1}{2} \sigma_t^2 \partial_{xx} f \right) dt + \sigma_t \partial_x f dB_t + [f(t, X_t) - f(t, X_{t-})] dN_t + \int_{\mathbb{R}} [f(t, X_{t-} + z) - f(t, X_{t-}) - z \partial_x f] \tilde{N}(dt, dz)$
Itô diffusion	$dX_t = \mu(t, X_t) dt + \sigma(t, X_t) dB_t$	$df = \left( \partial_t f + \mu \partial_x f + \frac{1}{2} \sigma^2 \partial_{xx} f \right) dt + \sigma \partial_x f dB_t$
Brownian motion	$dX_t = dB_t$	$df = \partial_t f dt + \partial_x f dB_t + \frac{1}{2} \partial_{xx} f dt$
GBM (Geometric Brownian Motion)	$dS_t = \mu S_t dt + \sigma S_t dB_t$	$df = \left( \partial_t f + \mu S \partial_S f + \frac{1}{2} \sigma^2 S^2 \partial_{SS} f \right) dt + \sigma S \partial_S f dB_t$
Poisson process	$dN_t$ jumps of size 1 $\lambda$ intensity	$df(t, N_t) = \partial_t f dt + [f(t, N_{t-} + 1) - f(t, N_{t-})] dN_t$
Lévy process	$dL_t = \gamma dt + \sigma dB_t + \int_{ z <1} z \tilde{N}(dt, dz) + \int_{ z \geq 1} z N(dt, dz)$	$df(t, L_t) = \left( \partial_t f + \gamma \partial_x f + \frac{1}{2} \sigma^2 \partial_{xx} f \right) dt + \sigma \partial_x f dB_t + \int_{\mathbb{R}} [f(t, L_{t-} + z) - f(t, L_{t-}) - z \mathbb{1}_{ z <1} \partial_x f] N(dt, dz)$

Table 2.2. Itô's formulas for different stochastic processes (continuous and jump cases)

- $\nu(dz, dt)$  is the compensated jump measure .
- Brownian motion is a special case of an Itô diffusion with  $\mu = 0$  and  $\sigma = 1$ .
- GBM (Geometric Brownian Motion) is widely used in finance for asset price modeling.
- $N_t$  is a pure counting process without a continuous component .
- $\tilde{N}(dt, dz) = N(dt, dz) - \nu(dz)dt$  is the compensated Poisson random measure .
- Notation:

$$\partial_t f \equiv \frac{\partial f}{\partial t}, \quad \partial_x f \equiv \frac{\partial f}{\partial x}, \quad \partial_{xx} f \equiv \frac{\partial^2 f}{\partial x^2}, \quad \partial_S f \equiv \frac{\partial f}{\partial S}, \quad \partial_{SS} f \equiv \frac{\partial^2 f}{\partial S^2}.$$

## 2.2.5 Moment Inequalities

This section establishes fundamental moment inequalities for stochastic integrals using Itô's formula, demonstrating its powerful applications. Let  $\{B(t)\}_{t \geq 0} = (B_1(t), \dots, B_m(t))^{\top}$  be a  $m$  dimensional Brownian motion defined on a complete probability space  $(\Omega, \mathcal{F}, \mathbf{P})$  with filtration  $\{\mathcal{F}_t\}_{t \geq 0}$ .

**Theorem 2.116 (Moment Inequality).** For  $p \geq 2$  and  $g \in \mathbb{M}^2([0, T], \mathbb{R}^{d \times m})$  satisfying

$$\mathbb{E} \left( \int_0^T \|g(s)\|^p ds \right) < \infty,$$

the following inequality holds

$$\mathbb{E} \left( \left\| \int_0^T g(s) dB(s) \right\|^p \right) \leq \left( \frac{p(p-1)}{2} \right)^{\frac{p}{2}} T^{\frac{p-2}{2}} \mathbb{E} \left( \int_0^T \|g(s)\|^p ds \right). \quad (2.8)$$

Equality holds when  $p = 2$ .

**Proposition 2.117 (Maximal Moment Inequality).** *Under the same conditions as Theorem 2.116, we have*

$$\mathbb{E} \left( \sup_{0 \leq t \leq T} \left\| \int_0^t g(s) dB(s) \right\|^p \right) \leq \left( \frac{p^3}{2(p-1)} \right)^{p/2} T^{\frac{p-2}{2}} \mathbb{E} \left( \int_0^T \|g(s)\|^p ds \right).$$

**Theorem 2.118 (Burkholder-Davis-Gundy Inequality).** *Let  $g \in \mathbf{L}^2(\mathbb{R}_+, \mathbb{R}^{d \times m})$ , and define*

$$x(t) = \int_0^t g(s) dB(s) \quad \text{and} \quad A(t) = \int_0^t \|g(s)\|^2 ds.$$

For any  $p > 0$ , there exist constants  $c_p, C_p > 0$  (depending only on  $p$ ) such that

$$c_p \mathbb{E}(|A(t)|^{p/2}) \leq \mathbb{E} \left( \sup_{0 \leq s \leq t} \|x(s)\|^p \right) \leq C_p \mathbb{E}(|A(t)|^{p/2}).$$

The constants can be specified as

$$\begin{aligned} 0 < p < 2 & \quad c_p = (p/2)^p, \quad C_p = (32/p)^{p/2}, \\ p = 2 & \quad c_p = 1, \quad C_p = 4, \\ p > 2 & \quad c_p = (2p)^{-p/2}, \quad C_p = \left( \frac{p^{p+1}}{2(p-1)^{p-1}} \right)^{p/2}. \end{aligned}$$

**Theorem 2.119 (Exponential Martingale Inequality).** *Let  $g \in \mathbf{L}^2(\mathbb{R}_+, \mathbb{R}^{1 \times m})$  and the positive constants  $T, \alpha, \beta$ . Then, we have*

$$\mathbf{P} \left( \sup_{0 \leq t \leq T} \left[ \int_0^t g(s) dB(s) - \frac{\alpha}{2} \int_0^t \|g(s)\|^2 ds \right] > \beta \right) \leq e^{-\alpha\beta}.$$

## 2.2.6 Gronwall-Type Inequalities

The integral inequalities of Gronwall type have been widely applied in the theory of ordinary differential equations and stochastic differential equations to prove results on existence, uniqueness, boundedness, comparison, continuous dependence, perturbation, and stability, among others. Naturally, Gronwall inequalities will play an important role in this work. For the reader's convenience, we present several well-known inequalities of this type in this subsection.

**Theorem 2.120 (Classical Gronwall Inequality).** *Let  $T > 0$  and  $c \geq 0$ . Let  $u(\cdot)$  be a Borel measurable, bounded, nonnegative function on  $[0, T]$ , and let  $v(\cdot)$  be a nonnegative integrable function on  $[0, T]$ . If*

$$u(t) \leq c + \int_0^t v(s) u(s) ds \quad \text{for all } 0 \leq t \leq T,$$

then

$$u(t) \leq c \exp \left( \int_0^t v(s) ds \right) \quad \text{for all } 0 \leq t \leq T.$$

**Theorem 2.121 (Bihari's Inequality).** Let  $T > 0$  and  $c > 0$ . Let  $K : \mathbb{R}_+ \rightarrow \mathbb{R}_+$  be a continuous non-decreasing function such that  $K(t) > 0$  for all  $t > 0$ . Let  $u : [0, T] \rightarrow \mathbb{R}_+$  be a bounded nonnegative Borel-measurable function, and let  $v : [0, T] \rightarrow \mathbb{R}_+$  be a nonnegative integrable function. If

$$u(t) \leq c + \int_0^t v(s) K(u(s)) ds \quad \text{for all } t \in [0, T],$$

then

$$u(t) \leq G^{-1} \left( G(c) + \int_0^t v(s) ds \right),$$

for all  $t \in [0, T]$  such that

$$G(c) + \int_0^t v(s) ds \in \text{Dom}(G^{-1}).$$

Here,  $G$  is defined by

$$G(r) = \int_1^r \frac{ds}{K(s)} \quad \text{for } r > 0,$$

and  $G^{-1}$  denotes the inverse function of  $G$ . The domain of  $G^{-1}$ ,  $\text{Dom}(G^{-1})$ , is the range of  $G$ .

**Theorem 2.122 (Nonlinear Gronwall-Type Inequality).** Let  $T > 0$ ,  $\alpha \in [0, 1)$ , and  $c > 0$ . Let  $u(\cdot)$  be a Borel measurable, bounded, nonnegative function on  $[0, T]$ , and let  $v(\cdot)$  be a nonnegative integrable function on  $[0, T]$ . If

$$u(t) \leq c + \int_0^t v(s)[u(s)]^\alpha ds \quad \text{for all } 0 \leq t \leq T,$$

then

$$u(t) \leq \left( c^{1-\alpha} + (1-\alpha) \int_0^t v(s) ds \right)^{\frac{1}{1-\alpha}},$$

holds for all  $t \in [0, T]$ .

## 2.3 Stochastic Differential Equations

### 2.3.1 Introduction

In this section, we focus on finding solutions to stochastic differential equations. More precisely, we investigate solutions to the nonlinear stochastic differential equation

$$dx(t) = f(x(t), t) dt + g(x(t), t) dB(t), \quad t \in [t_0, T], \tag{2.9}$$

with the initial condition  $x(t_0) = x_0$ , where  $0 \leq t_0 < T < \infty$ . The key questions we address are the following.

- What constitutes a solution?
- Do existence and uniqueness theorems hold for such solutions?
- What properties does the solution possess?
- How can the solution be obtained in practice?

We systematically explore these questions. Additionally, as an important application of stochastic differential equations, we derive the well-known **Feynman-Kac formula**, which provides a stochastic representation of the solution to a linear parabolic partial differential equation in terms of the solution to the corresponding stochastic differential equation.

Let  $(\Omega, \mathcal{F}, \mathbf{P})$  be a complete probability space equipped with a filtration  $\{\mathcal{F}_t\}_{t \geq 0}$  that satisfies the usual conditions.

Throughout this section, unless otherwise specified,  $B(t) = (B_1(t), \dots, B_m(t))^\top$  denotes an  $m$ -dimensional Brownian motion defined on this space. Let  $0 \leq t_0 < T < \infty$ , and let  $x_0$  be an  $\mathcal{F}_{t_0}$ -measurable  $\mathbb{R}^d$ -valued random variable such that  $\mathbb{E}[|x_0|^2] < \infty$ . Consider the Borel measurable functions  $f : \mathbb{R}^d \times [t_0, T] \rightarrow \mathbb{R}^d$  and  $g : \mathbb{R}^d \times [t_0, T] \rightarrow \mathbb{R}^{d \times m}$ .

By the definition of the stochastic differential, equation (2.9) is equivalent to the stochastic integral equation

$$x(t) = x_0 + \int_{t_0}^t f(x(s), s) ds + \int_{t_0}^t g(x(s), s) dB(s), \quad t_0 \leq t \leq T. \quad (2.10)$$

**Definition 2.123.** An  $\mathbb{R}^d$ -valued stochastic process  $\{x(t)\}_{t_0 \leq t \leq T}$  is called a solution to equation (2.9) if it satisfies the following properties:

1.  $\{x(t)\}$  is continuous and  $\mathcal{F}_t$ -adapted,
2.  $\{f(x(t), t)\} \in \mathbf{L}^1([t_0, T], \mathbb{R}^d)$  and  $\{g(x(t), t)\} \in \mathbf{L}^2([t_0, T], \mathbb{R}^{d \times m})$ ,
3. Equation (2.10) holds for every  $t \in [t_0, T]$  with probability 1.

A solution  $\{x(t)\}$  is said to be unique if any other solution  $\{\bar{x}(t)\}$  is indistinguishable from  $\{x(t)\}$ , i.e.,

$$\mathbf{P}\{x(t) = \bar{x}(t); \text{ for all } t_0 \leq t \leq T\} = 1.$$

**Remark 2.124.**

- Denote the solution of (2.9) by  $x(t, t_0, x_0)$ . From (2.10), for any  $s \in [t_0, T]$ ,

$$x(t) = x(s) + \int_s^t f(x(r), r) dr + \int_s^t g(x(r), r) dB(r), \quad s \leq t \leq T.$$

This describes a stochastic differential equation defined on the interval  $[s, T]$ , with its solution represented as  $x(t, s, x(s, t_0, x_0))$ . The initial condition is given by  $x(s) = x(s, t_0, x_0)$ . Consequently, the solution to equation (2.9) exhibits the semigroup property:

$$x(t, t_0, x_0) = x(t, s, x(s, t_0, x_0)), \quad t_0 \leq s \leq t \leq T.$$

- The coefficients  $f$  and  $g$  may depend on  $\omega$  in a general manner, provided they are adapted. For further details, see Gihman & Skorohod [97].
- Although we require the initial value  $x_0$  to be in  $\mathbf{L}^2$ , it is generally sufficient for  $x_0$  to be any  $\mathcal{F}_{t_0}$ -measurable random variable. For more details, refer to Gihman & Skorohod [97].

### 2.3.2 Existence and Uniqueness of Solutions

**Theorem 2.125 (Existence and Uniqueness).** Assume that there exist two positive constants  $\bar{K}$  and  $K$  such that

1. (*Lipschitz condition*) For all  $x, y \in \mathbb{R}^d$  and  $t \in [t_0, T]$ ,

$$|f(x, t) - f(y, t)|^2 \vee |g(x, t) - g(y, t)|^2 \leq \bar{K} |x - y|^2; \quad (2.11)$$

2. (*Linear growth condition*) For all  $(x, t) \in \mathbb{R}^d \times [t_0, T]$ ,

$$|f(x, t)|^2 \vee |g(x, t)|^2 \leq K(1 + |x|^2). \quad (2.12)$$

Then, there exists a unique solution  $x(t)$  to equation (2.9), and this solution belongs to the space  $\mathbb{M}^2([t_0, T], \mathbb{R}^d)$ .

**Lemma 2.126 (Solution Bound).** Assume that the linear growth condition (2.12) is satisfied. If  $x(t)$  is a solution of equation (2.9), then

$$\mathbb{E} \left( \sup_{t_0 \leq t \leq T} |x(t)|^2 \right) \leq (1 + 3\mathbb{E}(|x_0|^2)) e^{3K(T-t_0)(T-t_0+4)}.$$

In particular,  $x(t)$  belongs to  $\mathbb{M}^2([t_0, T], \mathbb{R}^d)$ .

**Theorem 2.127 (Convergence of Picard Iterations).** Let the assumptions of Theorem 2.125 hold. Let  $x(t)$  be the unique solution of (2.9), and let  $x_n(t)$  be Picard iterations. Then,

$$\mathbb{E} \left( \sup_{t_0 \leq t \leq T} |x_n(t) - x(t)|^2 \right) \leq \frac{8}{n!} C[M(T-t_0)]^n e^{8M(T-t_0)},$$

for all  $n \geq 1$ , where  $C$  and  $M$  are the same as defined in the proof of Theorem 2.125, i.e.,

$$C = 2K(T-t_0+1)(T-t_0)(1 + \mathbb{E}(|x_0|^2)) \quad \text{and} \quad M = 2K(T-t_0+1).$$

The Lipschitz condition (2.11) implies that the coefficients  $f(x, t)$  and  $g(x, t)$  do not change faster than a linear function of  $x$  when  $x$  varies. This particularly implies the continuity of  $f(x, t)$  and  $g(x, t)$  in  $x$  for all  $t \in [t_0, T]$ . Thus, functions that are discontinuous with respect to  $x$  are excluded as coefficients. Moreover, functions such as  $\sin(x^2)$  do not satisfy the Lipschitz condition. These observations indicate that the Lipschitz condition is restrictive. The following theorem generalizes Theorem 2.125 by replacing the (uniform) Lipschitz condition with a local Lipschitz condition.

**Theorem 2.128 (Local Lipschitz Existence and Uniqueness).** Assume that the linear growth condition (2.12) is satisfied, but the Lipschitz condition (2.11) is replaced with the following local Lipschitz condition: For every integer  $n \geq 1$ , there exists a positive constant  $K_n$  such that, for all  $t \in [t_0, T]$  and all  $x, y \in \mathbb{R}^d$  with  $|x| \vee |y| \leq n$ ,

$$|f(x, t) - f(y, t)|^2 \vee |g(x, t) - g(y, t)|^2 \leq K_n |x - y|^2. \quad (2.13)$$

Then, there exists a unique solution  $x(t)$  to equation (2.9) in  $\mathbb{M}^2([t_0, T], \mathbb{R}^d)$ .

The local Lipschitz condition allows us to include many functions as coefficients  $f(x, t)$  and  $g(x, t)$ , such as functions with continuous first-order partial derivatives with respect to  $x$  in  $\mathbb{R}^d \times [t_0, T]$ . However, the linear growth condition still excludes some important functions, such as  $-|x|^2 x$ , as coefficients. The following result improves this situation.

**Theorem 2.129 (Monotone Condition Existence and Uniqueness).** Assume that the local Lipschitz condition (2.13) is satisfied, but the linear growth condition (2.12) is replaced by the

following monotone condition: There exists a positive constant  $K$  such that for all  $(x, t) \in \mathbb{R}^d \times [t_0, T]$ ,

$$x^\top f(x, t) + \frac{1}{2}|g(x, t)|^2 \leq K(1 + |x|^2). \quad (2.14)$$

Then, there exists a unique solution  $x(t)$  to equation (2.9) in  $\mathbb{M}^2([t_0, T], \mathbb{R}^d)$ .

This theorem can be proved similarly to Theorem 2.128. The local Lipschitz condition guarantees that the solution exists on  $[t_0, \tau_\infty]$ , where  $\tau_\infty = \lim_{n \rightarrow \infty} \tau_n$ , but the monotone condition (instead of the linear growth condition) ensures that  $\tau_\infty = T$ , meaning that the solution exists on the entire interval  $[t_0, T]$ . We leave the details to the reader. Note that if the linear growth condition (2.12) holds, then the monotone condition (2.14) is satisfied, but the converse is not true.

In this note, we often discuss a stochastic differential equation on  $[t_0, \infty)$ , i.e.,

$$dx(t) = f(x(t), t) dt + g(x(t), t) dB(t) \quad \text{for } t \in [t_0, \infty), \quad (2.15)$$

with initial value  $x(t_0) = x_0$ . If the assumptions of the existence-and-uniqueness theorem hold on every finite subinterval  $[t_0, T]$  of  $[t_0, \infty)$ , then equation (2.15) has a unique solution  $x(t)$  on  $[t_0, \infty)$ . This solution is called the *global solution*. For convenience, we state the following theorem.

**Theorem 2.130 (Time-Dependent Local Lipschitz Condition).** Assume that for every real number  $T > t_0$  and integer  $n \geq 1$ , there exists a positive constant  $K_{T,n}$  such that for all  $t \in [t_0, T]$  and all  $x, y \in \mathbb{R}^d$  with  $|x| \vee |y| \leq n$ ,

$$|f(x, t) - f(y, t)|^2 \vee |g(x, t) - g(y, t)|^2 \leq K_{T,n}|x - y|^2.$$

Assume also that for every  $T > t_0$ , there exists a positive constant  $K_T$  such that for all  $(x, t) \in \mathbb{R}^d \times [t_0, T]$ ,

$$x^\top f(x, t) + \frac{1}{2}|g(x, t)|^2 \leq K_T(1 + |x|^2).$$

Then, there exists a unique global solution  $x(t)$  to equation (2.15), and the solution belongs to  $\mathbb{M}^2([t_0, \infty), \mathbb{R}^d)$ .

**Proposition 2.131 (Dynkin Formula).** For all  $x \in \mathbb{R}^q$ , all stopping times  $\tau$  with  $\mathbb{E}_x(\tau) < \infty$ , and all  $f \in \mathcal{C}^2(\mathbb{R}^q, \mathbb{R})$  with compact support,

$$\mathbb{E}_x(f(X_\tau)) = f(x) + \mathbb{E}_x \int_0^\tau \mathcal{L}f(X_s) ds.$$

If  $\tau$  is the exit time of a bounded set, then we can abandon the restriction of compactness of support.

**Definition 2.132 (Stochastic dynamics of the infected population in an SIRS model).** Let  $I(t)$  denote the size of the infected population at time  $t$ .

- **Exponential extinction.** The infected population undergoes exponential extinction almost surely if

$$\limsup_{t \rightarrow +\infty} \frac{\ln(I(t))}{t} < 0 \quad a.s..$$

- **Extinction.** The infected population becomes extinct almost surely if

$$\lim_{t \rightarrow +\infty} I(t) = 0 \quad a.s..$$

- **Non-persistence in the mean.** The infected population does not persist on average almost surely if

$$\lim_{t \rightarrow +\infty} \frac{1}{t} \int_0^t I(s) ds = 0 \quad a.s..$$

- **Weak persistence in the mean.** The infected population weakly persists on average almost surely if

$$\limsup_{t \rightarrow +\infty} \frac{1}{t} \int_0^t I(s) ds > 0 \quad a.s..$$

- **Strong persistence in the mean.** The infected population strongly persists on average almost surely if

$$\liminf_{t \rightarrow +\infty} \frac{1}{t} \int_0^t I(s) ds > 0 \quad a.s..$$

- **Stochastic permanence.** For every  $\varepsilon > 0$ , there exist positive constants  $C_1, C_2$  such that:

- ▶ **Ultimate boundedness.**

$$\limsup_{t \rightarrow +\infty} \mathbf{P}(I(t) > C_1) \leq \varepsilon.$$

- ▶ **Persistence.**

$$\liminf_{t \rightarrow +\infty} \mathbf{P}(I(t) \geq C_2) \geq 1 - \varepsilon.$$

- **Stochastically bounded.** For each  $\varepsilon > 0$ , there exists  $\gamma_\varepsilon = \gamma_\varepsilon(t_0, I_0) > 0$  such that

$$\inf_{t \in [t_0, T]} \mathbb{P}(|I(t)| \leq \gamma_\varepsilon) > 1 - \varepsilon.$$

If  $\gamma_\varepsilon$  depends only on  $I_0$ , then  $I(t)$  is called uniformly stochastically bounded.

- **Continuity on  $[t_0, T]$ .** There exists a constant  $\rho > 0$  such that for all  $s, t \in [t_0, T]$ ,

$$|I(t) - I(s)|^2 \leq \rho |t - s|,$$

where

$$I(t) - I(s) = \int_s^t f(u, I(u)) du + \int_s^t g(u, I(u)) dB(u).$$

**Theorem 2.133.** Let  $Y(t)$  and  $Z(t)$  be processes such that

$$\lim_{t \rightarrow \infty} \frac{Z(t)}{t} = 0 \quad a.s..$$

- If for all  $t \geq 0$

$$\ln(Y(t)) \geq vt - v_0 \int_0^t Y(s) ds + Z(t),$$

then

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t Y(s) ds \geq \frac{v}{v_0} \quad a.s..$$

- If for all  $t \geq 0$

$$\ln Y(t) \leq vt - v_0 \int_0^t Y(s) ds + Z(t),$$

then

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \int_0^t Y(s) ds \leq \frac{v}{v_0} \quad a.s..$$

### 2.3.3 Euler-Maruyama Approximate Solutions

We now discuss the Euler-Maruyama approximate solutions, defined as follows. For every integer  $n \geq 1$ , set  $x_n(t_0) = x_0$ , and for  $t_0 + (k-1)/n < t \leq (t_0 + k/n) \wedge T$ ,  $k = 1, 2, \dots$ , we have

$$\begin{aligned} x_n(t) &= x_n\left(t_0 + \frac{k-1}{n}\right) + \int_{t_0 + \frac{k-1}{n}}^t f\left(x_n\left(t_0 + \frac{k-1}{n}\right), s\right) ds \\ &\quad + \int_{t_0 + \frac{k-1}{n}}^t g\left(x_n\left(t_0 + \frac{k-1}{n}\right), s\right) dB(s). \end{aligned} \quad (2.16)$$

Note that if we define

$$\widehat{x}_n(t) = x_0 \mathbb{1}_{\{t_0\}}(t) + \sum_{k \geq 1} x_n\left(t_0 + \frac{k-1}{n}\right) \mathbb{1}_{(t_0 + \frac{k-1}{n}, t_0 + \frac{k}{n}]}(t),$$

for  $t_0 \leq t \leq T$ , then (2.16) implies

$$x_n(t) = x_0 + \int_{t_0}^t f(\widehat{x}_n(s), s) ds + \int_{t_0}^t g(\widehat{x}_n(s), s) dB(s).$$

**Lemma 2.134 (Bound on Euler-Maruyama Approximations).** *Under the linear growth condition (2.12), the Euler-Maruyama approximate solutions  $\{x_n(t)\}$  satisfy*

$$\sup_{t_0 \leq t \leq T} \mathbb{E}(|x_n(t)|^2) \leq C_1 = (1 + 3\mathbb{E}(|x_0|^2)) e^{3K(T-t_0)(T-t_0+1)}.$$

**Lemma 2.135 (Increment Bound).** *Under the linear growth condition (2.12), the Euler-Maruyama approximate solutions  $\{x_n(t)\}$  satisfy, for  $t_0 \leq s < t \leq T$  with  $t - s \leq 1$ ,*

$$\mathbb{E}(|x_n(t) - x_n(s)|^2) \leq C_2(t - s),$$

where  $C_2 = 4K(1 + C_1)$  and  $C_1$  is defined in Lemma 2.134.

**Theorem 2.136 (Convergence Rate).** *Assume the Lipschitz condition (2.11) and the linear growth condition (2.12) hold. Let  $x(t)$  be the unique solution of (2.9), and  $\{x_n(t)\}_{n \geq 1}$  the Euler-Maruyama approximate solutions. Then*

$$\mathbb{E}\left(\sup_{t_0 \leq t \leq T} |x_n(t) - x(t)|^2\right) \leq \frac{C_3}{n},$$

where  $C_3 = 4C_2\bar{K}(T - t_0)(T - t_0 + 4) \exp[4\bar{K}(T - t_0)(T - t_0 + 4)]$  and  $C_2$  is defined in Lemma 2.135.

We omit the proofs of these results. Moreover, we have the following more general convergence theorem.

**Theorem 2.137 (General Convergence).** *Under the same conditions, the Euler-Maruyama approximate solutions  $\{x_n(t)\}$  converge to the unique solution  $x(t)$  of (2.9) in the sense that*

$$\lim_{n \rightarrow \infty} \mathbb{E}\left[\sup_{t_0 \leq t \leq T} \|x_n(t) - x(t)\|^2\right] = 0. \quad (2.17)$$

This result was established by Kaneko & Nakao [98]. Theorem 2.137 shows that both the approximate solutions of Euler-Maruyama and Carathéodory converge to the unique solution of (2.9) under general conditions. However, it remains an open question whether Picard approximate solutions converge under these same conditions.

For time-homogeneous stochastic differential equations of the form

$$dx(t) = f(x(t)) dt + g(x(t)) dB(t),$$

the Euler-Maruyama approximation simplifies to  $x_n(t_0) = x_0$  and

$$\begin{aligned} x_n(t) &= x_n \left( t_0 + \frac{k-1}{n} \right) + f \left( x_n \left( t_0 + \frac{k-1}{n} \right) \right) \left[ t - t_0 - \frac{k-1}{n} \right] \\ &\quad + g \left( x_n \left( t_0 + \frac{k-1}{n} \right) \right) \left[ B(t) - B \left( t_0 + \frac{k-1}{n} \right) \right], \end{aligned} \quad (2.18)$$

for  $t_0 + \frac{k-1}{n} < t \leq (t_0 + \frac{k}{n}) \wedge T$ ,  $k = 1, 2, \dots$ .

**Remark 2.138.** In previous subsections, we worked on a given probability space  $(\Omega, \mathcal{F}, \mathbf{P})$  with filtration  $\{\mathcal{F}_t\}_{t \geq 0}$ , Brownian motion  $B(t)$ , and coefficients  $f(x, t)$ ,  $g(x, t)$ , and then constructed the solution  $x(t)$ . Such solutions are called strong solutions. If only the coefficients  $f(x, t)$  and  $g(x, t)$  are given, and we can construct an appropriate probability space, filtration, Brownian motion, and solution, the result is called a weak solution. Two solutions (weak or strong) are weakly unique if they have identical probability laws (i.e., the same finite-dimensional distributions). If any two weak solutions constructed under different probability spaces are indistinguishable, we say that pathwise uniqueness holds.

Clearly, every strong solution is a weak solution, but the converse is not generally true (see Tanaka's example in Rogers & Williams [99, Sec.V.16]). Pathwise uniqueness implies weak uniqueness. Moreover, the conditions given above (e.g., the Lipschitz condition) guarantee pathwise uniqueness since the uniqueness proofs hold for arbitrary probability spaces. In this work, we focus exclusively on strong solutions, unless otherwise stated.

## 2.4 Stability of Stochastic Differential Equations

### 2.4.1 Introduction

In 1892, A.M. Lyapunov introduced the concept of stability for dynamic systems. Roughly speaking, stability refers to the insensitivity of a system's state to small changes in the initial conditions or parameters. For a stable system, trajectories that are close to each other at a specific instant should remain close at all subsequent instants.

To better understand stochastic stability theory, let us first recall some basic facts about the stability of deterministic systems described by ordinary differential equations (ODEs). For details, see [100] and [101]. Consider a  $d$ -dimensional ODE

$$\dot{x}(t) = f(x(t), t) \quad \text{on } t \geq t_0. \quad (2.19)$$

Assume that for every initial value  $x(t_0) = x_0 \in \mathbb{R}^d$ , there exists a unique global solution, denoted by  $x(t, t_0, x_0)$ . Furthermore, assume that

$$f(0, t) = 0 \quad \text{for all } t \geq t_0.$$

Thus, Equation (2.19) has the solution  $x(t) \equiv 0$  corresponding to the initial value  $x(t_0) = 0$ . This solution is called the *trivial solution* or *equilibrium position*.

The trivial solution is said to be *stable* if, for every  $\varepsilon > 0$ , there exists  $\delta = \delta(\varepsilon, t_0) > 0$  such that

$$|x(t, t_0, x_0)| < \varepsilon \quad \text{for all } t \geq t_0,$$

whenever  $|x_0| < \delta$ . Otherwise, it is said to be *unstable*. The trivial solution is *asymptotically stable* if it is stable and there exists  $\delta_0 = \delta_0(t_0) > 0$  such that

$$\lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0,$$

whenever  $|x_0| < \delta_0$ .

If (2.19) could be solved explicitly, determining stability would be straightforward. However, explicit solutions are only possible in special cases. Fortunately, Lyapunov (1892) developed a method for assessing stability without solving the equation, now known as the *Lyapunov direct method* (or *second method*). To explain this method, we introduce some notation.

- Let  $\mathcal{K}$  denote the family of all continuous non-decreasing functions  $\mu : \mathbb{R}_+ \rightarrow \mathbb{R}_+$  such that  $\mu(0) = 0$  and  $\mu(r) > 0$  for  $r > 0$ .
- For  $h > 0$ , let  $S_h = \{x \in \mathbb{R}^d \mid |x| < h\}$ .

A continuous function  $V(x, t)$  defined on  $S_h \times [t_0, \infty)$  is *positive-definite* (in the Lyapunov sense) if  $V(0, t) \equiv 0$  and, for some  $\mu \in \mathcal{K}$ ,

$$V(x, t) \geq \mu(|x|) \quad \text{for all } (x, t) \in S_h \times [t_0, \infty).$$

A function  $V$  is *negative-definite* if  $-V$  is positive-definite. A continuous non-negative function  $V(x, t)$  is *decreasing* (i.e., has an arbitrarily small upper bound) if, for some  $\mu \in \mathcal{K}$ ,

$$V(x, t) \leq \mu(|x|) \quad \text{for all } (x, t) \in S_h \times [t_0, \infty).$$

A function  $V(x, t)$  defined on  $\mathbb{R}^d \times [t_0, \infty)$  is *radially unbounded* if

$$\lim_{|x| \rightarrow \infty} \inf_{t \geq t_0} V(x, t) = \infty.$$

Let  $C^{1,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$  denote the family of all continuous functions  $V(x, t)$  from  $S_h \times [t_0, \infty)$  to  $\mathbb{R}_+$  with continuous first partial derivatives in  $x$  and  $t$ . If  $x(t)$  is a solution of (2.19) and  $V(x, t) \in C^{1,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$ , then  $v(t) = V(x(t), t)$  has the derivative

$$\dot{v}(t) = V_t(x(t), t) + V_x(x(t), t)f(x(t), t) = \frac{\partial V}{\partial t}(x(t), t) + \sum_{i=1}^d \frac{\partial V}{\partial x_i}(x(t), t)f_i(x(t), t).$$

If  $\dot{v}(t) \leq 0$ , then  $v(t)$  does not increase, which means that the distance from equilibrium (measured by  $V(x(t), t)$ ) does not grow. If  $\dot{v}(t) < 0$ , then  $v(t)$  decreases to zero, implying  $x(t) \rightarrow 0$ . These ideas form the basis of Lyapunov's direct method and lead to the following theorem.

### Theorem 2.139.

1. If there exists a positive-definite function  $V(x, t) \in C^{1,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$  such that

$$\dot{V}(x, t) = V_t(x(t), t) + V_x(x(t), t)f(x(t), t) \leq 0,$$

for all  $(x, t) \in S_h \times [t_0, \infty)$ , then the trivial solution of (2.19) is stable.

2. If there exists a positive-definite, decreasing function  $V(x, t) \in C^{1,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$  such that  $\dot{V}(x, t)$  is negative-definite, then the trivial solution of (2.19) is asymptotically stable.

A function  $V(x, t)$  satisfying the conditions of Theorem 2.139 is called a *Lyapunov function* for the ODE. When extending Lyapunov stability theory to stochastic systems, several questions arise:

- What is an appropriate definition of stochastic stability?
- What conditions should a stochastic Lyapunov function satisfy?
- How should inequality  $\dot{V}(x, t) \leq 0$  be modified to obtain stability results?

There are at least three types of stochastic stability:

- Stability in probability,
- Moment stability,
- Almost sure stability.

In 1965, Bucy showed that a stochastic Lyapunov function should have the supermartingale property and provided simple criteria for stability in probability and moment stability. Almost sure stability was studied by Hasminskii (1967) for linear stochastic differential equations (SDEs). Stochastic stability remains an active research area, with contributions from many mathematicians, including Arnold, Baxendale, Chow, Curtain, Elworthy, Friedman, Ichikawa, Kliemann, Kolmanovskii, Kushner, Ladde, Lakshmikantham, Mohammed, Pardoux, Pinsky, Pritchard, Truman, Wihstutz, Zabczyk, and others.

In this section, we investigate these stability types for the  $d$ -dimensional SDE

$$dx(t) = f(x(t), t)dt + g(x(t), t)dB(t) \quad \text{on } t \geq t_0.$$

For stability analysis, it suffices (as we will explain later) to consider constant initial values  $x_0 \in \mathbb{R}^d$  rather than  $\mathcal{F}_{t_0}$ -measurable random variables  $x_0 \in \mathbf{L}^2(\Omega, \mathbb{R}^d)$ . Throughout, we assume that the conditions of Theorem 2.130 (existence and uniqueness) hold. Thus, for any initial value  $x(t_0) = x_0 \in \mathbb{R}^d$ , equation (2.9) has a unique global solution  $x(t, t_0, x_0)$  with continuous sample paths and finite moments. Moreover, assume

$$f(0, t) = 0 \quad \text{and} \quad g(0, t) = 0 \quad \text{for all } t \geq t_0.$$

Hence, (2.9) has the trivial solution  $x(t) \equiv 0$  corresponding to  $x(t_0) = 0$ .

Additional notation: Let  $0 < h \leq \infty$ . Denote by  $\mathcal{C}^{2,1}(S_h \times \mathbb{R}_+, \mathbb{R}_+)$  the family of non-negative functions  $V(x, t)$  in  $S_h \times \mathbb{R}_+$  that are twice continuously differentiable in  $x$  and once in  $t$ . The differential operator  $L$  associated with (2.9) is defined as

$$\mathcal{L} = \frac{\partial}{\partial t} + \sum_{i=1}^d f_i(x, t) \frac{\partial}{\partial x_i} + \frac{1}{2} \sum_{i,j=1}^d [g(x, t)g^T(x, t)]_{ij} \frac{\partial^2}{\partial x_i \partial x_j}.$$

For  $V \in \mathcal{C}^{2,1}(S_h \times \mathbb{R}_+, \mathbb{R}_+)$ ,

$$\mathcal{L}V(x, t) = V_t(x, t) + V_x(x, t)f(x, t) + \frac{1}{2} \text{trace}[g^\top(x, t)V_{xx}(x, t)g(x, t)].$$

(See Definition 2.110 for definitions of  $V_t$ ,  $V_x$ , and  $V_{xx}$ .) By Itô's formula, if  $x(t) \in S_h$ , then

$$dV(x(t), t) = \mathcal{L}V(x(t), t)dt + V_x(x(t), t)g(x(t), t)dB(t).$$

This justifies the definition of  $L$ . In stochastic stability, the condition  $\dot{V}(x, t) \leq 0$  is replaced by  $\mathcal{L}V(x, t) \leq 0$ .

Stochastic Process	Notation	Infinitesimal Generator $\mathcal{L}$
Standard Brownian motion	$(B_t)_{t \geq 0}$	$\mathcal{L}f(x) = \frac{1}{2} \frac{d^2 f}{dx^2}(x)$
Homogeneous Poisson process	$(N_t)_{t \geq 0}$	$\mathcal{L}f(x) = \lambda(f(x+1) - f(x))$
Itô diffusion process	$(X_t)_{t \geq 0}, dX_t = \mu(t, X_t)dt + \sigma(t, X_t)dB_t$	$\mathcal{L}f(x) = \mu(t, x)\partial_x f(x) + \frac{1}{2}\sigma^2(t, x)\partial_{xx}f(x)$ (generator) $\partial_t f + \mu(t, x)\partial_x f + \frac{1}{2}\sigma^2(t, x)\partial_{xx}f$ (Kolmogorov operator)
Lévy process	$(L_t)_{t \geq 0}$	$\mathcal{L}f(x) = \gamma f'(x) + \frac{1}{2}\sigma^2 f''(x)$ $+ \int_{\mathbb{R} \setminus \{0\}} [f(x+z) - f(x) - z \mathbb{1}_{\{ z  < 1\}} f'(x)] \nu(dz)$
Geometric Brownian motion	$(S_t)_{t \geq 0}$ $dS_t = \mu S_t dt + \sigma S_t dB_t$	$\mathcal{L}f(t, x) = \partial_t f + \mu x \partial_x f + \frac{1}{2}\sigma^2 x^2 \partial_{xx}f$
Markov-switching diffusion	$(X_t, \alpha_t)_{t \geq 0}$ ( $\alpha_t$ Markov chain)	$\mathcal{L}f(x, i) = \mu_i(x)\partial_x f(x, i) + \frac{1}{2}\sigma_i^2(x)\partial_{xx}f(x, i)$ $+ \sum_{j \neq i} q_{ij}(f(x, j) - f(x, i))$

Table 2.3. Stochastic Processes and Their Infinitesimal Generators

- Markov Regime-Switching Diffusions
  - ▶  $Q = (q_{ij})$  generator matrix of  $\alpha_t$
  - ▶  $\mu_i, \sigma_i$  regime-dependent parameters
- $\nu$  denotes the Lévy measure
- $\mathcal{L}$  characterizes the evolution through the test functions  $f$

## 2.4.2 Stability in Probability

In this section, we discuss stability in probability. We emphasize that throughout this section, the initial value  $x_0$  is assumed to be a constant (in  $\mathbb{R}^d$ ) and not a random variable. We will explain why it suffices to consider constant initial values after defining stability in probability.

### Definition 2.140.

1. *The trivial solution of equation (2.9) is said to be stochastically stable or stable in probability if, for every pair  $\varepsilon \in (0, 1)$  and  $r > 0$ , there exists a  $\delta = \delta(\varepsilon, r, t_0) > 0$  such that*

$$\mathbf{P}\{|x(t, t_0, x_0)| < r \text{ for all } t \geq t_0\} \geq 1 - \varepsilon,$$

whenever  $|x_0| < \delta$ . Otherwise, it is said to be stochastically unstable.

2. *The trivial solution is said to be stochastically asymptotically stable if it is stochastically stable and, moreover, for every  $\varepsilon \in (0, 1)$ , there exists a  $\delta_0 = \delta_0(\varepsilon, t_0) > 0$  such that*

$$\mathbf{P}\left\{\lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0\right\} \geq 1 - \varepsilon,$$

whenever  $|x_0| < \delta_0$ .

3. The trivial solution is said to be stochastically asymptotically stable in the large if it is stochastically stable and, moreover, for all  $x_0 \in \mathbb{R}^d$ ,

$$\mathbf{P} \left\{ \lim_{t \rightarrow \infty} x(t, t_0, x_0) = 0 \right\} = 1.$$

Let us now explain why it is sufficient to consider constant initial values. Suppose instead that the initial value  $x_0$  is a random variable. Then, for example, replace  $|x_0| < \delta$  with  $|x_0| < \delta$  a.s. in the definition. Although this appears more general, it is, in fact, equivalent to the above definition. For example, if (1) holds, then for any random variable  $x_0$  with  $|x_0| < \delta$  a.s., we have

$$\begin{aligned} \mathbf{P} \{ |x(t, t_0, x_0)| < r \text{ for all } t \geq t_0 \} &= \int_{S_\delta} \mathbf{P} \{ |x(t, t_0, y)| < r \text{ for all } t \geq t_0 \} \mathbf{P} \{ x_0 \in dy \} \\ &\geq \int_{S_\delta} (1 - \varepsilon) \mathbf{P} \{ x_0 \in dy \} = 1 - \varepsilon. \end{aligned}$$

It should also be noted that when  $g(x, t) \equiv 0$ , these definitions reduce to their deterministic counterparts. We now extend Lyapunov's Theorem 2.139 to the stochastic case.

**Theorem 2.141.** *If there exists a positive-definite function  $V(x, t) \in \mathcal{C}^{2,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$  such that*

$$\mathcal{L}V(x, t) \leq 0,$$

*for all  $(x, t) \in S_h \times [t_0, \infty)$ , then the trivial solution of equation (2.9) is stochastically stable.*

**Theorem 2.142.** *If there exists a positive-definite decrescent function  $V(x, t) \in \mathcal{C}^{2,1}(S_h \times [t_0, \infty), \mathbb{R}_+)$  such that  $\mathcal{L}V(x, t)$  is negative-definite, then the trivial solution of (2.9) is stochastically asymptotically stable.*

**Theorem 2.143.** *If there exists a function  $V(x, t) \in \mathcal{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty); \mathbb{R}_+)$  that is positive-definite, decrescent, and radially unbounded such that  $\mathcal{L}V(x, t)$  is negative-definite, then the trivial solution of (2.9) is stochastically asymptotically stable in the large.*

The functions  $V(x, t)$  used in Theorems 2.141 and 2.143 are called *stochastic Lyapunov functions*, and the application of these theorems depends on the construction of such functions. As in the deterministic case, several techniques can be employed to find suitable functions. For example, the quadratic function

$$V(x, t) = x^T Q x,$$

where  $Q$  is a symmetric positive-definite matrix, is a candidate provided that

$$\mathcal{L}V(x, t) = 2x^T Q f(x, t) + \text{trace}[g^T(x, t) Q g(x, t)] \leq 0,$$

or is negative-definite in some neighborhood of  $x = 0$  for  $t \geq t_0$ . Additionally, a positive-definite solution can be sought for equation  $\mathcal{L}V(x, t) = 0$  or the inequality  $\mathcal{L}V(x, t) \leq 0$ . We now present several examples to illustrate the theory.

### 2.4.3 Almost Sure Exponential Stability

We begin by giving a formal definition of almost sure exponential stability.

**Definition 2.144.** The trivial solution of equation (2.9) is said to be almost surely exponentially stable if

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| < 0 \quad a.s., \quad (2.20)$$

for all  $x_0 \in \mathbb{R}^d$ .

The left-hand side of (2.20) defines the *sample Lyapunov exponent* of the solution. We conclude that the trivial solution is almost surely exponentially stable if and only if all sample Lyapunov exponents are negative.

As discussed in the previous section, almost sure exponential stability implies that, with probability one, all sample paths of the solution converge exponentially fast to the equilibrium position  $x = 0$ .

To clarify why we may restrict our analysis to constant initial values, consider a general initial condition  $x_0$  (that is, a  $\mathcal{F}_{t_0}$ -measurable random variable in  $\mathbf{L}^2(\Omega, \mathbb{R}^d)$ ). Equation (2.11) shows that

$$\begin{aligned} \mathbf{P} \left\{ \limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| < 0 \right\} &= \int_{\mathbb{R}^d} \mathbf{P} \left\{ \limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, y)| < 0 \right\} \mathbf{P}\{x_0 \in dy\} \\ &= \int_{\mathbb{R}^d} \mathbf{P}\{x_0 \in dy\} = 1, \end{aligned}$$

that is,

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| < 0 \quad a.s..$$

To establish the theorems on almost sure exponential stability, we need the following useful lemma. Recall that we assume, throughout this subsection, that the assumptions of the existence-and-uniqueness Theorem 2.130 are fulfilled and, moreover,  $f(0, t) \equiv 0$ ,  $g(0, t) \equiv 0$ . Under these standing hypotheses, we have the following lemma.

**Lemma 2.145.** For all  $x_0 \neq 0$  in  $\mathbb{R}^d$ ,

$$\mathbf{P}\{x(t, t_0, x_0) \neq 0 \text{ on } t \geq t_0\} = 1.$$

That is, almost all sample paths of any solution starting from a non-zero state will never reach the origin.

**Theorem 2.146.** Assume that there exists a function  $V \in \mathcal{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty), \mathbb{R}_+)$ , and constants  $p > 0$ ,  $c_1 > 0$ ,  $c_2 \in \mathbb{R}$ ,  $c_3 \geq 0$ , such that for all  $x \neq 0$  and  $t \geq t_0$

- $c_1|x|^p \leq V(x, t)$ ,
- $\mathcal{L}V(x, t) \leq c_2V(x, t)$ ,
- $|V_x(x, t)g(x, t)|^2 \geq c_3V^2(x, t)$ .

Then

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| \leq -\frac{c_3 - 2c_2}{2p} \quad a.s..$$

for all  $x_0 \in \mathbb{R}^d$ . In particular, if  $c_3 > 2c_2$ , the trivial solution of equation (2.9) is almost surely exponentially stable.

**Corollary 2.147.** Assume that there exists a function  $V \in \mathcal{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty), \mathbb{R}_+)$ , and positive constants  $p, \alpha, \lambda$ , such that for all  $x \neq 0, t \geq t_0$

$$\alpha|x|^p \leq V(x, t) \quad \text{and} \quad \mathcal{L}V(x, t) \leq -\lambda V(x, t),$$

then

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| \leq -\frac{\lambda}{p} \quad \text{a.s.,}$$

for all  $x_0 \in \mathbb{R}^d$ . In other words, the trivial solution of equation (2.9) is almost surely exponentially stable.

This corollary follows immediately from Theorem 2.146 letting  $c_1 = \alpha$ ,  $c_2 = -\lambda$ , and  $c_3 = 0$ . These results provide an upper bound for the sample Lyapunov exponents. We now turn our attention to the lower bound.

**Theorem 2.148.** Assume that there exists a function  $V \in \mathcal{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty), \mathbb{R}_+)$ , and constants  $p > 0$ ,  $c_1 > 0$ ,  $c_2 \in \mathbb{R}$ ,  $c_3 > 0$ , such that for all  $x \neq 0$  and  $t \geq t_0$

- $c_1|x|^p \geq V(x, t) > 0$ ,
- $\mathcal{L}V(x, t) \geq c_2 V(x, t)$ ,
- $|V_x(x, t)g(x, t)|^2 \leq c_3 V^2(x, t)$ .

Then

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \log |x(t, t_0, x_0)| \geq \frac{2c_2 - c_3}{2p} \quad \text{a.s.,}$$

for all  $x_0 \neq 0$  in  $\mathbb{R}^d$ . In particular, if  $2c_2 > c_3$ , then almost all sample paths of  $|x(t, t_0, x_0)|$  will tend to infinity, and we say in this case that the trivial solution of equation (2.9) is almost surely exponentially unstable.

## 2.4.4 Moment Exponential Stability

In this subsection, we discuss the exponential stability of the  $p$ -th moment for equation (2.9), where we always assume  $p > 0$ . We first define the exponential stability of the  $p$ -th moment.

**Definition 2.149.** The trivial solution of equation (2.9) is said to be  $p$ -th moment exponentially stable if there exist positive constants  $\lambda$  and  $C$  such that

$$\mathbb{E}(|x(t, t_0, x_0)|^p) \leq C|x_0|^p e^{-\lambda(t-t_0)} \quad \text{for all } t \geq t_0, \tag{2.21}$$

for all  $x_0 \in \mathbb{R}^d$ . When  $p = 2$ , it is usually said to be exponentially stable in mean square.

Clearly,  $p$ -th moment exponential stability implies that the  $p$ -th moment of the solution tends to zero exponentially fast. Moreover, it follows from (2.21) that

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \log (\mathbb{E}(|x(t, t_0, x_0)|^p)) < 0. \tag{2.22}$$

The left-hand side of (2.22) defines the  $p$ -th moment Lyapunov exponent of the solution. Therefore, in this case, the  $p$ -th moment is exponentially stable in the Lyapunov sense, which means that the Lyapunov exponent is negative.

Moreover, if the initial value is an  $\mathcal{F}_{t_0}$ -measurable random variable  $x_0 \in \mathbf{L}^p(\Omega, \mathbb{R}^d)$ , then by (2.21),

$$\begin{aligned}\mathbb{E}(|x(t, t_0, x_0)|^p) &= \int_{\mathbb{R}^d} \mathbb{E}(|x(t, t_0, y)|^p) \mathbf{P}\{x_0 \in dy\} \\ &\leq \int_{\mathbb{R}^d} C|y|^p e^{-\lambda(t-t_0)} \mathbf{P}\{x_0 \in dy\} = C\mathbb{E}(|x_0|^p) e^{-\lambda(t-t_0)}.\end{aligned}$$

Furthermore, since  $(\mathbb{E}(|x(t)|^p))^{\hat{p}/p} \leq (\mathbb{E}(|x(t)|^p))^{1/p}$  for  $0 < \hat{p} < p$ , we see that the exponential stability of the  $p$ -th moment implies the exponential stability of the  $\hat{p}$ -th moment.

In general,  $p$ -th moment exponential stability and almost sure exponential stability do not imply each other; additional conditions are required to deduce one from the other. The following theorem provides conditions under which  $p$ -th moment exponential stability implies almost sure exponential stability.

**Theorem 2.150.** *Assume that there exists a positive constant  $K$  such that*

$$x^\top f(x, t) \vee |g(x, t)|^2 \leq K|x|^2 \quad \text{for all } (x, t) \in \mathbb{R}^d \times [t_0, \infty). \quad (2.23)$$

*Then, the  $p$ -th moment exponential stability of the trivial solution of (2.9) implies almost sure exponential stability.*

Although condition (2.23) is not guaranteed by the assumptions of the existence-and-uniqueness Theorem 2.130 (which are assumed throughout this chapter), it holds in many important cases. For example, if the coefficients  $f(x, t)$  and  $g(x, t)$  are uniformly Lipschitz continuous, then (2.23) is satisfied, noting that we always assume  $f(0, t) \equiv 0$  and  $g(0, t) \equiv 0$  in this chapter.

Moreover, for the  $d$ -dimensional linear stochastic differential equation

$$dx(t) = F(t)x(t)dt + \sum_{i=1}^m G_i(t)x(t)dB_i(t),$$

condition (2.23) holds if  $F$  and  $G_i$  are bounded  $d \times d$  matrix-valued functions. Hence, we obtain the following useful corollary.

**Corollary 2.151.** *Let  $F$  and  $G_i$  be bounded  $d \times d$  matrix-valued functions. Then, the  $p$ -th moment exponential stability of the trivial solution to the linear equation implies almost sure exponential stability.*

We now establish a sufficient condition for  $p$ -th moment exponential stability via a Lyapunov function.

**Theorem 2.152.** *Assume there exists a function  $V(x, t) \in \mathscr{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty), \mathbb{R}_+)$  and positive constants  $c_1, c_2, c_3$  such that*

$$c_1|x|^p \leq V(x, t) \leq c_2|x|^p \quad \text{and} \quad \mathcal{L}V(x, t) \leq -c_3V(x, t),$$

*for all  $(x, t) \in \mathbb{R}^d \times [t_0, \infty)$ . Then,*

$$\mathbb{E}|x(t, t_0, x_0)|^p \leq \frac{c_2}{c_1}|x_0|^p e^{-c_3(t-t_0)} \quad \text{for all } t \geq t_0,$$

*for all  $x_0 \in \mathbb{R}^d$ . In other words, the trivial solution of equation (2.9) is  $p$ -th moment exponentially stable, and the  $p$ -th moment Lyapunov exponent is no greater than  $-c_3$ .*

Similarly, we can prove the following theorem, which provides a sufficient condition for the  $q$ -th moment exponential instability.

**Theorem 2.153.** *Let  $q > 0$ . Assume there exists a function  $V(x, t) \in \mathcal{C}^{2,1}(\mathbb{R}^d \times [t_0, \infty), \mathbb{R}_+)$  and positive constants  $c_1, c_2, c_3$  such that*

$$c_1|x|^q \leq V(x, t) \leq c_2|x|^q \quad \text{and} \quad \mathcal{L}V(x, t) \geq c_3V(x, t),$$

for all  $(x, t) \in \mathbb{R}^d \times [t_0, \infty)$ . Then,

$$\mathbb{E}|x(t, t_0, x_0)|^q \geq \frac{c_1}{c_2}|x_0|^q e^{c_3(t-t_0)} \quad \text{for all } t \geq t_0,$$

for all  $x_0 \in \mathbb{R}^d$ . In this case, the trivial solution of equation (2.9) is  $q$ -th moment exponentially unstable.

Since  $(\mathbb{E}|x(t)|^q)^{1/\hat{q}} \geq (\mathbb{E}|x(t)|^q)^{1/q}$  for  $\hat{q} > q$ ,  $q$ -th moment exponential instability implies  $\hat{q}$ -th moment exponential instability. We now use Theorem 2.152 to derive a useful corollary.

**Corollary 2.154.** *Assume there exists a symmetric positive-definite  $d \times d$  matrix  $Q$  and constants  $\alpha_1, \alpha_2, \alpha_3$  such that for all  $(x, t) \in \mathbb{R}^d \times [t_0, \infty)$*

$$x^\top Qf(x, t) + \frac{1}{2} \operatorname{trace}[g^\top(x, t)Qg(x, t)] \leq \alpha_1 x^\top Qx,$$

and

$$\alpha_2 x^\top Qx \leq |x^\top Qg(x, t)| \leq \alpha_3 x^\top Qx.$$

- 1. If  $\alpha_1 < 0$ , then the trivial solution of equation (2.9) is  $p$ -th moment exponentially stable provided  $p < 2 + 2|\alpha_1|/\alpha_3^2$ .
- 2. If  $0 \leq \alpha_1 < \alpha_2^2$ , then the trivial solution of equation (2.9) is  $p$ -th moment exponentially stable provided  $p < 2 - 2\alpha_1/\alpha_2^2$ .

Similarly, we can use Theorem 2.153 to establish the following result on moment exponential instability.

**Corollary 2.155.** *Assume there exists a symmetric positive-definite  $d \times d$  matrix  $Q$  and positive constants  $\beta_1, \beta_2$  such that for all  $(x, t) \in \mathbb{R}^d \times [t_0, \infty)$ ,*

$$x^\top Qf(x, t) + \frac{1}{2} \operatorname{trace}[g^\top(x, t)Qg(x, t)] \geq \beta_1 x^\top Qx,$$

and

$$|x^\top Qg(x, t)| \leq \beta_2 x^\top Qx.$$

Then, the trivial solution of equation (2.9) is  $q$ -th moment exponentially unstable provided  $q > 0 \vee (2 - 2\beta_1/\beta_2^2)$ .

## 2.5 Ergodic Properties of Solutions of Stochastic Equations

## 2.5.1 Formulation and Preliminaries

### 2.5.1.1 Switching Diffusion

Recall that  $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbf{P})$  is a complete probability space with a filtration  $\{\mathcal{F}_t\}_{t \geq 0}$  satisfying the usual conditions (that is, it is right-continuous with  $\mathcal{F}_0$  containing all  $\mathbf{P}$ -null sets). Let  $x \in \mathbb{R}^r$ ,  $\mathcal{E} = \{1, \dots, m_0\}$ , and  $Q(x) = (q_{ij}(x))$  be an  $m_0 \times m_0$  matrix depending on  $x$  such that  $q_{ij}(x) \geq 0$  for  $i \neq j$  and  $\sum_{j=1}^{m_0} q_{ij}(x) = 0$  for all  $x \in \mathbb{R}^r$ . For any twice continuously differentiable function  $h(\cdot, i)$ , where  $i \in \mathcal{E}$ , define the operator  $\mathcal{L}$  by

$$\begin{aligned}\mathcal{L}h(x, i) &= \underbrace{\sum_{j=1}^r b_j(x, i) \frac{\partial h(x, i)}{\partial x_j}}_{\text{drift}} + \underbrace{\frac{1}{2} \sum_{j,k=1}^r a_{jk}(x, i) \frac{\partial^2 h(x, i)}{\partial x_j \partial x_k}}_{\text{diffusion}} + \underbrace{Q(x)h(x, \cdot)(i)}_{\text{jump}} \\ &= \underbrace{b'(x, i)\nabla h(x, i)}_{\text{drift}} + \underbrace{\frac{1}{2} \text{tr}(a(x, i)\nabla^2 h(x, i))}_{\text{diffusion}} + \underbrace{Q(x)h(x, \cdot)(i)},\end{aligned}\quad (2.24)$$

where  $\nabla h(\cdot, i)$  and  $\nabla^2 h(\cdot, i)$  denote the gradient and Hessian of  $h(\cdot, i)$ . The term  $b'(x, i)\nabla h(x, i)$  represents the Euclidean inner product in  $\mathbb{R}^r$ , where  $z'$  denotes the transpose of an arbitrary matrix  $z \in \mathbb{R}^{t_1 \times t_2}$  with  $t_1, t_2 \geq 1$ . Moreover,

$$Q(x)h(x, \cdot)(i) = \sum_{j=1}^{m_0} q_{ij}(x)h(x, j) = \sum_{j \in \mathcal{E}} q_{ij}(x)(h(x, j) - h(x, i)), \quad i \in \mathcal{E}. \quad (2.25)$$

Consider a Markov process  $Y(t) = (X(t), \alpha(t))$  whose associated operator is given by  $\mathcal{L}$ . Note that  $Y(t)$  has two components: an  $r$ -dimensional continuous component  $X(t)$  and a discrete component  $\alpha(t)$  taking values in  $\mathcal{E} = \{1, \dots, m_0\}$ .

The process  $Y(t) = (X(t), \alpha(t))$  may be described by the following pair of equations:

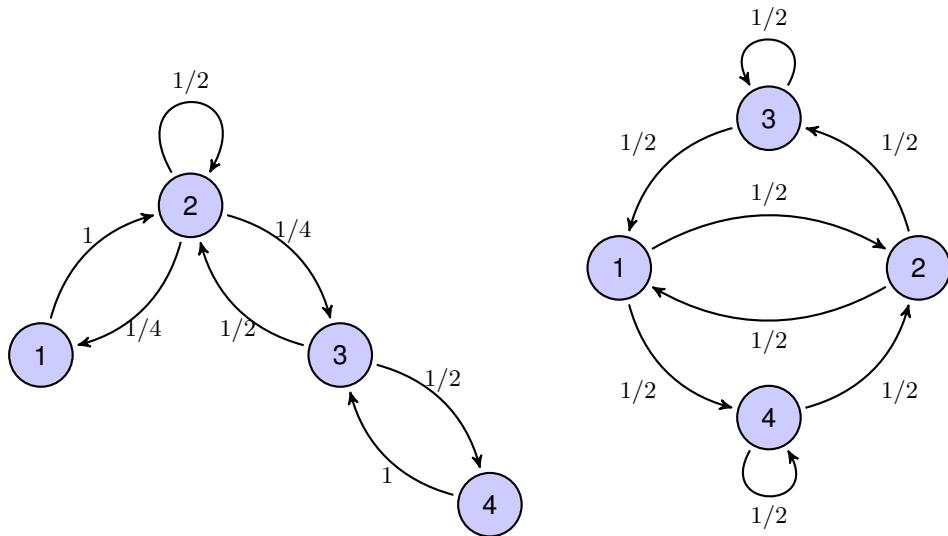
$$\begin{cases} dX(t) = b(X(t), \alpha(t))dt + \sigma(X(t), \alpha(t))dw(t), \\ X(0) = x, \quad \alpha(0) = \alpha, \end{cases} \quad (2.26)$$

and

$$\mathbf{P}\{\alpha(t + \Delta) = j \mid \alpha(t) = i, X(s), \alpha(s), s \leq t\} = q_{ij}(X(t))\Delta + o(\Delta), \quad i \neq j, \quad (2.27)$$

where  $w(t)$  is a  $d$ -dimensional standard Brownian motion,  $b(\cdot, \cdot) : \mathbb{R}^r \times \mathcal{E} \rightarrow \mathbb{R}^r$ , and  $\sigma(\cdot, \cdot) : \mathbb{R}^r \times \mathcal{E} \rightarrow \mathbb{R}^{r \times d}$  satisfies  $\sigma(x, i)\sigma'(x, i) = a(x, i)$ . Note that (2.26) describes the dynamics of the system, whereas (2.27) delineates the probability structure of the jump process. If  $\alpha(\cdot)$  is a continuous-time Markov chain independent of the Brownian motion  $w(\cdot)$  and  $Q(x) = Q$  or  $Q(x) = Q(t)$  (independent of  $x$ ), then (2.26) together with the generator  $Q$  or  $Q(t)$  suffices to characterize the underlying process. However, if there is  $x$ -dependence, (2.27) is necessary to fully describe the dynamics of the switching diffusion.

In this section, our study is carried out using the operator  $\mathcal{L}$  given in (2.24). Throughout the chapter, we assume that for each  $i \in \mathcal{E}$ , both  $b(\cdot, i)$  and  $\sigma(\cdot, i)$  satisfy the usual local Lipschitz and linear growth conditions, and that  $Q(\cdot)$  is bounded and continuous. As described in Theorem 2.1, the system (2.26) and (2.27) has a unique strong solution. Henceforth, we denote the solution of (2.26) and (2.27) by  $(X^{x,\alpha}(t), \alpha^{x,\alpha}(t))$  when referring to the dependence on the initial data. To study the recurrence and ergodicity of the process  $Y(t) = (X(t), \alpha(t))$ , we further assume that the following condition (2.156) holds throughout the chapter. For convenience, we also include the boundedness and continuity of  $Q(\cdot)$  in (2.156).



**Table 2.4.** Two different irreducible Markov chains

**Axiom 2.156.** *The operator  $\mathcal{L}$  satisfies the following conditions. For each  $i \in \mathcal{E}$ , the matrix  $a(x, i) = (a_{jk}(x, i))$  is symmetric and satisfies*

$$\kappa_1 |\xi|^2 \leq \xi' a(x, i) \xi \leq \kappa_1^{-1} |\xi|^2, \quad \text{for all } \xi \in \mathbb{R}^r, \quad (2.28)$$

*with some constant  $\kappa_1 \in (0, 1]$  for all  $x \in \mathbb{R}^r$ . The function  $Q(\cdot) : \mathbb{R}^r \rightarrow \mathbb{R}^{m_0 \times m_0}$  is continuous and bounded. Moreover,  $Q(x)$  is irreducible for each  $x \in \mathbb{R}^r$ .*

### 2.5.1.2 Definitions of Recurrence and Positive Recurrence

This subsection presents the definitions of recurrence, positive recurrence, and null recurrence. First, we introduce the following notation and conventions. For any  $D \subset \mathbb{R}^r$ ,  $J \subset \mathcal{E}$ , and  $U = D \times J \subset \mathbb{R}^r \times \mathcal{E}$ , define

$$\begin{cases} \tau_U &= \inf\{t \geq 0 \mid (X(t), \alpha(t)) \notin U\}, \\ \sigma_U &= \inf\{t \geq 0 \mid (X(t), \alpha(t)) \in U\}. \end{cases} \quad (2.29)$$

In particular, if  $U = D \times \mathcal{E}$  is a cylinder, we set

$$\begin{cases} \tau_D &= \inf\{t \geq 0 \mid X(t) \notin D\}, \\ \sigma_D &= \inf\{t \geq 0 \mid X(t) \in D\}. \end{cases} \quad (2.30)$$

**Definition 2.157 (Recurrence, Positive Recurrence, and Null Recurrence).** *The concepts of recurrence, positive recurrence, and null recurrence are defined as follows:*

- **Recurrence and Transience.** *For  $U = D \times J$ , where  $J \subset \mathcal{E}$  and  $D \subset \mathbb{R}^r$  is an open set with compact closure, let*

$$\sigma_U^{x, \alpha} = \inf \{t \geq 0 \mid (X^{x, \alpha}(t), \alpha^{x, \alpha}(t)) \in U\}.$$

A regular process  $(X^{x,\alpha}(\cdot), \alpha^{x,\alpha}(\cdot))$  is recurrent with respect to  $U$  if  $\mathbf{P}\{\sigma_U^{x,\alpha} < \infty\} = 1$  for all  $(x, \alpha) \in D^c \times \mathcal{E}$ , where  $D^c$  denotes the complement of  $D$ . Otherwise, the process is transient with respect to  $U$ .

■ **Positive Recurrence and Null Recurrence.** A recurrent process  $(X^{x,\alpha}(\cdot), \alpha^{x,\alpha}(\cdot))$  is positive recurrent with respect to  $U = D \times J$  (where  $J \subset \mathcal{E}$  and  $D \subset \mathbb{R}^r$  is a bounded open set with compact closure) if the mean recurrence time  $\mathbf{E}[\sigma_U^{x,\alpha}]$  is finite for some  $(x, \alpha) \in D^c \times \mathcal{E}$ . If the process is recurrent but not positive recurrent, it is called null recurrent with respect to  $U$ .

### 2.5.1.3 Preparatory Results

We begin by proving the following theorem, which establishes that under **Assumption (2.156)**, the process  $Y(t) = (X(t), \alpha(t))$  exits every bounded cylinder with a finite mean exit time.

**Theorem 2.158.** Let  $D \subset \mathbb{R}^r$  be a nonempty open set with compact closure  $\overline{D}$ . Let  $\tau_D = \inf\{t \geq 0 \mid X(t) \notin D\}$ . Then

$$\mathbb{E}_{x,i}(\tau_D) < \infty, \quad \text{for all } (x, i) \in D \times \mathcal{E}.$$

**Remark 2.159.** A careful analysis of the proof reveals that the conclusion of **Theorem 2.158** remains valid under a weaker condition than uniform ellipticity (2.28). Specifically, it suffices that there exists some  $\iota \in \{1, 2, \dots, r\}$  and a positive constant  $\kappa$  such that

$$a_{\iota\iota}(x, i) \geq \kappa \quad \text{for all } (x, i) \in D \times \mathcal{E}.$$

We now recall the definition of  $\mathcal{L}$ -harmonic functions. For any  $U = D \times J$ , where  $D \subset \mathbb{R}^r$  is a nonempty domain and  $J \subset \mathcal{E}$ , a Borel measurable function  $u : U \rightarrow \mathbb{R}$  is called  $\mathcal{L}$ -harmonic in  $U$  if

- $u$  is bounded on compact subsets of  $U$ ;
- For all  $(x, i) \in U$  and any  $V = \widetilde{D} \times \widetilde{J}$  with  $\widetilde{D} \subset D$  being a neighborhood of  $x$  and  $i \in \widetilde{J} \subset J$ , we have

$$u(x, i) = \mathbb{E}_{x,i}(u(X(\tau_V), \alpha(\tau_V))),$$

where  $\tau_V$  denotes the first exit time of  $(X(t), \alpha(t))$  from  $V$ , and  $\widetilde{D} \subset D$  means  $\overline{\widetilde{D}} \subset D$  with  $\widetilde{D} = \widetilde{D} \cup \partial\widetilde{D}$  being compact.

**Lemma 2.160.** For any  $U = D \times J \subset \mathbb{R}^r \times \mathcal{E}$ , where  $D \subset \mathbb{R}^r$  is a nonempty domain, the functions

$$f(x, i) = \mathbf{P}_{x,i}\{\tau_U < \infty\} \quad \text{and} \quad g(x, i) = \mathbb{E}_{x,i}(\varphi(X(\tau_U), \alpha(\tau_U))),$$

are  $\mathcal{L}$ -harmonic in  $U$ , where  $\varphi$  is any bounded Borel measurable function on  $\partial D \times \mathcal{E}$ .

Building upon the classical arguments in [102, Vol. II, Chapter 13], we establish the following lemmas. (We note that **Lemma 2.161** was also proved in [103, Lemma 4.3] and in [104] for the case where  $\mathcal{L}$  is in divergence form.)

**Lemma 2.161.** *Under Assumption (2.156), let  $U = D \times \mathcal{E} \subset \mathbb{R}^r \times \mathcal{E}$  and  $f : U \rightarrow \mathbb{R}$ , where  $D \subset \mathbb{R}^r$  is a nonempty domain. Then*

$$\mathcal{L}f(x, i) = 0 \quad \text{for all } (x, i) \in U, \quad (2.31)$$

*if and only if  $f$  is  $\mathcal{L}$ -harmonic in  $U$ . Furthermore, if  $\partial D$  is sufficiently smooth,  $\overline{D}$  is compact, and  $\varphi(\cdot, i)$  is continuous on  $\partial D$  for each  $i \in \mathcal{E}$ , then*

$$u(x, i) = \mathbb{E}_{x,i} (\varphi(X(\tau_U), \alpha(\tau_U))),$$

*is the unique solution of (2.31) with boundary condition*

$$\lim_{\substack{x \rightarrow x_0 \\ x \in D}} u(x, i) = \varphi(x_0, i) \quad \text{for all } (x_0, i) \in \partial D \times \mathcal{E}.$$

Using similar techniques, we obtain the following result.

**Lemma 2.162.** *Let  $U = D \times \mathcal{E} \subset \mathbb{R}^r \times \mathcal{E}$ , where  $D \subset \mathbb{R}^r$  is a nonempty open set with compact closure. Suppose  $g(\cdot, i) \in C_b(D)$  and  $f : D \times \mathcal{E} \rightarrow \mathbb{R}$ . Then  $f$  solves the boundary value problem*

$$\begin{cases} \mathcal{L}f(x, i) = -g(x, i), & (x, i) \in D \times \mathcal{E}, \\ f(x, i) = 0, & (x, i) \in \partial D \times \mathcal{E}, \end{cases}$$

*if and only if*

$$f(x, i) = \mathbb{E}_{x,i} \left( \int_0^{\tau_D} g(X(t), \alpha(t)) dt \right), \quad \text{for all } (x, i) \in D \times \mathcal{E}.$$

Using **Lemmas 2.161** and **2.162**, we establish that if the process  $Y(t) = (X(t), \alpha(t))$  is recurrent (respectively, positive recurrent) with respect to some cylinder  $D \times \mathcal{E} \subset \mathbb{R}^r \times \mathcal{E}$ , then it is recurrent (respectively, positive recurrent) with respect to any cylinder  $E \times \mathcal{E} \subset \mathbb{R}^r \times \mathcal{E}$ , where  $D$  is an arbitrary nonempty domain in  $\mathbb{R}^r$  with compact closure. These results are formalized in the following lemmas.

**Lemma 2.163.** *Let  $D \subset \mathbb{R}^r$  be an open nonempty set with compact closure. If*

$$\mathbf{P}_{x,i}\{\sigma_D < \infty\} = 1 \quad \text{for all } (x, i) \in D^c \times \mathcal{E},$$

*then for any nonempty open set  $E \subset \mathbb{R}^r$ ,*

$$\mathbf{P}_{x,i}\{\sigma_E < \infty\} = 1 \quad \text{for all } (x, i) \in E^c \times \mathcal{E}.$$

**Lemma 2.164.** *Let  $D \subset \mathbb{R}^r$  be an open nonempty set with compact closure. If*

$$\mathbb{E}_{x,i} (\sigma_D) < \infty \quad \text{for all } (x, i) \in D^c \times \mathcal{E},$$

*then for any nonempty open set  $E \subset \mathbb{R}^r$ ,*

$$\mathbb{E}_{x,i} (\sigma_E) < \infty \quad \text{for all } (x, i) \in E^c \times \mathcal{E}.$$

The following lemma demonstrates that if the process  $Y(t) = (X(t), \alpha(t))$  reaches the cylinder  $D \times \mathcal{E}$  in finite time almost surely under  $\mathbf{P}_{x,i}$ , then it will visit the set  $D \times \{\ell\}$  in finite time almost surely under  $\mathbf{P}_{x,i}$  for any  $\ell \in \mathcal{E}$ .

**Lemma 2.165.** *Let  $D \subset \mathbb{R}^r$  be a nonempty open set with compact closure satisfying*

$$\mathbf{P}_{y,j}\{\sigma_D < \infty\} = 1 \quad \text{for all } (y, j) \in D^c \times \mathcal{E}.$$

*Then for any  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$ ,*

$$\mathbf{P}_{x,i}\{\sigma_{D \times \{\ell\}} < \infty\} = 1 \quad \text{for all } \ell \in \mathcal{E}.$$

Building upon **Lemma 2.165**, we now prove that if  $Y(t) = (X(t), \alpha(t))$  is positive recurrent with respect to some cylinder  $D \times \mathcal{E}$ , then it is positive recurrent with respect to  $D \times \{\ell\} \subset \mathbb{R}^r \times \mathcal{E}$ .

**Lemma 2.166.** *Let  $D \subset \mathbb{R}^r$  be a nonempty open set with compact closure satisfying*

$$\mathbb{E}_{y,j}(\sigma_D) < \infty \quad \text{for all } (y, j) \in D^c \times \mathcal{E}.$$

*Then for any  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$ ,*

$$\mathbb{E}_{x,i}(\sigma_{D \times \{\ell\}}) < \infty \quad \text{for all } \ell \in \mathcal{E}.$$

**Remark 2.167.** *By virtue of **Lemmas 2.163** and **2.166**, under **Assumption (2.156)**, the process  $Y(t) = (X(t), \alpha(t))$  is recurrent (respectively, positive recurrent) with respect to some cylinder  $D \times \mathcal{E}$  if and only if it is recurrent (respectively, positive recurrent) with respect to  $D \times \{\ell\} \subset \mathbb{R}^r \times \mathcal{E}$  for any  $\ell \in \mathcal{E}$ . Moreover, these properties are independent of the choice of  $D$ . We summarize these results in the following theorem.*

**Theorem 2.168.** *Under **Assumption (2.156)**, the following assertions hold:*

- *The process  $(X(t), \alpha(t))$  is recurrent (respectively, positive recurrent) with respect to  $D \times \mathcal{E}$  if and only if it is recurrent (respectively, positive recurrent) with respect to  $D \times \{\ell\}$ , where  $D \subset \mathbb{R}^r$  is a nonempty open set with compact closure and  $\ell \in \mathcal{E}$ .*
- *If  $(X(t), \alpha(t))$  is recurrent (respectively, positive recurrent) with respect to some  $U = D \times \mathcal{E}$ , where  $D \subset \mathbb{R}^r$  is a non-empty open set with compact closure, then it is recurrent (respectively, positive recurrent) with respect to any  $\tilde{U} = \tilde{D} \times \mathcal{E}$ , where  $\tilde{D} \subset \mathbb{R}^r$  is an arbitrary non-empty open set.*

**Remark 2.169.** *In light of **Theorem 2.168**, we make the following observations:*

- *Recurrence is independent of the chosen region; consequently, a process  $(X(t), \alpha(t))$  with generator  $\mathcal{L}$  satisfying **(2.156)** is called recurrent if it is recurrent with respect to some  $U = D \times \{\ell\}$ , where  $D \subset \mathbb{R}^r$  is a nonempty bounded open set and  $\ell \in \mathcal{E}$ ; otherwise, it is called transient.*
- *A recurrent process  $(X(t), \alpha(t))$  is called positive recurrent if it is positive recurrent with respect to some bounded domain  $U = D \times \{\ell\} \subset \mathbb{R}^r \times \mathcal{E}$ ; otherwise, it is called null recurrent.*

## 2.5.2 Recurrence and Transience

### 2.5.2.1 Recurrence

To analyze the recurrence properties of the process  $(X(t), \alpha(t))$ , we first establish a criterion based on the existence of **Lyapunov functions**.

**Theorem 2.170.** *Assume there exists a nonempty bounded open set  $D \subset \mathbb{R}^r$  and a function  $V(\cdot, \cdot) : D^c \times \mathcal{E} \rightarrow \mathbb{R}^+$  satisfying*

$$V_n = \inf_{|x| \geq n, i \in \mathcal{E}} V(x, i) \rightarrow \infty \quad \text{as } n \rightarrow \infty,$$

$$\mathcal{L}V(x, i) \leq 0 \quad \text{for all } (x, i) \in D^c \times \mathcal{E},$$

where  $\mathcal{L}$  denotes the generator of the process. Then the process  $(X(t), \alpha(t))$  is recurrent.

Although Theorem 2.170 provides a general framework, constructing Lyapunov functions can be challenging. The following theorem offers more concrete conditions on the process coefficients.

**Theorem 2.171.** *The process  $(X(t), \alpha(t))$  is recurrent if either of the following conditions holds:*

1. *There exist constants  $\gamma > 0$  and  $c_i \in \mathbb{R}$  for  $i \in \mathcal{E}$  such that for all  $(x, i) \in \{x \in \mathbb{R}^r \mid |x| \geq 1\} \times \mathcal{E}$ ,*

$$\frac{x'b(x, i)}{|x|^2} + \frac{\text{tr}(a(x, i))}{2|x|^2} + (\gamma - 2) \frac{x'a(x, i)x}{2|x|^4} - \frac{1}{k - \gamma c_i} \sum_{j=1}^{m_0} q_{ij}(x)c_j \leq 0,$$

where  $k > 0$  is sufficiently large to ensure  $k - \gamma c_i > 0$  for each  $i \in \mathcal{E}$ .

2. *There exist  $\gamma > 0$  and symmetric positive definite matrices  $P_i$  for  $i \in \mathcal{E}$  such that for all  $(x, i) \in \{x \in \mathbb{R}^r \mid |x| \geq 1\} \times \mathcal{E}$ ,*

$$\frac{x'P_ib(x, i)}{x'P_ix} + \frac{\text{tr}(\sigma'(x, i)P_i\sigma(x, i))}{2x'P_ix} + (\gamma - 2) \frac{|\sigma(x, i)'P_ix|^2}{|x'P_ix|^2} + \sum_{j=1}^{m_0} q_{ij}(x) \frac{|x'P_jx|^{\gamma/2}}{|x'P_ix|^{\gamma/2}} \leq 0.$$

**Lemma 2.172.** *If there exists  $(x_0, \ell) \in \mathbb{R}^r \times \mathcal{E}$  such that for every  $\varepsilon > 0$ ,*

$$\mathbf{P}_{x_0, \ell}\{(X(t_n), \alpha(t_n)) \in B(x_0, \varepsilon) \times \{\ell\} \text{ for some sequence } t_n \uparrow \infty\} = 1, \quad (2.32)$$

then for any  $U = D \times \{j\} \subset \mathbb{R}^r \times \mathcal{E}$  with  $D \subset \mathbb{R}^r$  bounded and  $j \in \mathcal{E}$ ,

$$\mathbf{P}_{x_0, \ell}\{\sigma_U < \infty\} = 1,$$

where  $\sigma_U$  denotes the first hitting time of  $U$ . In particular, if (2.32) holds for all  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$ , then  $(X(t), \alpha(t))$  is recurrent.

**Lemma 2.173.** *If  $(X(t), \alpha(t))$  is recurrent, then for every  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$  and  $\varepsilon > 0$ ,*

$$\mathbf{P}_{x, i}\{(X(t_n), \alpha(t_n)) \in B(x, \varepsilon) \times \{i\} \text{ for some sequence } t_n \uparrow \infty\} = 1.$$

Combining Lemmas 2.172 and 2.173, we obtain the following result.

**Theorem 2.174.** *The process  $(X(t), \alpha(t))$  is recurrent if and only if every  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$  is recurrent, meaning that for all  $\varepsilon > 0$ ,*

$$\mathbf{P}_{x,i}\{(X(t_n), \alpha(t_n)) \in B(x, \varepsilon) \times \{i\} \text{ for some sequence } t_n \uparrow \infty\} = 1.$$

Theorem 2.174 leads to a recurrence criterion in terms of mean sojourn time , inspired by [105]. For any  $U = D \times J \subset \mathbb{R}^r \times \mathcal{E}$  and  $\lambda \geq 0$ , define

$$R_\lambda(x, i, U) = \mathbb{E}_{x,i} \left[ \int_0^\infty e^{-\lambda t} \mathbb{1}_U(X(t), \alpha(t)) dt \right],$$

where  $R_0(x, i, U)$  represents the mean sojourn time in  $U$ .

**Proposition 2.175.** *Under (2.156), if for some  $(x_0, \ell) \in \mathbb{R}^r \times \mathcal{E}$  and all  $\rho > 0$ ,*

$$R_0(x_0, \ell, B(x_0, \rho) \times \{\ell\}) = \infty, \quad (2.33)$$

*then  $(x_0, \ell)$  is recurrent. Moreover, if (2.33) holds for all  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$ , then  $(X(t), \alpha(t))$  is recurrent.*

### 2.5.2.2 Transience

We establish that the process  $(X(t), \alpha(t))$  is transient if and only if the norm of its continuous component satisfies  $|X(t)| \rightarrow \infty$  almost surely as  $t \rightarrow \infty$ . We present two criteria for transience: one in terms of mean stay time and another using **Lyapunov functions** . For systems with linearizable coefficients, we provide verifiable conditions for transience.

**Theorem 2.176.** *The process  $(X(t), \alpha(t))$  is transient if and only if*

$$\lim_{t \rightarrow \infty} |X(t)| = \infty \quad a.s. \quad \mathbf{P}_{x,\alpha} \text{ for all } (x, \alpha) \in \mathbb{R}^r \times \mathcal{E},$$

where  $\mathbf{P}_{x,\alpha}$  denotes the probability measure conditioned on  $(X(0), \alpha(0)) = (x, \alpha)$ .

The proof of Theorem 2.176 follows the approach in [106]. We now present a transience criterion under specific regularity conditions.

**Proposition 2.177.** *Assume the following conditions hold:*

- For each  $i \in \mathcal{E}$ , the coefficients  $b(\cdot, i)$ ,  $\sigma(\cdot, i)$ , and  $Q(\cdot)$  are **Hölder continuous** with exponent  $\gamma \in (0, 1]$ .
- $Q(x)$  is **irreducible** for all  $x \in \mathbb{R}^r$ .
- For each  $i \in \mathcal{E}$ , the matrix  $a(x, i) = \sigma(x, i)\sigma'(x, i)$  satisfies

$$\langle a(x, i)\xi, \xi \rangle \geq \kappa|\xi|^2 \quad \text{for all } \xi \in \mathbb{R}^r,$$

with some constant  $\kappa > 0$  independent of  $x$ .

If for some  $U = D \times J \subset \mathbb{R}^r \times \mathcal{E}$  that contains  $(x_0, \ell)$ , where  $D$  is bounded and open,  $R_0(x_0, \ell, U) < \infty$ , then  $(X(t), \alpha(t))$  is transient.

**Remark 2.178.** *Combining Propositions 2.175 and 2.177, we conclude that  $(X(t), \alpha(t))$  is recurrent if and only if for every  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$  and  $\rho > 0$ ,*

$$R_0(x, i, B(x, \rho) \times \{i\}) = \infty.$$

We now establish a sufficient condition for transience using Lyapunov functions.

**Theorem 2.179.** *Assume that there exists a bounded domain  $D \subset \mathbb{R}^r$  and a function  $V : D^c \times \mathcal{E} \rightarrow \mathbb{R}$  satisfying*

- $\sup_{(x,i) \in \partial D \times \mathcal{E}} V(x,i) \leq 0$ ,
- $\mathcal{L}V(x,i) \geq 0$  for all  $(x,i) \in D^c \times \mathcal{E}$ ,
- $\sup_{(x,i) \in D^c \times \mathcal{E}} V(x,i) \leq M < \infty$ ,
- $V(y,\ell) > 0$  for some  $(y,\ell) \in D^c \times \mathcal{E}$ ,

where  $\mathcal{L}$  is the generator of the process. Then the process  $(X(t), \alpha(t))$  is transient or not regular

For linearizable systems, we introduce an additional assumption.

**Axiom 2.180.** *For each  $i \in \mathcal{E}$ , there exist matrices  $b(i), \sigma_j(i) \in \mathbb{R}^{r \times r}$  ( $j = 1, \dots, d$ ) and an irreducible generator matrix  $\widehat{Q} = (\widehat{q}_{ij})$  such that as  $|x| \rightarrow \infty$*

$$\begin{aligned}\frac{b(x,i)}{|x|} &= b(i) \frac{x}{|x|} + o(1), \\ \frac{\sigma(x,i)}{|x|} &= \sum_{j=1}^d \sigma_j(i) \frac{x}{|x|} + o(1), \\ Q(x) &= \widehat{Q} + o(1),\end{aligned}$$

where  $o(1) \rightarrow 0$  uniformly. The Markov chain  $\widehat{\alpha}(t)$  with generator  $\widehat{Q}$  has a stationary distribution  $\pi = (\pi_1, \dots, \pi_{m_0})$ .

Under these conditions, we obtain a verifiable transience criterion.

**Theorem 2.181.** *Assume (2.156) and (2.180). If for each  $i \in \mathcal{E}$ ,*

$$\lambda_{\min} \left( b(i) + b'(i) + \sum_{j=1}^d \sigma_j(i) \sigma'_j(i) \right) - \frac{1}{2} \sum_{j=1}^d [\rho(\sigma_j(i) + \sigma'_j(i))]^2 > 0,$$

where  $\lambda_{\min}(\cdot)$  denotes the smallest eigenvalue and  $\rho(\cdot)$  is the spectral radius, then  $(X(t), \alpha(t))$  is transient.

## 2.5.3 Positive and Null Recurrence

This section examines positive recurrence using **Lyapunov function** methods. Recall that the process  $Y(t) = (X(t), \alpha(t))$  is recurrent (respectively, positive recurrent) with respect to a cylinder  $D \times \mathcal{E}$  if and only if it is recurrent (respectively, positive recurrent) with respect to  $D \times \{\ell\}$ , where  $D \subset \mathbb{R}^r$  is a non-empty open set with compact closure and  $\ell \in \mathcal{E}$ . These properties are independent of the choice of  $D$  or  $\ell$ .

### 2.5.3.1 General Criteria for Positive Recurrence

**Theorem 2.182.** *The following conditions are equivalent for positive recurrence with respect to  $U = D \times \{\ell\} \subset \mathbb{R}^r \times \mathcal{E}$ :*

1. For each  $i \in \mathcal{E}$ , there exists a nonnegative twice continuously differentiable function  $V(\cdot, i) : D^c \rightarrow \mathbb{R}$  satisfying

$$\mathcal{L}V(x, i) = -1, \quad (x, i) \in D^c \times \mathcal{E},$$

where  $\mathcal{L}$  is the generator of the process.

2. The hitting time  $u(x, i) = \mathbb{E}_{x,i}(\sigma_D)$  is the minimal positive solution of

$$\begin{cases} \mathcal{L}u(x, i) = -1, & (x, i) \in D^c \times \mathcal{E}, \\ u(x, i) = 0, & (x, i) \in \partial D \times \mathcal{E}, \end{cases}$$

where  $\partial D$  denotes the boundary of  $D$ .

**Theorem 2.183.** A necessary and sufficient condition for positive recurrence with respect to  $U = D \times \{\ell\} \subset \mathbb{R}^r \times \mathcal{E}$  is the existence of nonnegative twice continuously differentiable functions  $V(\cdot, i) : D^c \rightarrow \mathbb{R}$  ( $i \in \mathcal{E}$ ) and a constant  $\gamma > 0$  such that

$$\mathcal{L}V(x, i) \leq -\gamma, \quad (x, i) \in D^c \times \mathcal{E}.$$

### 2.5.3.2 Path Excursions

Positive recurrence criteria allow analysis of path excursions. Let  $Y(t) = (X(t), \alpha(t))$  be positive recurrent with Lyapunov functions  $V(x, i)$  from Theorem 2.183 and the associated set  $D$ . For any bounded open set  $D_0$  with  $\overline{D} \subset D_0$ , define

- $\tau_1 = \inf\{t > 0 \mid X(t) \notin D_0\}$  (first exit time).
- $\sigma_1 = \inf\{t > \tau_1 \mid X(t) \in D_0\}$  (first return time).

We obtain the following estimates:

$$\begin{aligned} \mathbf{P}\left(\sup_{\tau_1 \leq t \leq \sigma_1} V(X(t), \alpha(t)) \geq \gamma\right) &\leq \frac{\mathbb{E}[V(X(\tau_1), \alpha(\tau_1))]}{\gamma}, \\ \mathbb{E}(\sigma_1 - \tau_1) &\leq \frac{\mathbb{E}[V(X(\tau_1), \alpha(\tau_1))]}{\gamma}, \end{aligned}$$

where  $\gamma$  is from Theorem 2.183. For  $k \geq 1$ , define recursively

$$\begin{aligned} \tau_{k+1} &= \inf\{t > \sigma_k \mid X(t) \notin D_0\}, \\ \sigma_{k+1} &= \inf\{t \geq \tau_{k+1} \mid X(t) \in D_0\}. \end{aligned}$$

These yield estimates for  $\mathbb{E}(\sigma_{k+1} - \tau_{k+1})$ , characterizing the expected duration of the  $(k+1)$ -st excursion.

### 2.5.3.3 Positive Recurrence under Linearization

This subsection examines positive recurrence for regime-switching diffusions with linearizable continuous components. Linear systems are particularly important in applications because of their tractability.

Building on Theorem 2.184, we establish positive recurrence conditions for systems that satisfy the linearization assumption (2.180).

**Theorem 2.184.** Under assumptions (2.156) and (2.180), if

$$\sum_{i=1}^{m_0} \pi_i \lambda_{\max} \left( b(i) + b'(i) + \sum_{j=1}^d \sigma_j(i) \sigma'_j(i) \right) < 0,$$

where  $\lambda_{\max}(\cdot)$  denotes the largest eigenvalue, then  $(X(t), \alpha(t))$  is positive recurrent.

**Corollary 2.185.** For one-dimensional  $X(t)$  with asymptotic behavior

$$\begin{aligned}\frac{b(x, i)}{|x|} &= b_i \frac{x}{|x|} + o(1), \\ \frac{\sigma(x, i)}{|x|} &= \sigma_i \frac{x}{|x|} + o(1),\end{aligned}$$

where  $b_i, \sigma_i$  are constants, if

$$\sum_{i=1}^{m_0} \pi_i \left( b_i - \frac{\sigma_i^2}{2} \right) < 0,$$

then,  $(X(t), \alpha(t))$  is positive recurrent.

**Theorem 2.186.** Suppose that there exist a bounded domain  $D$  and functions  $V, W : D^c \times \mathcal{E} \rightarrow \mathbb{R}$  satisfying

1.  $V(x, i) \geq 0$  and  $0 \leq \mathcal{L}V(x, i) \leq k$  for all  $(x, i) \in D^c \times \mathcal{E}$ ,
2.  $W(x, i) \leq 0$  on  $\partial D \times \mathcal{E}$  and  $\mathcal{L}W(x, i) \geq 0$  on  $D^c \times \mathcal{E}$ ,
3. For domains  $E_n \supset D$  with  $\Gamma_n = \partial E_n$

$$\frac{\inf_{\Gamma_n \times \mathcal{E}} V(x, i)}{\sup_{\Gamma_n \times \mathcal{E}} W(x, i)} = R_n \rightarrow \infty \text{ as } n \rightarrow \infty.$$

If  $W(x, \alpha) > 0$  for some  $(x, \alpha) \in D^c \times \mathcal{E}$ , then  $(X(t), \alpha(t))$  is not positive recurrent, and  $\mathbb{E}_{x, \alpha}[\sigma_D] = \infty$  for all such  $(x, \alpha)$ .

#### 2.5.3.4 Null Recurrence

**Null recurrence** presents greater analytical challenges. We consider a special case of driftless diffusions:

**Theorem 2.187.** For the regime-switching diffusion

$$\begin{cases} dX(t) = \sigma(X(t), \alpha(t))dw(t), \\ \mathbf{P}\{\alpha(t + \Delta) = j | \alpha(t) = i, \mathcal{F}_t\} = q_{ij}(X(t))\Delta + o(\Delta), \end{cases} \quad (2.34)$$

if for some  $0 \leq \beta \leq 1$  and  $k_1, k_2 > 0$

$$\sigma^2(x, i) \leq k_1|x|^{1-\beta} \quad \forall (x, i) \in \{|x| \geq k_2\} \times \mathcal{E},$$

then  $(X(t), \alpha(t))$  is null recurrent.

## 2.5.4 Ergodicity

### 2.5.4.1 Introduction

This section investigates the ergodic properties of switching-diffusion processes. Many control and optimization problems require the minimization of long-term average costs, which makes ergodicity analysis crucial. When systems possess ergodic properties, time-dependent measures can be replaced by stationary measures for long time horizons. We establish conditions for

ergodicity and characterize ergodic measures through cycle decomposition and induced **Markov chains**.

For background on ergodic control problems in diffusion processes, see [107, 108]. Our approach focuses on positive recurrent systems and their stationary distributions.

#### 2.5.4.2 Ergodic Properties

Consider the process  $Y(t) = (X(t), \alpha(t))$  to be positive and recurrent with respect to  $U = E \times \{\ell\}$ , where  $E \subset \mathbb{R}^r$  has a smooth boundary and  $\ell \in \mathcal{E}$ . Under assumption (2.156), Theorem 2.174 guarantees positive recurrence for all nonempty open sets.

Let  $D \supset \bar{E}$  be a bounded domain with a smooth boundary. Define stopping times

- $s_{2n+1} = \inf\{t > s_{2n} Y(t) \in \partial E \times \{\ell\}\}$  (**entrance time**).
- $s_{2n+2} = \inf\{t > s_{2n+1} Y(t) \in \partial D \times \{\ell\}\}$  (**exit time**).

The sample path is broken down into cycles  $(s_{2n}, s_{2n+2})$ . Positive recurrence ensures that all  $s_n$  are finite a.s. Assuming  $Y(0) = (x, \ell) \in \partial D \times \{\ell\}$ , the sequence  $\{Y_n\}_{n \geq 0}$  with  $Y_n = Y(s_{2n})$  forms a **Markov chain** on  $\partial D \times \{\ell\}$ . Define transition probabilities

$$\tilde{P}(x, A) = P_{(x, \ell)}(Y_1 \in A \times \{\ell\}), \quad A \in \mathcal{B}(\partial D),$$

where  $\mathcal{B}(\partial D)$  denotes the **Borel  $\sigma$ -algebra**.

For any measurable function  $f : \mathbb{R}^r \rightarrow \mathbb{R}$ , let

$$\mathbb{E}_x[f(X_1)] = \int_{\partial D} f(y) \tilde{P}(x, dy).$$

**Lemma 2.188.** *The Markov chain  $\{Y_n\}$  has a unique stationary distribution  $m(\cdot)$  satisfying*

$$|\tilde{P}^{(n)}(x, A) - m(A)| \leq C\lambda^n, \quad \forall A \in \mathcal{B}(\partial D),$$

for some  $C > 0$  and  $\lambda \in (0, 1)$ , where  $\tilde{P}^{(n)}$  denotes the  $n$ -step transition probability.

**Theorem 2.189.** *The process  $Y(t)$  has a unique stationary distribution  $\nu = (\nu(\cdot, i))_{i \in \mathcal{E}}$ .*

**Theorem 2.190.** *Let  $\mu(\cdot, \cdot)$  be the stationary density of  $\nu$ , and let  $f : \mathbb{R}^r \times \mathcal{E} \rightarrow \mathbb{R}$  be measurable with*

$$\sum_{i=1}^{m_0} \int_{\mathbb{R}^r} |f(x, i)| \mu(x, i) dx < \infty.$$

*Then for all  $(x, i) \in \mathbb{R}^r \times \mathcal{E}$ ,*

$$\mathbf{P}_{x, i} \left( \lim_{T \rightarrow \infty} \frac{1}{T} \int_0^T f(X(t), \alpha(t)) dt = \bar{f} \right) = 1,$$

*where  $\bar{f} = \sum_{i=1}^{m_0} \int_{\mathbb{R}^r} f(x, i) \mu(x, i) dx$ .*

**Corollary 2.191.** *For the Cauchy problem*

$$\begin{cases} \frac{\partial u}{\partial t} = \mathcal{L}u, & t > 0, (x, i) \in \mathbb{R}^r \times \mathcal{E}, \\ u(0, x, i) = f(x, i), & f(\cdot, i) \in C_b(\mathbb{R}^r), \end{cases}$$

where  $C_b(\mathbb{R}^r)$  denotes the space of bounded continuous functions , we have

$$\frac{1}{T} \int_0^T u(t, x, i) dt \rightarrow \bar{f} \quad \text{as } T \rightarrow \infty.$$

# CHAPTER 3

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## Exploring the Impact of Jump Perturbations on Stochastic SIRS Dynamics

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### Abstract

This study develops a comprehensive stochastic framework to examine the impact of Lévy perturbations on the dynamics of the SIRS model (SIRS). Initially, we establish the existence and uniqueness of the solution, ensuring a solid foundation for our analysis. We identify the critical conditions for the persistence of the disease that are essential for evaluating the applicability of the model in real-world scenarios. In addition, we determine the criteria for disease extinction. To support our theoretical findings, we conducted extensive numerical simulations.

### Keywords

Stochastic SIRS model, Lévy jumps, persistence, extinction, Lyapunov function, threshold.

### 3.1 Introduction

During the early 20th century, epidemiology underwent a significant transformation, driven by the groundbreaking contributions of eminent scientists such as Anderson Gray McKendrick and William Ogilvy Kermack. Their pioneering work introduced the concept of mathematical modeling, which has since evolved into an indispensable tool in the field. This mathematical approach has profoundly impacted outbreak and epidemic management, playing a key role in guiding evidence-based public health interventions.

Epidemiology has evolved significantly, thanks to notable physicians such as Quinto Tiberio Angelerio, who demonstrated remarkable proficiency in managing the plague outbreak in Alghero, Sardinia, in 1582. However, the emergence of modern epidemiology as a formal scientific discipline occurred during the 19th century. Often referred to as the "father of modern epidemiology," John Snow made a meaningful breakthrough when he meticulously traced a devastating cholera outbreak in London to water contamination from the Broad Street pump. This groundbreaking investigation marked a pivotal moment that laid the foundation for contemporary epidemiology.

Epidemiology is a scientific discipline that investigates epidemics, diseases, and various

health-related conditions. Its roots can be traced to ancient Greece, notably through the influential work of Hippocrates of Kos, who made notable contributions by distinguishing between epidemic and endemic diseases. Epidemiology, in its broader scope, also includes the study of diseases affecting plants, domestic animals, and livestock. An epidemic is characterized by a significant and abnormal occurrence of disease within a population, usually manifesting rapidly.

Many factors influence the intricate process of transmission of the disease [109–113], which includes the characteristics of the infectious agent and the complex dynamics of the host population. Regarding the infectious agent, its inherent characteristics, such as its mode of transmission (e.g., respiratory droplets, direct contact), the duration of infectivity, and its responsiveness to medical interventions such as treatments and vaccines, are crucial factors that determine its ability to spread among individuals. Equally important are the elements of the host population that influence the dynamics of the epidemic. Factors such as social interactions, demographics (e.g., age, sex), cultural practices, geographic distribution, and economic conditions are pivotal in determining a population's susceptibility and resilience in the face of disease.

Throughout recorded history, human civilization has faced recurrent epidemics and pandemics. These outbreaks of infectious diseases have caused significant human suffering, societal upheaval, and economic turmoil. Given these challenges, accurately predicting the progression of outbreaks becomes paramount in minimizing their adverse impacts. Epidemiological modeling is fundamental to understand the dynamics of disease transmission and to formulate informed containment and prevention strategies.

Infectious diseases continue to have substantial effects on communities worldwide, despite advances in modern medicine and medical science. These impacts extend to various aspects, including public health, healthcare infrastructure, the economy, education, population dynamics, and international cooperation.

The investigation of epidemic models holds considerable importance across multiple domains due to its numerous advantages. These benefits include better understanding and forecasting of epidemics, optimization of public health resource allocation, more effective emergency response planning, advancement of public health research, increased public awareness, and the development of training programs in public health. This facilitates informed public health decision making, improves epidemic readiness, and ultimately contributes to saving lives.

This problem has attracted mathematicians since the 18th century. Kermack and McKendrick established the classical deterministic SIR model in 1927 [114], which served as a fundamental framework for analyzing epidemic dynamics within a closed population, categorizing individuals into three compartments. The SIR model, which delimits individuals into susceptible ( $S$ ), infected ( $I$ ), and recovered ( $R$ ) compartments, does not account for individuals who lose immunity post-recovery. To address this deficiency, an extended version known as the SIRS model has been introduced [115–117]. In the SIRS model, people who have previously recovered from an infection may become susceptible again.

Mathematically, this phenomenon is expressed as follows:

$$\begin{cases} dS_t = [\rho(1 - S_t) + \eta R_t - \alpha S_t I_t] dt, \\ dI_t = [\alpha S_t I_t - (\rho + \lambda) I_t] dt, \\ dR_t = [\lambda I_t - (\rho + \eta) R_t] dt, \end{cases} \quad (3.1)$$

where  $\rho$  represents the birth and death rate,  $\alpha$  is the infection coefficient,  $\lambda$  is the recovery rate, and  $\eta$  is the immunity loss rate.

Later, many studies used the stochastic approach to analyze these models [118]. Adding Lévy perturbations to the SIRS model can enrich epidemiological modeling by better capturing the complexity of real epidemic phenomena, including stochastic variations and rare events that significantly impact disease dynamics. This can provide valuable information for public health

decision making and epidemic management. The reader can refer to [119, 120]. In this study, we examine a system modeled as a stochastic SIRS model incorporating a jump perturbation:

$$\begin{cases} dS_t = [\rho(1 - S_t) + \eta R_t - \alpha S_t I_t] dt - \int_{\mathbb{D}} \varsigma_v S_{(t^-)} I_{(t^-)} \tilde{N}(dt, dv), \\ dI_t = [\alpha S_t I_t - (\rho + \lambda) I_t] dt + \int_{\mathbb{D}} \varsigma_v S_{(t^-)} I_{(t^-)} \tilde{N}(dt, dv), \\ dR_t = [\lambda I_t - (\rho + \eta) R_t] dt, \end{cases} \quad (3.2)$$

where  $dt$  is the Lebesgue measure, and  $\tilde{N}(dt, dv)$  is the compensated Poisson measure, such that

$$\tilde{N}(dt, dv) = N(dt, dv) - \pi(dv)dt.$$

Here,  $N(dt, dv)$  denotes a Poisson counting measure, while  $\pi$  represents a Lévy measure defined on  $\mathbb{D} \subset \mathbb{R}^+$ . The continuously differentiable function  $\varsigma(\cdot)$  characterizes the impact of random jumps within the population, with  $-1 < \varsigma_v < 1$  for every  $v \in \mathbb{D}$ .

The terms  $S_{(s^-)}$  and  $I_{(s^-)}$  represent the left-hand limits of  $S_s$  and  $I_s$ , respectively. Henceforth, we will refer to these simply as  $S_s$  and  $I_s$  for practicality.

This work stems from the need to improve traditional epidemiological models by incorporating Lévy perturbations, which account for sudden, random changes in disease dynamics. The research aims to better understand the persistence and extinction of diseases by developing a more realistic stochastic SIRS model, leading to improved strategies for managing infectious diseases in unpredictable environments.

The manuscript is organized as follows. Section 3.2 discusses the positivity and existence of solutions for system (3.2). Section 3.3 derives the necessary conditions for the persistence of the disease. Section 3.4 analyzes the extinction of disease. Section 3.5 presents numerical simulations to validate the analytical results. Section 3.6 provides a detailed discussion of the results and future directions. Finally, Section 3.7 concludes with a summary of the findings and research implications.

## 3.2 Positivity and Existence

This section explores the global existence and positivity of solutions for the stochastic differential equation (SDE) system (3.2).

**Definition 3.1 (Meyer Angle Bracket).** Let  $(M_t)_{t \geq 0}$  be a continuous local martingale. The Meyer angle-bracket, or the predictable quadratic variation of  $M$ , denoted by  $\langle M \rangle_t$ , is the unique predictable increasing process such that  $M_t^2 - \langle M \rangle_t$  is a local martingale.

**Lemma 3.2.** Let  $M_t$  be a local martingale starting from zero at time 0. For  $t \geq 0$ , define

$$\varphi_{M_t} := \int_0^t (1+s)^{-2} d\langle M \rangle_s.$$

Then,

$$\mathbf{P} \left( \lim_{t \rightarrow \infty} \frac{M_t}{t} = 0 \right) = 1,$$

provided that

$$\mathbf{P} \left( \lim_{t \rightarrow \infty} \varphi_{M_t} < \infty \right) = 1.$$

**Proof.** See [121]. ■

We first establish the existence and uniqueness of solutions to our model within the domain:

$$\Delta = \{(x_1, x_2, x_3) \in (0, 1)^3; x_1 + x_2 + x_3 = 1\},$$

with parameters  $(\alpha, \eta, \lambda, \rho) \in (0, 1)^4$ .

**Theorem 3.3.** For  $v \in \mathbb{D}$  and  $(S, I) \in (0, 1)^2$ , define

$$\Psi(v, S, I) = [1 - \varsigma_v I] [1 + \varsigma_v S],$$

and assume

$$\sup_{0 < S, I < 1} \int_{\mathbb{D}} \ln [\Psi^{-1}(v, S, I)] \pi(dv) < \infty. \quad (3.3)$$

Then, for each initial value  $(S_0, I_0, R_0) \in \Delta$ , system (3.2) admits a unique solution  $(S_t, I_t, R_t) \in \Delta$  for all  $t \geq 0$ .

**Proof.** Let  $N_t = S_t + I_t + R_t$  represent the total population. From (3.2), we derive:

$$dN_t = -\rho(N_t - 1)dt.$$

Integrating yields:

$$N_t - 1 = (N_0 - 1)e^{-\rho t} \quad \text{a.s.},$$

which implies:

$$(S_t, I_t, R_t) \in (0, 1)^3 \text{ and } N_t = 1 \text{ a.s. for all } t \geq 0. \quad (3.4)$$

Since the coefficients are locally Lipschitz continuous [117, 122–124], there exists a unique maximal local solution  $(S_t, I_t, R_t)$  on  $[0, \tau_e]$ , where  $\tau_e$  is the explosion time. For  $\epsilon > 0$ , define the stopping time:

$$\tau_\epsilon = \inf\{t \in [0, \tau_e) \mid \min(S_t, I_t, R_t) \leq \epsilon\}. \quad (3.5)$$

Consider the Lyapunov function:

$$\Sigma_t = -\ln(S_t I_t R_t).$$

Applying Itô's formula yields:

$$\begin{aligned} d\Sigma_t &= \left( 3\rho + \lambda + \eta - \frac{\rho}{S_t} + \alpha I_t - \frac{\eta R_t}{S_t} - \alpha S_t - \frac{\lambda I_t}{R_t} \right) dt \\ &\quad - \int_{\mathbb{D}} \{ \ln [(1 + \varsigma_v S_t)(1 - \varsigma_v I_t)] + (I_t - S_t)\varsigma_v \} \pi(dv) dt \\ &\quad - \int_{\mathbb{D}} \ln [(1 + \varsigma_v S_t)(1 - \varsigma_v I_t)] \tilde{N}(dt, dv). \end{aligned}$$

Using (3.3) and (3.4), we obtain:

$$\begin{aligned} d\Sigma_t &\leq \left[ \kappa + \sup_{0 < S, I < 1} \int_{\mathbb{D}} \ln [\Psi^{-1}(v, S, I)] \pi(dv) \right] dt \\ &\quad - \int_{\mathbb{D}} \ln [(1 + \varsigma_v S_t)(1 - \varsigma_v I_t)] \tilde{N}(dt, dv), \end{aligned} \quad (3.6)$$

where  $\kappa = 3\rho + \lambda + \eta + \alpha + \pi(\mathbb{D})$ .

Integrating and taking expectations gives:

$$\mathbb{E}[\Sigma_{t \wedge \tau_\epsilon}] \leq \Sigma_0 + \kappa t - 3 \ln(\epsilon_0). \quad (3.7)$$

If  $\tau_e < \infty$ , there exists  $t > 0$  such that  $\mathbf{P}(\tau_e < t) > 0$ , implying:

$$-\ln(\epsilon) \mathbf{P}(\tau_e \leq t) \leq \mathbb{E}[\Sigma_{t \wedge \tau_\epsilon}]. \quad (3.8)$$

Combining (3.7) and (3.8) yields:

$$\mathbf{P}(\tau_e \leq t) \leq \frac{3}{\ln(\epsilon)} [\ln(\epsilon_0) - \kappa t].$$

Letting  $\epsilon \rightarrow 0$  leads to  $\mathbf{P}(\tau_e \leq t) = 0$ , contradicting  $\tau_e < \infty$ . Thus,  $\tau_e = \infty$  a.s. ■

The next section examines the persistence of the disease and determines the threshold conditions for long-term control of the disease.

### 3.3 Persistence

In epidemiological studies of the disease, it is essential to focus on the cases in which it persists and does not disappear spontaneously, as these cases can provide valuable information on its long-term impact and treatment. In what follows, we will demonstrate the persistence of the disease [125–127]. Let us define

$$\begin{cases} H(S) = -(\rho + \lambda) + \alpha S - \left[ \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right] S^2, \\ \mathcal{T}^1 = \alpha \left[ \rho + \lambda + \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right]^{-1}, \\ \Pi(S) = -(\rho + \lambda) + \alpha S - \left[ \frac{1}{2} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right] S^2, \\ \mathcal{T}^2 = \alpha \left[ \rho + \lambda + \frac{1}{2} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right]^{-1}. \end{cases} \quad (3.9)$$

**Theorem 3.4.** *Let (3.3) and*

$$\sup_{0 < y < 1} \int_{\mathbb{D}} \ln^2 [1 + \varsigma_v y] \pi(dv) < \infty, \quad (3.10)$$

*hold. For  $(S_0, I_0, R_0) \in \Delta$ , if  $\mathcal{T}^1 > 1$ ,  $\mathcal{T}^2 > 1$  and  $|\varsigma_v| < 1$  for each  $v \in \mathbb{D}$ , then*

- i)**  $\limsup_{t \rightarrow \infty} S_t \geq \varrho$ , a.s.,
- ii)**  $\liminf_{t \rightarrow \infty} I_t \leq (\rho + \eta)(\rho + \eta + \lambda)^{-1}(1 - \varrho)$ , a.s.,
- iii)**  $\liminf_{t \rightarrow \infty} R_t \leq \lambda(\rho + \eta + \lambda)^{-1}(1 - \varrho)$ , a.s.,
- iv)**  $\liminf_{t \rightarrow \infty} S_t \leq \varrho'$ , a.s.,
- v)**  $\limsup_{t \rightarrow \infty} I_t \geq (\rho + \eta)(\rho + \eta + \lambda)^{-1}(1 - \varrho')$ , a.s.,
- vi)**  $\limsup_{t \rightarrow \infty} R_t \geq \lambda(\rho + \eta + \lambda)^{-1}(1 - \varrho')$ , a.s.,

where  $\varrho$  and  $\varrho'$  denote the positive roots on the interval  $(0, 1)$  of equations  $H(S) = 0$  and  $\Pi(S) = 0$ , respectively.

**Remark 3.5.** Since  $-1 < \varsigma_v < 1$  for all  $v \in \mathbb{D}$ , it follows that for all  $S \in (0, 1)$ ,  $\Pi(S) < H(S)$ , and therefore  $\varrho < \varrho'$ .

**Proof.**

(i) From (3.2) and using Itô's formula, one obtains

$$\begin{aligned} \ln(I_t) &= \ln(I_0) - \int_0^t [(\rho + \lambda) - \alpha S_s] ds + \int_{\mathbb{D}} \int_0^t [\ln(1 + \varsigma_v S_s) - \varsigma_v S_s] \pi(dv) \\ &\quad + \int_{\mathbb{D}} \int_0^t \ln[1 + \varsigma_v S_s] \tilde{N}(ds, dv). \end{aligned} \quad (3.11)$$

Applying the inequality:

$$\ln(1 + x) - x < -\frac{x^2}{4}, \quad -1 < x \leq 1, \quad (3.12)$$

we obtain

$$\ln(I_t) \leq \ln(I_0) + \int_0^t H(S_s) ds + \int_{\mathbb{D}} \int_0^t \ln[1 + \varsigma_v S_s] \tilde{N}(ds, dv). \quad (3.13)$$

Since  $H(0) = -(\rho + \lambda) < 0$  and when  $\mathcal{T}^1 > 1$ , it follows that  $H(1) > 0$ . Consequently, equation  $H(S) = 0$  possesses a unique root  $\varrho \in (0, 1)$ . If  $\mathcal{T}^1 > 1$ , then  $H'(S_0) = 0$  for some  $S_0 > 1$ . Therefore,  $H$  increases monotonically in the interval  $(0, 1)$ , particularly in  $(0, \varrho)$ . For any sufficiently small  $\epsilon > 0$ , when  $0 < S \leq \varrho - \epsilon$ , we have

$$H(S) \leq H(\varrho - \epsilon) < 0. \quad (3.14)$$

Now, we prove the assertion (i). Assume the contrary, implying that there exists a sufficiently small  $\epsilon > 0$  such that

$$\mathbf{P}\left[\limsup_{t \rightarrow \infty} S_t \leq \varrho - 2\epsilon\right] > 0.$$

Define

$$\Omega_1 = \left\{\limsup_{t \rightarrow \infty} S_t \leq \varrho - 2\epsilon\right\}.$$

For every  $\omega \in \Omega_1$ , there exists  $\tau(\omega) > 0$  such that

$$S_t \leq \varrho - \epsilon < 1, \quad \text{for each } t \geq \tau(\omega). \quad (3.15)$$

By (3.14) and (3.15), we get for each  $s \geq \tau(\omega)$ ,

$$H(S_s) \leq H(\varrho - \epsilon) < 0. \quad (3.16)$$

By equation (3.10) and Lemma (3.2), we can easily prove the existence of a set  $\Omega_2 \subset \Omega$  with  $\mathbf{P}(\Omega_2) = 1$ , where for each  $\omega \in \Omega_2$ ,

$$\lim_{t \rightarrow \infty} \int_{\mathbb{D}} \int_0^t \frac{\ln[1 + \varsigma_v S_s]}{t} \tilde{N}(ds, dv) = 0. \quad (3.17)$$

Now, fix any  $\omega \in \Omega_1 \cap \Omega_2$ . By (3.13) and (3.16), for  $t \geq \tau(\omega)$ , we obtain

$$\begin{aligned} \ln(I_t) - \ln(I_0) &\leq \int_0^{\tau(\omega)} H(S_s)ds + \int_{\tau(\omega)}^t H(\varrho - \epsilon)ds \\ &\quad + \int_{\mathbb{D}} \int_0^t \ln[1 + \varsigma_v S_s] \tilde{N}(ds, dv). \end{aligned} \quad (3.18)$$

From (3.17) and (3.18), we deduce

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{\ln(I_t)}{t} &\leq H(\varrho - \epsilon) \\ &< 0. \end{aligned} \quad (3.19)$$

Thus,

$$\lim_{t \rightarrow \infty} I_t = 0. \quad (3.20)$$

By integrating the last equation of (3.2), we obtain

$$R_t = R_0 \exp\{-(\rho + \eta)t\} + \lambda \int_0^t I_{(t-s)} \exp\{-(\rho + \eta)s\} ds. \quad (3.21)$$

Using (3.21) and Fatou's lemma, we deduce

$$\limsup_{t \rightarrow \infty} R_t \leq \lambda(\rho + \eta)^{-1} \limsup_{t \rightarrow \infty} I_t. \quad (3.22)$$

Combined with (3.20), this implies  $\lim_{t \rightarrow \infty} R_t = 0$  and thus  $\lim_{t \rightarrow \infty} S_t = 1$ . However, this contradicts (3.15).

(iv) Similarly, based on Itô's formula as in (3.11) and applying

$$-\frac{x^2}{2} \leq \ln(1 + x) - x, \text{ for each } x \geq 0,$$

we obtain

$$\ln(I_t) \geq \ln(I_0) + \int_0^t \Pi(S_s)ds + \int_{\mathbb{D}} \int_0^t \ln[1 + \varsigma_v S_s] \tilde{N}(ds, dv).$$

Suppose (iv) is false, implying there exists a sufficiently small  $\epsilon' > 0$  with  $\mathbf{P}(\Omega_3) > 0$ , where

$$\Omega_3 = \left\{ \liminf_{t \rightarrow \infty} S_t \geq \varrho' + 2\epsilon' \right\}.$$

For each  $\omega \in \Omega_3$ , there exists  $\tau'(\omega) > 0$  such that

$$S_t \geq \varrho' + \epsilon', \text{ for each } t \geq \tau'(\omega). \quad (3.23)$$

Similarly to (3.16), it is straightforward to verify, by selecting  $\epsilon' > 0$  sufficiently small, that

$$\Pi(S_s) \geq \Pi(\varrho' + \epsilon') > 0, \text{ for } s \geq \tau'(\omega). \quad (3.24)$$

Using (3.17), (3.23), (3.24), and following a similar reasoning as in (3.18), we obtain

$$\limsup_{t \rightarrow \infty} \frac{\ln(I_t)}{t} \geq \Pi(\varrho' + \epsilon') > 0.$$

Thus,

$$\lim_{t \rightarrow \infty} I_t = \infty.$$

This contradicts condition  $I_t < 1$ .

(ii) By (i) and (3.4), we have

$$\liminf_{t \rightarrow \infty} I_t + \liminf_{t \rightarrow \infty} R_t \leq 1 - \varrho, \text{ a.s..} \quad (3.25)$$

Using (3.21) and Fatou's lemma yields

$$\liminf_{t \rightarrow \infty} I_t \leq \lambda^{-1}(\rho + \eta) \liminf_{t \rightarrow \infty} R_t. \quad (3.26)$$

Combining (3.25) and (3.26), we establish the assertion (ii).

(v) Similar to (ii), the conclusion follows from (3.22), (iv), and (3.4).

(iii)-(vi) These results follow directly from (3.4), (i), (iv), (ii), and (v). ■

In the next section, we will analyze the extinction of the stochastic differential equation (SDE) (3.2) to determine the critical threshold necessary for disease control or complete eradication.

## 3.4 Extinction

This section examines the extinction phenomenon in the SDE system (3.2).

**Theorem 3.6.** *Let  $(S_0, I_0, R_0) \in \Delta$  and (3.3) hold. Assume that*

$$\sup_{0 < y < 1} \int_{\mathbb{D}} \ln^2 [1 + \varsigma_v y] \pi(dv) < \infty. \quad (3.27)$$

We define the new threshold

$$\mathcal{T}^3 = \alpha \left[ \rho + \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right]^{-1}, \quad (3.28)$$

and

$$\mathcal{T}^4 = \frac{1}{2} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv). \quad (3.29)$$

If  $\mathcal{T}^3 < 1$  and  $\alpha \geq \mathcal{T}^4$ , then the system governed by (3.2) exhibits extinction with an exponential decay rate.

**Proof.** Let

$$\Sigma_t(Z_t) = \ln(Z_t),$$

where  $Z_t = I_t + R_t$ . Using the Itô formula, one obtains

$$\begin{aligned} d\Sigma_t &= \frac{1}{Z_t} [-\rho I_t - (\rho + \eta)R_t + \alpha S_t I_t] dt + \int_{\mathbb{D}} \left[ \ln \left( 1 + \varsigma_v \frac{S_t I_t}{Z_t} \right) - \varsigma_v \frac{S_t I_t}{Z_t} \right] \pi(dv) dt \\ &\quad + \int_{\mathbb{D}} \ln \left( 1 + \varsigma_v \frac{S_t I_t}{Z_t} \right) \tilde{N}(dt, dv). \end{aligned} \quad (3.30)$$

Using (3.12) and the following inequalities:

$$-1 < \varsigma_v < 1, \quad \frac{SI}{Z} \leq 1, \quad (3.31)$$

and

$$\frac{1}{Z} [-\rho I - (\rho + \eta)R] \leq -\rho.$$

Thus

$$\begin{aligned} d\Sigma_t &\leq \left[ -\rho + \alpha \frac{S_t I_t}{Z_t} - \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \left( \frac{S_t I_t}{Z_t} \right)^2 \right] dt + \int_{\mathbb{D}} \ln \left( 1 + \varsigma_v \frac{S_t I_t}{Z_t} \right) \tilde{N}(dt, dv) \\ &\leq \sup_{0 < \delta \leq 1} \Phi(\delta) dt + \int_{\mathbb{D}} \ln \left( 1 + \varsigma_v \frac{S_t I_t}{Z_t} \right) \tilde{N}(dt, dv), \end{aligned} \quad (3.32)$$

where

$$\Phi(\delta) = -\rho + \alpha\delta - \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \delta^2.$$

If  $\alpha \geq \frac{1}{2} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv)$ , then  $\Phi$  increases on  $(0, 1)$ . By integration, we get

$$\Sigma_t \leq \Sigma_0 + \int_0^t \left( -\rho + \alpha - \frac{1}{4} \int_{\mathbb{D}} \varsigma_v^2 \pi(dv) \right) ds + M_t, \quad (3.33)$$

where  $M_t$  is a real-valued local martingale such that

$$M_t = \int_{\mathbb{D}} \int_0^t \ln \left( 1 + \varsigma_v \frac{S_s I_s}{Z_s} \right) \tilde{N}(ds, dv).$$

Moreover,

$$\begin{aligned} \langle M \rangle_t &= \int_{\mathbb{D}} \int_0^t \ln^2 \left( 1 + \varsigma_v \frac{S_s I_s}{Z_s} \right) \pi(dv) ds \\ &\leq \left[ \sup_{0 < y < 1} \int_{\mathbb{D}} \ln^2(1 + \varsigma_v y) \pi(dv) \right] t < \infty. \end{aligned}$$

From (3.27) and (3.2), we get

$$\limsup_{t \rightarrow \infty} \frac{M_t}{t} = 0 \quad \text{a.s..} \quad (3.34)$$

The assertion is validated with (3.33) and (3.34). ■

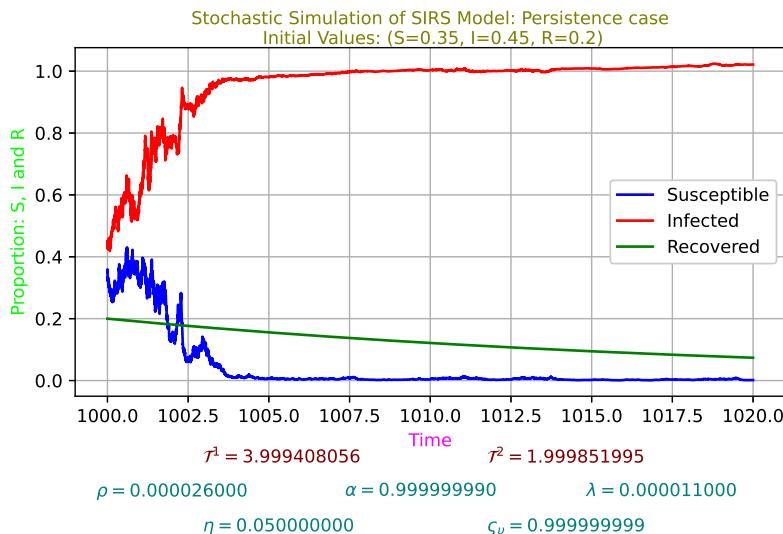
Next, we will examine the computer simulations of SDE (3.2) to validate and reinforce our theoretical findings.

## 3.5 Computer Simulations

In this section, we propose a few examples of numerical computer simulations to illustrate the theoretical results of Theorem 3.4 for persistence and Theorem 3.6, which prove the extinction of the disease by using the Maruyama Euler method (see, e.g., [128] for further information and the references cited therein).

### 3.5.1 Graphical representations in case of persistence Theorem 3.4

The graphical representation plots the proportions of susceptible ( $S(t)$ ), infected ( $I(t)$ ), and recovered ( $R(t)$ ) individuals over time. Each case specified in the case list corresponds to a set of parameter values defining the transmission rate  $\rho$ , the infection rate  $\alpha$ , the recovery rate  $\lambda$ , the reintroduction rate  $\eta$ , and the volatility parameter  $\varsigma$ . For each case, the model is simulated and the results are plotted on separate graphs, with time on the **x-axis** and the proportion of individuals on the **y-axis**. The plots provide insight into how different combinations of parameters affect disease dynamics, allowing for comparative analysis. The Python code that generates the graphs in the following is referenced as A.10.



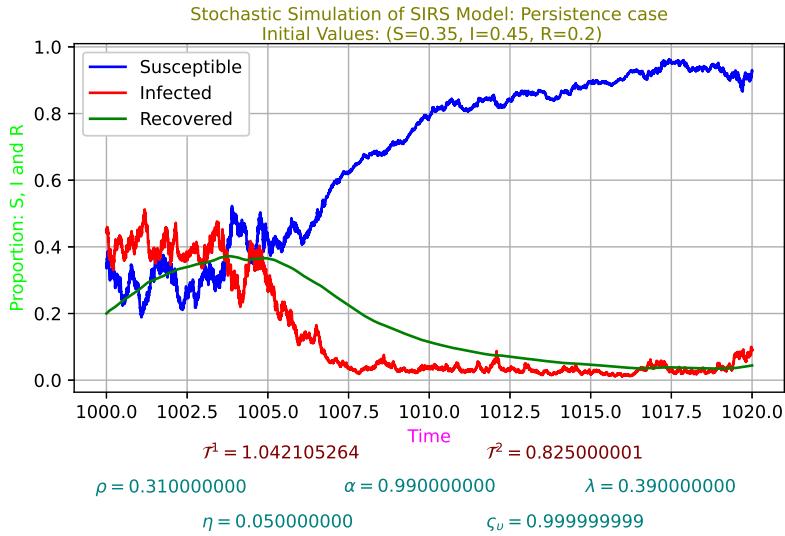
**Figure 3.1.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.4.

### 3.5.2 Analysis, comparison and interpretation in case of persistence Theorem 3.4

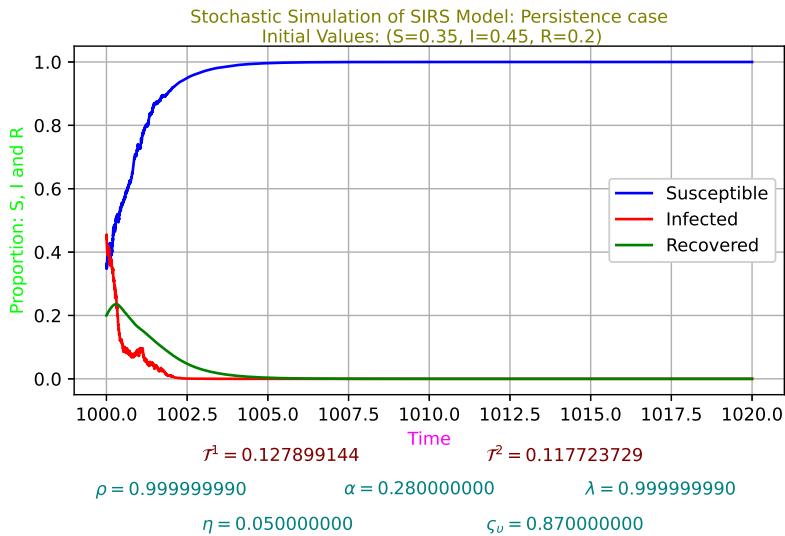
Variations in the dynamics of susceptible, infected, and recovered populations are analyzed with respect to the values of  $T^1$  and  $T^2$ , particularly when  $T^1 > 1$  and  $T^2 > 1$  (see Figures 3.1 and 3.4), and in the case of negation of these conditions (see Figures 3.2 and 3.3). The parameters  $T^1$  and  $T^2$  are derived from the model equations and represent critical thresholds related to transmission, infection, and recovery rates.

### 3.5. COMPUTER SIMULATIONS

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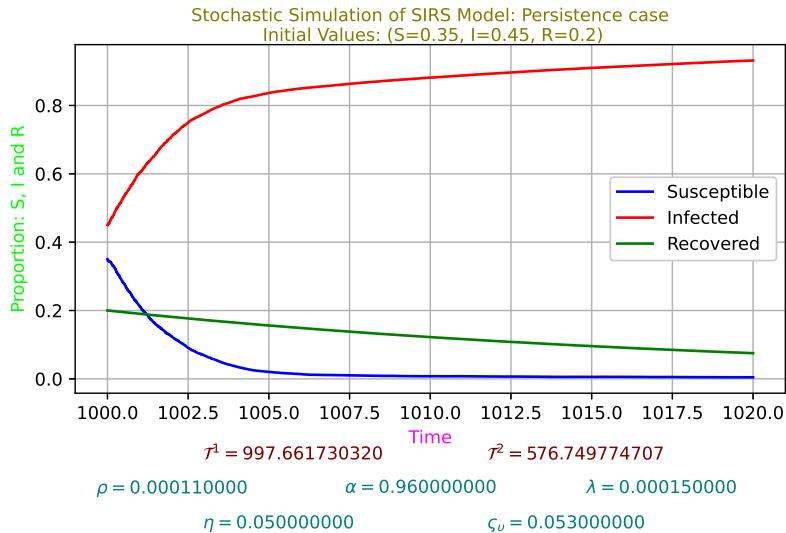


**Figure 3.2.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.4.



**Figure 3.3.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.4.

By comparing the simulations under different parameter conditions, insights are gained into how parameter changes impact the disease spread, including the emergence of endemic or epidemic behaviors.



**Figure 3.4.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.4.

### 3.5.3 Graphical representations in case of Theorem extinction 3.6

The graphical representation illustrates the proportions of susceptible ( $S(t)$ ), infectious ( $I(t)$ ), and recovered ( $R(t)$ ) individuals over time for each simulated scenario. Each scenario corresponds to a unique combination of parameter values, including the transmission rate  $\rho$ , the infection rate  $\alpha$ , the recovery rate  $\lambda$ , the reintroduction rate into the susceptible population  $\eta$ , and the volatility  $\zeta$ . The plots visually demonstrate how variations in these parameters influence the dynamics of disease spread, allowing a comparative analysis between different scenarios. The figures below, generated by the Python code referenced as A.11, provide a clear representation of these relationships.

### 3.5.4 Analysis, comparison, and interpretation in case of Theorem extinction 3.6

Variations in susceptible, infectious, and recovered populations are analyzed in relation to the values of  $T^3$  and  $T^4$ , particularly when  $T^3 < 1$  and  $T^4 \leq \alpha$  (see Figure 3.6), and when these conditions are negated (see Figures 3.5, 3.7 and 3.8). The parameters  $T^3$  and  $T^4$  are derived from the model equations and represent critical thresholds related to transmission and infection rates. By comparing simulations under different parameter conditions, insight is gained into how changes in these parameters impact the disease dynamics, including the emergence of endemic or epidemic behaviors.

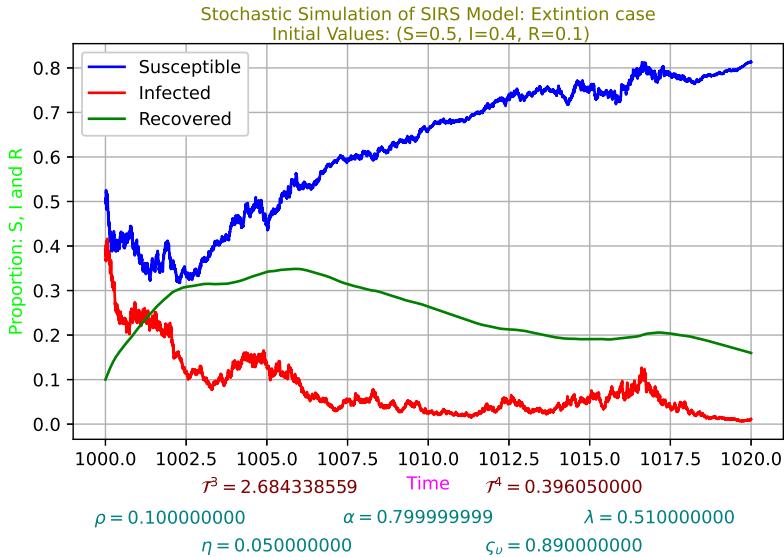
## 3.6 Perspective

In recent years, the study of stochastic dynamics in epidemiology has gained significant traction, particularly in understanding the complexities of infectious disease transmission.

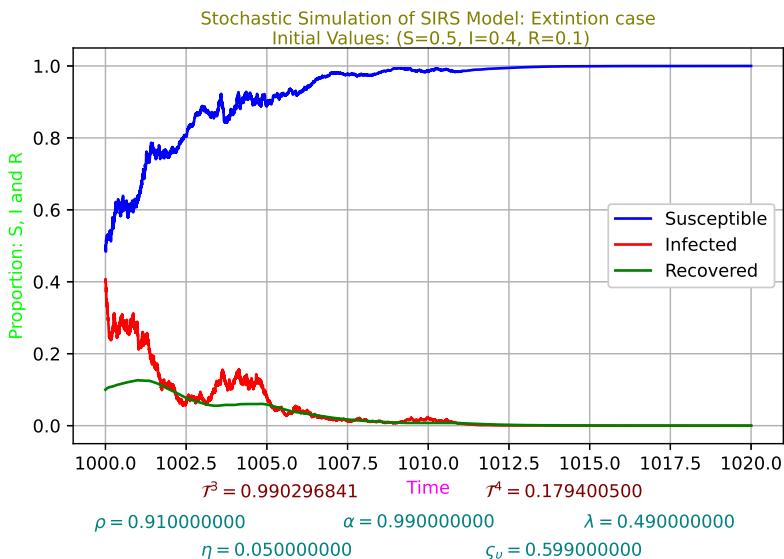
One such model, the stochastic SIRS model (*SIRS*), provides a valuable framework for

### 3.6. PERSPECTIVE

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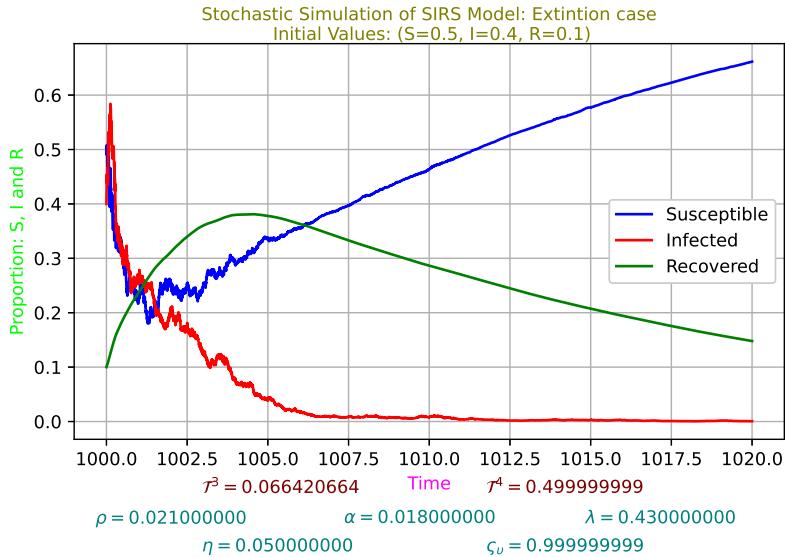
**Figure 3.5.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.6.



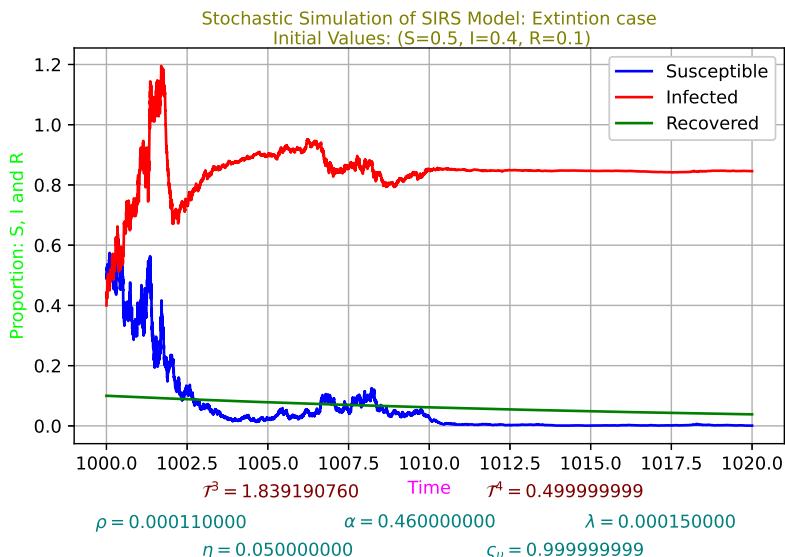
**Figure 3.6.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.6.

investigating the dynamics of infectious diseases in populations where individuals move between susceptible states ( $S$ ), infectious states ( $I$ ), and recovered ( $R$ ) states.

However, traditional SIRS models often assume continuous transitions between these states, overlooking the potential impact of sudden large-scale perturbations, or “jumps.” Exploring the effects of jump perturbations on stochastic SIRS dynamics presents a novel avenue for under-



**Figure 3.7.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.6.



**Figure 3.8.** Trajectories of  $S_t$ ,  $I_t$ , and  $R_t$  for system (3.2) with respect to Theorem 3.6.

standing the behavior of infectious diseases in real-world scenarios. Unlike gradual transitions, jump perturbations can arise from various factors, such as sudden changes in environmental conditions, mass gatherings, or interventions such as vaccination campaigns or policy changes. These perturbations can significantly alter the dynamics of disease transmission, leading to counterintuitive outcomes and challenging traditional modeling assumptions.

### **3.7. CONCLUSION**

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From a scientific perspective, investigating jump perturbations in stochastic SIRS dynamics offers insights into the resilience and vulnerability of populations to abrupt changes in disease dynamics. Researchers can capture the inherent uncertainty and variability observed in real-world epidemiological data by incorporating stochasticity and jump processes into SIRS models. This allows for more accurate predictions of disease spread, improved assessment of intervention strategies, and better understanding of the underlying mechanisms driving disease emergence and persistence.

Professionally, this research has far-reaching implications for public health policy, infectious disease surveillance, and epidemic preparedness. Understanding how jump perturbations influence stochastic SIRS dynamics can inform the development of more effective disease control measures and response strategies. By identifying critical thresholds, tipping points, and high-risk scenarios, policymakers and public health officials can proactively mitigate the impact of sudden perturbations and minimize the risk of epidemic outbreaks or resurgence.

In conclusion, exploring the impact of jump perturbations on stochastic SIRS dynamics represents a valuable interdisciplinary endeavor at the intersection of epidemiology, mathematics, and complex systems science [129–135]. Through rigorous scientific inquiry and collaboration, researchers can advance our understanding of infectious disease dynamics, enhance predictive capabilities, and ultimately contribute to more resilient and adaptive public health systems.

## **3.7 Conclusion**

In conclusion, the stochastic SIRS model with jump perturbation provides a valuable framework for exploring and understanding the dynamics of infectious diseases with abrupt changes in transmission rates. This model offers insights into the potential impact of sporadic events or interventions on the spread of disease within a population. Its mathematical rigor and stochastic nature make it a valuable tool for researchers and policymakers to evaluate and design disease control and prevention strategies in dynamic and uncertain environments. Further research and refinement of this model may continue to contribute to our understanding of epidemiological processes and the development of effective public health measures.

# CHAPTER 4

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## Necessary and Sufficient Criteria for Stochastic SIRS Epidemic Models Under Switched Transmission Rate Exponents

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### Abstract

This paper examines a nonlinear SIRS epidemic model that incorporates white noise and telegraphic noise, with environmental conditions governed by a finite-state Markov chain. The model includes a switched exponent nonlinearity in the bilinear transmission rate, where the interaction between susceptible and infected populations is described by the term  $S^{\rho_{\xi(t)}} I^{\zeta_{\xi(t)}}$ , providing a more realistic representation of the dynamics of the epidemic. Notably, the necessary and sufficient conditions for the persistence and extinction of the stochastic SIRS epidemic model, incorporating transmission rate exponents governed by Markovian switching, have not been explored in the literature. The core contribution of this work lies in identifying these necessary and sufficient conditions. Specifically, a threshold parameter  $\Lambda$ , expressed in terms of the switching exponents  $\rho_{\xi(t)}$  and  $\zeta_{\xi(t)}$ , is derived. That is, if  $\Lambda > 0$ , the disease exhibits strong stochastic persistence; conversely, if  $\Lambda < 0$ , the disease-free equilibrium state becomes globally asymptotically stable in probability, leading to eventual disease extinction. In the special case where there is no regime switching and  $\rho_{\xi(t)} = \zeta_{\xi(t)} = 1$ , our model recovers the classical threshold found in the literature. To complement the theoretical analysis, numerical simulations are presented to illustrate the model's behavior under varying environmental conditions.

### Keywords

SIRS epidemic model, hybrid switching diffusion, nonlinear incidence rate, Markov chains, disease extinction, asymptotic stability, almost sure stability, moment stability, stochastic persistence.

### 4.1 Introduction

Infectious diseases have caused some of the most significant crises in human history. The effort to understand and manage these diseases has relied on a multidisciplinary approach, with

## 4.1. INTRODUCTION

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mathematical modeling playing an essential role in analyzing epidemic dynamics and their progression [136, 137]. These models serve as a structured method for examining the transmission and development of diseases in human populations, enabling accurate predictions and supporting informed public health strategies.

In 1760, Bernoulli pioneered the use of mathematical models in epidemiology, specifically to analyze the dynamics of smallpox transmission and the effects of inoculation. This groundbreaking work laid the foundation for later models, such as the compartmental approach introduced by Kermack and McKendrick in 1927 [138]. Their SIR model classifies the population into three distinct compartments: susceptible ( $S$ ), infected ( $I$ ), and removed or recovered ( $R$ ). The model posits that individuals in the susceptible group become infected upon contact with infectious individuals, whereas those infected either recover and gain lasting immunity or are removed from the population. This framework has been widely explored and expanded in epidemiological studies [139].

Although the traditional SIR model assumes permanent immunity after recovery, real-world evidence indicates that immunity to certain diseases, such as influenza [140], viral diarrheal infections [141], and foot and mouth disease [142], can be temporary. In these instances, recovered individuals can eventually lose immunity and rejoin the susceptible population, prompting the development of the SIRS model (Susceptible-Infected-Recovered-Susceptible) to better capture these dynamics [143].

The incidence rate plays an essential role in understanding the spread of infectious diseases and has been the subject of significant research. In particular, non-linear incidence rates have offered a more realistic representation of epidemic dynamics. In this context, the SIRS model with non-linear incidence functions reveals a rich spectrum of dynamic behaviors, including bifurcations, oscillations, and multiple equilibria. This framework improves our understanding of epidemic dynamics by capturing the complexities of disease spread under varying assumptions.

Although standard incidence rates have been thoroughly analyzed in studies such as [144–146], saturation incidence rates of the form  $\beta SI$  have received attention in works such as [147]. In addition, alternative non-linear formulations, including  $\beta S^\rho I^\zeta$ , have been introduced to better represent the intricacies of disease transmission dynamics [148–150]. For a broader perspective on general incidence functions, foundational contributions can be found in [151–155].

The nonlinear incidence function  $\beta S^\rho I^\zeta$  was investigated by Liu et al. [156, 157] within the framework of the following system of differential equations:

$$\begin{cases} \frac{dS}{dt} &= \mu - \mu S - \beta S^\rho I^\zeta + \eta R, \\ \frac{dI}{dt} &= -(\mu + \delta)I + \beta S^\rho I^\zeta, \\ \frac{dR}{dt} &= -(\mu + \eta)R + \delta I, \end{cases}$$

where:

- $\mu$  represents the natural death rate of the population (per capita death rate),
- $\beta$  denotes the infection transmission rate,
- $\rho$  describes the effect of the susceptible population  $S$  on the transmission dynamics ,
- $\zeta$  represents the effect of the infected population  $I$  on the transmission dynamics ,
- $\eta$  is the recovery rate from the recovered compartment  $R$  to the susceptible compartment  $S$  ,
- $\delta$  is the recovery rate from the infected compartment  $I$  to the recovered compartment  $R$ .

The authors highlight significant changes in solution behavior as the incidence rate transitions from the classical bilinear mass action form,  $\beta IS$ , to a generalized nonlinear rate,  $\beta I^\zeta S^\rho$ . Although choosing a  $\rho$  value different from 1 has minimal effect, selecting a  $\zeta$  value different from 1 can induce significant qualitative changes in the phase space portrait. The primary lesson of

Liu et al. [156, 157] is that the behavior of solutions is governed by the following quantities:

$$\begin{aligned}\mathcal{T}^* &= \frac{(\zeta + \rho - 1)^{\zeta + \rho - 1}}{(H(\zeta - 1))^{\zeta - 1} \rho^\rho}, \\ \mathcal{T} &= \frac{\beta}{\mu + \delta}, \\ H &= \frac{\mu + \eta}{\delta + \mu + \eta}.\end{aligned}$$

That is, for  $\zeta = 1$ , if  $\mathcal{T} \leq 1$ , the disease dies out; when  $\mathcal{T} > 1$ , the disease remains endemic and approaches a unique equilibrium. For  $0 < \zeta < 1$ , the threshold condition vanishes entirely, as the disease always remains endemic and approaches a unique equilibrium. For  $\zeta > 1$ , the threshold result becomes more complex. Specifically:

- If  $\mathcal{T} < \mathcal{T}^*$ , the disease always dies out.
- If  $\mathcal{T} > \mathcal{T}^*$ , the smaller non-trivial equilibrium is an unstable saddle.
- For  $\mathcal{T} > \mathcal{T}^*$  and  $\zeta > 1$ , the disease dies out for some initial conditions, but remains endemic and approaches the larger nontrivial equilibrium for others.
- When  $\mathcal{T} \leq 1$ , the trivial equilibrium is locally asymptotically stable for  $\zeta > 1$ . As  $\zeta$  decreases through 1, the trivial equilibrium transitions to an unstable saddle, while a locally asymptotically stable nontrivial equilibrium emerges through bifurcation.
- When  $\mathcal{T} > 1$ , the trivial equilibrium is an unstable saddle for  $\zeta < 1$ . As  $\zeta$  increases through 1, the trivial equilibrium becomes locally asymptotically stable, and an unstable nontrivial equilibrium bifurcates from it.
- For  $\zeta > 1$ , a saddle-node bifurcation occurs at  $\mathcal{T} = \mathcal{T}^*$ . For  $\mathcal{T} > \mathcal{T}^*$ , the smaller nontrivial equilibrium is a saddle, while the larger nontrivial equilibrium may be locally asymptotically stable in some cases and unstable in others.

In the study of infectious disease dynamics, the parameters of the SIRS model are not constant but are influenced by environmental variability. To account for these fluctuations, stochastic approaches have been developed to include random perturbations. Lu [118] extended the classical SIRS model by incorporating stochastic perturbations into the infection rate. Specifically, the infection coefficient  $\beta$  is replaced by  $\beta + \sigma \frac{dW}{dt}$ , where  $W$  represents a Brownian motion, and  $\sigma$  denotes the intensity of the noise. This perturbation leads to the following system:

$$\begin{cases} dS_t &= [\mu - \mu S_t - \beta S_t I_t + \eta R_t] dt - \sigma S_t I_t dW_t, \\ dI_t &= [-(\mu + \delta)I_t + \beta S_t I_t] dt + \sigma S_t I_t dW_t, \\ dR_t &= [-(\mu + \eta)R_t + \delta I_t] dt. \end{cases} \quad (4.1)$$

Numerous researchers have studied the SIRS model with Brownian perturbations, investigating its stability and dynamic behaviors. In [158–162], the authors analyzed various aspects of the model, including its equilibrium states and the impact of stochastic effects on the dynamics of the epidemic. Building on these foundational studies, the SIRS model (4.1) was further expanded by Lahrouz et al. [163], who introduced a saturated incidence rate and disease-related mortality. They also established global stability conditions for the equilibrium state  $E_0$ , both in probability and at the  $q$ -th moment.

In [164], Lahrouz and Settati refined the earlier findings in the literature by deriving the necessary condition that governs the dichotomy between the extinction and persistence of the epidemic. Additionally, they formulated an exact expression for the critical thresholds that determine the asymptotic dynamics of the SIRS system, represented by the following stochastic threshold:

$$\mathcal{R}_s \triangleq \frac{\beta}{\mu + \delta + \frac{1}{2}\sigma^2}.$$

## 4.1. INTRODUCTION

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Beyond white noise, environmental fluctuations can be modeled using colored noise, such as telegraph noise [165–169]. This type of noise reflects the alternations between different environmental regimes, which can correspond to variations in factors such as nutrition, climate, or sociocultural conditions. These transitions are typically memoryless, with waiting times between regime changes following an exponential distribution, thereby introducing additional complexity into the modeling of infectious disease dynamics.

Recent studies have significantly improved our understanding of the SIRS model by incorporating non-linear incidence rates. Notable contributions include those by Cui and Zhao [170], Wang et al. [171], Liu et al. [172], Pan et al. [173], and Wang et al. [174]. However, to our knowledge, none of these studies have considered a non-linear binomial-type incidence rate, wherein the exponents of the susceptible and infected populations are strictly positive and coupled through a Markov chain.

To address this gap, we propose an innovative formulation for the incidence rate that incorporates a generalized non-linear rate, expressed as  $S^{\rho_{\xi(t)}} I^{\zeta_{\xi(t)}}$ , where the exponents are strictly positive and depend on the state of the Markov chain. We analyze the asymptotic behavior of the system under stochastic perturbations and establish the necessary and sufficient conditions for disease extinction and persistence. This approach provides valuable information on the long-term effects of environmental fluctuations on epidemic outcomes.

Telegraph noise, characterized by abrupt transitions between environmental states, influences disease transmission rates, either amplifying or mitigating the spread. These regime shifts are modeled as transitions in a finite-state Markov chain. To begin with, let  $\xi(t)$  denote a Markov chain defined in a finite state space  $\mathcal{E} = \{1, 2, \dots, r\}$  with the generator matrix  $\Gamma = (\gamma_{ij})_{1 \leq i, j \leq r}$ . The transition probabilities over a short time interval  $\varsigma > 0$  are given by

$$\mathbf{P}(\xi(t + \varsigma) = j | \xi(t) = i) = \begin{cases} \gamma_{ij}\varsigma + o(\varsigma), & \text{if } i \neq j, \\ 1 + \gamma_{ii}\varsigma + o(\varsigma), & \text{if } i = j, \end{cases} \quad \text{where } \gamma_{ii} = -\sum_{j \neq i} \gamma_{ij}. \quad (4.2)$$

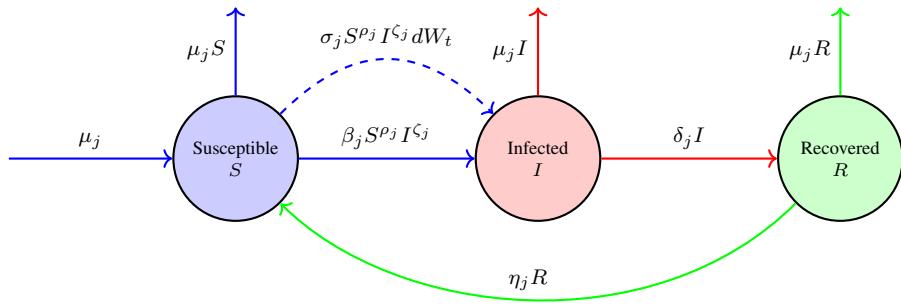
Assuming irreducibility of the Markov chain  $\xi(t)$ , the system admits a unique stationary distribution  $\pi = (\pi_1, \pi_2, \dots, \pi_r) \in \mathbb{R}^r$ . Consequently, we present a stochastic SIRS model characterized by a non-linear incidence rate, with its parameters dynamically governed by the state of the Markov chain  $\xi(t)$ . The dynamics of the model are characterized by the following system of differential equations:

$$\begin{cases} dS_t &= \left( \mu_{\xi(t)} - \mu_{\xi(t)} S_t - \beta_{\xi(t)} S_t^{\rho_{\xi(t)}} I_t^{\zeta_{\xi(t)}} + \eta_{\xi(t)} R_t \right) dt - \sigma_{\xi(t)} S_t^{\rho_{\xi(t)}} I_t^{\zeta_{\xi(t)}} dW_t, \\ dI_t &= \left( -(\mu_{\xi(t)} + \delta_{\xi(t)}) I_t + \beta_{\xi(t)} S_t^{\rho_{\xi(t)}} I_t^{\zeta_{\xi(t)}} \right) dt + \sigma_{\xi(t)} S_t^{\rho_{\xi(t)}} I_t^{\zeta_{\xi(t)}} dW_t, \\ dR_t &= (-(\mu_{\xi(t)} + \eta_{\xi(t)}) R_t + \delta_{\xi(t)} I_t) dt. \end{cases} \quad (4.3)$$

For each state  $i \in \mathcal{E}$ , the parameters  $\mu_i, \beta_i, \rho_i, \zeta_i, \eta_i, \delta_i$  and  $\sigma_i$  represent positive constants, retaining the same meanings as previously defined.

Initially, at time  $\tau_0$ , if the Markov chain is in state  $\xi(\tau_0) = i_0$  and transits, in time  $\tau_k$ , to a new state  $\xi(\tau_k) = j$ , where  $j \in \mathcal{E}$ , the system (4.3) is well described by the following stochastic differential equations under regime switching:

$$\begin{cases} dS_t &= \left( \mu_j - \mu_j S_t - \beta_j S_t^{\rho_j} I_t^{\zeta_j} + \eta_j R_t \right) dt - \sigma_j S_t^{\rho_j} I_t^{\zeta_j} dW_t, \\ dI_t &= \left( -(\mu_j + \delta_j) I_t + \beta_j S_t^{\rho_j} I_t^{\zeta_j} \right) dt + \sigma_j S_t^{\rho_j} I_t^{\zeta_j} dW_t, \\ dR_t &= (-(\mu_j + \eta_j) R_t + \delta_j I_t) dt. \end{cases} \quad (4.4)$$



**Figure 4.1.** Diagram of the SIRS model with a non-linear exponent in the transmission rate

The primary challenge in analyzing such a stochastic model lies in determining the conditions under which the epidemic either dies out or persists within the population. Until now, the necessary and sufficient conditions for the persistence and extinction of the system (4.3) have not been explored in the literature. The primary objective of this study is to rigorously establish these necessary and sufficient conditions for the stochastic SIRS epidemic model (4.3), which incorporates transmission rate exponents governed by Markovian switching dynamics. Our work presents the first comprehensive and rigorous demonstration of these conditions, addressing a significant gap in understanding the mechanisms of extinction and persistence in such systems.

This work is organized as follows. In Section 4.2, we present the formulation of the problem and outline the auxiliary results that serve as the foundation for the subsequent analysis. Section 4.3 explores the conditions under which the epidemic model (4.3) becomes extinct, employing various convergence criteria. In Section 4.4, we establish the criteria required for the persistence of the epidemic. Numerical simulations are provided in Section 4.5 to illustrate and validate the theoretical findings. Finally, Section 4.6 concludes with a summary of the main results and a discussion of their broader implications.

## 4.2 Formulation and auxiliary results

We denote  $\mathbb{R}_+^3 = \{x \mid x_i > 0, i = 1, 2, 3\}$ , and  $\Delta = \{x \in \mathbb{R}_+^3 \mid x_1 + x_2 + x_3 = 1\}$ .

Let  $X(t)$  represent a regular time-homogeneous Markov process in  $\mathbb{R}_+^3$ , which can be characterized by the following stochastic differential equations.

$$dX(t) = b(X(t), \xi(t)) dt + h(X(t), \xi(t)) dW(t), \quad (4.5)$$

where  $X(t) = (S(t), I(t), R(t))$ . Here,  $W(t)$  is a standard Brownian motion defined in the complete probability space  $(\Omega, \mathcal{F}, (\mathcal{F}_t)_{t \geq 0}, \mathbf{P})$ , and  $(\xi(t))_{t \geq 0}$  is a right-continuous Markov chain in the same probability space, taking values in the finite state space  $\mathcal{E} = \{1, 2, \dots, r\}$ . The Markov chain is characterized by the generator matrix  $\Gamma = (\gamma_{ij})_{1 \leq i, j \leq r}$ , as defined in equations (4.2).

Following the framework introduced by Zhu and Yin [175], the diffusion matrix  $A(x, j)$  for each  $j \in \mathcal{E}$  and  $x \in \mathbb{R}_+^3$  is given by

$$A(x, j) = h(x, j)h^T(x, j).$$

For the purpose of the subsequent analysis, we introduce the generator  $\mathcal{L}$  associated with the SDE system in equation (4.5). For each  $j \in \mathcal{E}$ ,  $x \in \mathbb{R}_+^3$  and any twice continuously differentiable

## 4.2. FORMULATION AND AUXILIARY RESULTS

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function  $V(x, j)$ , the generator  $\mathcal{L}$  is defined as

$$\mathcal{L}V(x, j) = b^T(x, j) \cdot \nabla V(x, j) + \frac{1}{2} \text{Tr} (h^T(x, j) \cdot \nabla^2 V(x, j) \cdot h(x, j)) + \Gamma(x)V(x, \cdot)(j),$$

where

$$\Gamma(x)V(x, \cdot)(j) = \sum_{k \neq j, k \in \mathcal{E}} \gamma_{jk} (V(x, k) - V(x, j)).$$

To ensure the biological relevance of the model (4.3), it is crucial to show that the solution remains within the set  $\Delta$ , this defines the feasible region for the proportion of population. Following the approaches of Khasminskii et al. [176], Yuan and Mao [177], Han and Zhao [178] and Lah et al. [163], we can easily establish the following theorem. For the sake of brevity, the proof is omitted.

**Theorem 4.1.** *The set  $\Delta$  is almost surely positively invariant in the system (4.3). That is, for any initial condition  $(S_0, I_0, R_0, \xi_0) \in \Delta \times \mathcal{E}$ , we have*

$$\mathbf{P}((S(t), I(t), R(t), \xi(t)) \in \Delta \times \mathcal{E}, \text{ for all } t \geq 0) = 1.$$

Let us introduce the following lemma, which will be instrumental in proving the extinction condition. To begin with, let us consider, for  $a, b > 0$ , the function  $f_{(a,b)}$  defined by

$$f_{(a,b)}(x, y) = x^a y^b, \quad \text{for } x, y \in \mathbf{D},$$

where

$$\mathbf{D} = \{(x, y) \in \mathbb{R}_+^2 ; x + y \leq 1\}.$$

**Lemma 4.2.** *Let  $a, b > 0$ . The function  $f_{(a,b)}$ , defined in the domain  $\mathbf{D}$ , attains its global maximum at the critical point  $\left(\frac{a}{a+b}, \frac{b}{a+b}\right)$ . The maximum value of  $f_{(a,b)}$  at this critical point is given by*

$$f_{(a,b)}\left(\frac{a}{a+b}, \frac{b}{a+b}\right) = \frac{a^a b^b}{(a+b)^{a+b}}.$$

Moreover, the function  $f_{(a,b)}$  attains the global minimum 0 when  $x = 0$  or  $y = 0$ , and we have the following inequalities, for  $x \in \left(0, \frac{a}{a+b}\right]$  and  $y \in \left(0, \frac{b}{a+b}\right]$ ,

$$\begin{aligned} f_{(a,b)}(x, y) &\leq f_{(a,b)}\left(x, \frac{b}{a+b}\right) \leq f_{(a,b)}\left(\frac{a}{a+b}, \frac{b}{a+b}\right), \\ f_{(a,b)}(x, y) &\leq f_{(a,b)}\left(\frac{a}{a+b}, y\right) \leq f_{(a,b)}\left(\frac{a}{a+b}, \frac{b}{a+b}\right). \end{aligned}$$

**Proof.** Consider the set  $\mathbf{D} = \{(x, y) \in \mathbb{R}_+^2 \mid x + y \leq 1\}$ . The closure of  $\mathbf{D}$ , denoted  $\overline{\mathbf{D}}$ , is both closed and bounded in  $\mathbb{R}^2$ , implying that  $\overline{\mathbf{D}}$  is compact by the Heine-Borel theorem. The function  $f_{(a,b)}(x, y) = x^a y^b$  is continuous on  $\overline{\mathbf{D}}$ . Consequently,  $f_{(a,b)}$  is bounded on  $\overline{\mathbf{D}}$  and attains its

extrema in this compact domain. On the boundary of  $\mathbf{D}$ , where  $x = 0$  or  $y = 0$ , we observe that  $f_{(a,b)}(x, y) = 0$ . Moreover, since the partial functions  $f_{(a,b)}(x, \cdot)$  and  $f_{(a,b)}(\cdot, y)$  increase with respect to  $x$  and  $y$ , the maximum of  $f_{(a,b)}$  must occur along the boundary of the line  $x + y = 1$  within the domain  $\mathbf{D}$ . To determine the critical points of  $f_{(a,b)}$ , we compute its partial derivatives. Differentiating with respect to  $x$ , we compute:

$$\begin{aligned}\frac{df_{(a,b)}(x, y)}{dx} &= \frac{\partial f_{(a,b)}(x, y)}{\partial x} + \frac{\partial y}{\partial x} \frac{\partial f_{(a,b)}(x, y)}{\partial y} \\ &= ax^{a-1}y^b - bx^ay^{b-1}.\end{aligned}$$

Setting this derivative equal to zero and using the constraint  $x + y = 1$ , we obtain

$$x = \frac{a}{a+b} \quad \text{and} \quad y = \frac{b}{a+b}.$$

Therefore, the critical point is  $\left(\frac{a}{a+b}, \frac{b}{a+b}\right)$ . Next, we evaluate  $f_{(a,b)}$  along the boundary of  $\mathbf{D}$ , where  $x + y = 1$ . At the critical point, we have

$$f_{(a,b)}\left(\frac{a}{a+b}, \frac{b}{a+b}\right) = \frac{a^ab^b}{(a+b)^{a+b}}.$$

It is clear that as  $x \rightarrow 0$  or  $y \rightarrow 0$ , the function tends to zero, that is,  $f_{(a,b)}(0, y) = 0$  or  $f_{(a,b)}(x, 0) = 0$ . By the Extreme Value Theorem, since  $f_{(a,b)}$  is continuous on  $\overline{\mathbf{D}}$ , it attains a global maximum and a global minimum in  $\overline{\mathbf{D}}$ . Therefore, the global maximum  $\frac{a^ab^b}{(a+b)^{a+b}}$  occurs at the point  $\left(\frac{a}{a+b}, \frac{b}{a+b}\right)$ , and the global minimum 0 is attained at the boundary of  $\mathbf{D}$ . ■

To analyze the stability properties of the disease-free equilibrium, we invoke the following result based on Lyapunov functions.

**Lemma 4.3.** *If there exist a Lyapunov function  $V \in \mathcal{C}^2(\mathbb{R}^3 \times \mathcal{E}; \mathbb{R}^+)$  and a function  $w : \mathbb{R}^3 \rightarrow \mathbb{R}^+$  that is zero only at the equilibrium point  $E_0$ . If the following conditions are satisfied*

$$\mathcal{L}V(x, j) \leq -w(x), \quad \text{for all } (x, j) \in \mathbb{R}^3 \times \mathcal{E}, \quad \text{and} \quad \lim_{|x| \rightarrow \infty} \inf_{j \in \mathcal{E}} V(x, j) = \infty,$$

*then, the equilibrium state  $E_0$  of the system (4.3) is globally asymptotically stable in probability.*

This lemma provides a sufficient condition for global asymptotic stability in probability, a concept rigorously formulated by Khasminskii et al. [176]. Next, we derive the threshold condition, denoted by  $\Lambda$ , between extinction and persistence of the disease, expressed in terms of the model parameters as:

$$\Lambda = \begin{cases} \sum_{j=1}^r \pi_j \tilde{\Lambda}_j, & \text{if } \zeta_j < 1, \\ \sum_{j=1}^r \pi_j \bar{\Lambda}_j, & \text{if } \zeta_j \geq 1, \end{cases} \quad (4.6)$$

where

$$\tilde{\Lambda}_j = \frac{\beta_j^2}{2(1 - \zeta_j)\sigma_j^2} - (\mu_j + \delta_j), \quad (4.7)$$

$$\bar{\Lambda}_j = \begin{cases} \frac{\beta_j^2}{2\sigma_j^2} - (\mu_j + \delta_j), & \text{if } \frac{\beta_j}{\sigma_j^2} \leq \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}, \\ \Theta_j \left( \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}} \right), & \text{if } \frac{\beta_j}{\sigma_j^2} > \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}, \end{cases} \quad (4.8)$$

and

$$\Theta_j(x) = -\frac{1}{2}\sigma_j^2 x^2 + \beta_j x - (\mu_j + \delta_j), \quad \forall x \in \mathbb{R}, \quad j \in \mathcal{E}.$$

## 4.3 Extinction

In this section, we analyze the extinction condition of the system (4.3). To begin with, let us cite the following theorem investigating the global stability of the disease-free equilibrium.

### 4.3.1 Global Stability of the Disease-Free Equilibrium state

**Theorem 4.4.** *Let  $(S_0, I_0, R_0, \xi_0) \in \Delta \times \mathcal{E}$ . The disease-free equilibrium state  $E_0$  is globally asymptotically stable in probability if one of the following conditions is satisfied*

i) If  $\zeta_j < 1$ , for all  $j \in \mathcal{E}$ , and

$$\Lambda = \sum_{j=1}^r \pi_j \tilde{\Lambda}_j < 0. \quad (4.9)$$

ii) If  $\zeta_j \geq 1$ , for all  $j \in \mathcal{E}$ , and

$$\Lambda = \sum_{j=1}^r \pi_j \bar{\Lambda}_j < 0. \quad (4.10)$$

**Proof.** Let  $(S_0, I_0, R_0) \in \Delta$ . We define the following Lyapunov function

$$V(S, I, R, j) = \vartheta_1(1-S)^2 + \left( \frac{1}{\varphi} + b_j \right) I^\varphi + \vartheta_2 R^2, \quad \forall j \in \mathcal{E},$$

where  $\vartheta_1 > 0$ ,  $\varphi > 0$ ,  $\vartheta_2 > 0$  and  $b_j$  are real values that will be identified in the subsequent steps. From Itô's formula, we have

$$\begin{aligned} \mathcal{L}V = & -2\vartheta_1 \mu_j (1-S)^2 + 2\vartheta_1 \beta_j S^{\rho_j} I^{\zeta_j} (1-S) - 2\vartheta_1 \eta_j R (1-S) + \vartheta_1 \sigma_j^2 S^{2\rho_j} I^{2\zeta_j} \\ & - \varphi \left( \frac{1}{\varphi} + b_j \right) (\mu_j + \delta_j) I^\varphi + \varphi \left( \frac{1}{\varphi} + b_j \right) \beta_j S^{\rho_j} I^{\varphi + (\zeta_j - 1)} \\ & + \frac{1}{2} \varphi (\varphi - 1) \left( \frac{1}{\varphi} + b_j \right) \sigma_j^2 S^{2\rho_j} I^{\varphi + 2(\zeta_j - 1)} - 2\vartheta_2 (\mu_j + \eta_j) R^2 + 2\vartheta_2 \delta_j I R \\ & + I^\varphi \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk} (b_k - b_j). \end{aligned} \quad (4.11)$$

(i) Consider the case where  $\zeta_j < 1$ . Since  $S, I \in (0, 1)$ , and  $I \leq 1 - S$ , then if  $\varphi < \min_{j \in \mathcal{E}} \{\zeta_j\}$ , (4.11) yields to

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)RI + 2\vartheta_1\beta_jI^\varphi \\ &\quad + \vartheta_1\sigma_j^2I^\varphi - (1+b_j\varphi)(\mu_j + \delta_j)I^\varphi + (1+b_j\varphi)\beta_jS^{\rho_j}I^{\varphi+(\zeta_j-1)} \\ &\quad - \frac{1}{2}(1-\varphi)(1+b_j\varphi)\sigma_j^2S^{2\rho_j}I^{\varphi+2(\zeta_j-1)} + I^\varphi \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j). \end{aligned} \quad (4.12)$$

For  $\varphi < \min \left\{ \min_{j \in \mathcal{E}} \{\zeta_j\}, \frac{1}{\max_{j \in \mathcal{E}} \{|b_j|\}} \right\}$ , the inequality (4.12) becomes

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)RI + (1+b_j\varphi)I^\varphi \\ &\quad \times \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} - \frac{1}{2}(1\zeta_j)\sigma_j^2 \left( S^{\rho_j}I^{(\zeta_j-1)} \right)^2 + \beta_jS^{\rho_j}I^{(\zeta_j-1)} - (\mu_j + \delta_j) \right. \\ &\quad \left. + \frac{1}{1+b_j\varphi} \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right]. \end{aligned} \quad (4.13)$$

Since  $\zeta_j < 1$ , the quadratic function  $-(\mu_j + \delta_j) + \beta_jx - \frac{1}{2}(1 - \zeta_j)\sigma_j^2x^2$  reaches its maximum value at  $x = \frac{\beta_j}{(1 - \zeta_j)\sigma_j^2}$ . Consequently, (4.13) implies

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)RI + (1+b_j\varphi)I^\varphi \\ &\quad \times \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{\beta_j^2}{2(1 - \zeta_j)\sigma_j^2} - (\mu_j + \delta_j) \right. \\ &\quad \left. + \frac{1}{1+b_j\varphi} \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right]. \end{aligned} \quad (4.14)$$

In accordance with the definition of  $\tilde{\Lambda}_j$  in (4.7), from (4.14), we have

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)RI + (1+b_j\varphi)I^\varphi \\ &\quad \times \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \tilde{\Lambda}_j - \frac{b_j\varphi}{1+b_j\varphi} \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right. \\ &\quad \left. + \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right]. \end{aligned} \quad (4.15)$$

Since the generator matrix  $\Gamma$  is irreducible, there exists a vector  $B = (b_1, \dots, b_r)^\top$  that satisfies the Poisson system associated with  $\tilde{\Lambda} = (\tilde{\Lambda}_1, \dots, \tilde{\Lambda}_r)^\top$  (see [176] for more details).

$$\Gamma B = -\tilde{\Lambda} + \left( \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right) e, \quad (4.16)$$

where  $\mathbf{e}$  denotes the column vector with all entries equal to 1. By substituting equation (4.16) into inequality (4.15), we obtain

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR + (1+b_j\varphi)I^\varphi \\ &\quad \times \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{b_j\varphi}{1+b_j\varphi} \left( \tilde{\Lambda}_j - \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right) + \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right]. \end{aligned}$$

By (4.9), we can choose a sufficiently small value for  $\varphi_0$  such that

$$\max_{j \in \mathcal{E}} \left\{ \frac{b_j\varphi_0}{1+b_j\varphi_0} \left( \tilde{\Lambda}_j - \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right) + \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right\} < 0.$$

Next, we choose  $\varphi$ ,  $\vartheta_1$ , and  $\vartheta_2$  satisfying the following conditions

$$\varphi < \min \left\{ \frac{1}{\max_{j \in \mathcal{E}} \{|b_j|\}}, \min_{j \in \mathcal{E}} \{\zeta_j\}, \varphi_0 \right\},$$

$$0 < \vartheta_1$$

$$< -\frac{1 + \varphi \min_{j \in \mathcal{E}} \{b_j\}}{2 \max_{j \in \mathcal{E}} \{\beta_j\} + \max_{j \in \mathcal{E}} \{\sigma_j^2\}} \times \max_{j \in \mathcal{E}} \left\{ \frac{b_j\varphi}{1+b_j\varphi} \left( \tilde{\Lambda}_j - \sum_{k=1}^r \pi_k \tilde{\Lambda}_k \right) + \sum_{k=1}^r \pi_k \tilde{\Lambda}_k \right\},$$

and

$$\vartheta_2 < \min_{j \in \mathcal{E}} \left\{ \frac{\vartheta_1 \eta_j}{\delta_j} \right\}.$$

Hence,

$$\mathcal{L}V \leq -(A_j(1-S)^2 + C_j I^\varphi + B_j R^2), \quad (4.17)$$

where

$$A_j = 2\vartheta_1\mu_j > 0, \quad B_j = 2\vartheta_2(\mu_j + \eta_j) > 0,$$

and

$$C_j = -(1+b_j\varphi) \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{b_j\varphi}{1+b_j\varphi} \left( \tilde{\Lambda}_j - \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right) + \sum_{j=1}^r \pi_j \tilde{\Lambda}_j \right] > 0.$$

(ii) Now, let us consider the case where  $\zeta_j \geq 1$ . For all  $\varphi \leq 1$ , given that  $S, I \in (0, 1)$  and  $I \leq 1 - S$ , the expression in (4.11) can be rewritten as follows

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 + 2\vartheta_1\beta_j I^\varphi + \vartheta_1\sigma_j^2 I^\varphi - (1+b_j\varphi)(\mu_j + \delta_j)I^\varphi \\ &\quad + (1+b_j\varphi)\beta_j S^{\rho_j} I^{\varphi+(\zeta_j-1)} + \frac{1}{2}\varphi\sigma_j^2 - \frac{1}{2}(1+b_j\varphi)\sigma_j^2 S^{2\rho_j} I^{\varphi+2(\zeta_j-1)} \\ &\quad - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR + I^\varphi \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j). \end{aligned}$$

Rearranging, we obtain

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR + (1+b_j\varphi)I^\varphi \\ &\quad \times \left( \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{1}{2}\varphi\sigma_j^2 + \Theta_j(S^{\rho_j}I^{(\zeta_j-1)}) \right. \\ &\quad \left. + \frac{1}{1+b_j\varphi} \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right). \end{aligned} \quad (4.18)$$

By Lemma 4.2 and the fact that the quadratic function  $\Theta_j$  increases in the interval  $\left[0, \frac{\beta_j}{\sigma_j^2}\right]$  and reaches its maximum at  $\frac{\beta_j}{\sigma_j^2}$ , the inequality (4.18) leads to the following two cases.

- If  $\frac{\beta_j}{\sigma_j^2} \leq \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ ,

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR \\ &\quad + (1+b_j\varphi)I^\varphi \times \left( \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{1}{2}\varphi\sigma_j^2 + \Theta_j\left(\frac{\beta_j}{\sigma_j^2}\right) \right. \\ &\quad \left. + \frac{\sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j)}{1+b_j\varphi} \right). \end{aligned} \quad (4.19)$$

- If  $\frac{\beta_j}{\sigma_j^2} > \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ ,

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR \\ &\quad + (1+b_j\varphi)I^\varphi \times \left\{ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{1}{2}\varphi\sigma_j^2 \right. \\ &\quad \left. + \Theta_j\left(\frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}\right) + \frac{\sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j)}{1+b_j\varphi} \right\}. \end{aligned} \quad (4.20)$$

By (4.19), (4.20) and the definition of  $\bar{\Lambda}_j$ , as given in (4.8), one easily have

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)IR + (1+b_j\varphi)I^\varphi \\ &\quad \times \left( \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{1}{2}\varphi\sigma_j^2 + \bar{\Lambda}_j - \frac{b_j\varphi}{1+b_j\varphi} \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right. \\ &\quad \left. + \sum_{k \in \mathcal{E}, k \neq j} \gamma_{jk}(b_k - b_j) \right). \end{aligned}$$

Using a reasoning similar to the first case (i), and slightly adjusting the choice of  $\varphi$  and  $\vartheta_1$ , it follows that there exist positive real constants  $A_j$ ,  $D_j$ , and  $B_j$ . By combining equations

(4.10) and (4.16), we can deduce

$$\begin{aligned} \mathcal{L}V &\leq -2\vartheta_1\mu_j(1-S)^2 - 2\vartheta_2(\mu_j + \eta_j)R^2 + 2(\vartheta_2\delta_j - \vartheta_1\eta_j)RI + (1+b_j\varphi)I^\varphi \\ &\quad \times \left[ \frac{\vartheta_1(2\beta_j + \sigma_j^2)}{1+b_j\varphi} + \frac{1}{2}\varphi\sigma_j^2 + \frac{b_j\varphi}{1+b_j\varphi} \left( \bar{\Lambda}_j - \sum_{j=1}^r \pi_j \bar{\Lambda}_j \right) + \sum_{j=1}^r \pi_j \bar{\Lambda}_j \right] \\ &\triangleq -(A_j(1-S)^2 + D_j I^\varphi + B_j R^2). \end{aligned} \quad (4.21)$$

Applying Lemma 4.3 and using inequalities (4.17) and (4.21), we conclude the proof of the theorem. ■

### 4.3.2 Almost Sure Exponential Extinction

**Theorem 4.5.** Let  $(S_0, I_0, R_0, \xi_0) \in \Delta \times \mathcal{E}$ . Then, the disease-free equilibrium state  $E_0$  of system (4.3) is almost surely exponentially extinct if one of the following conditions holds:

i) If  $\zeta_j \geq 1$ ,  $\frac{\beta_j}{2\sigma_j^2} > \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{2(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ ,  $j \in \mathcal{E}$ , and

$$\sum_{j=1}^r \pi_j \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{2(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}} \right) < 0, \quad (4.22)$$

where

$$\tilde{\Psi}_j(x) = -\mu_j + 2\beta_j x - 2\sigma_j^2 x^2, \quad j \in \mathcal{E}, x \in \mathbb{R}.$$

ii) If  $\zeta_j \geq 1$ ,  $\frac{\beta_j}{2\sigma_j^2} \leq \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{2(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ ,  $j \in \mathcal{E}$ , and

$$\sum_{j=1}^r \pi_j \left( \frac{\beta_j^2 - 2\mu_j\sigma_j^2}{2\sigma_j^2} \right) < 0. \quad (4.23)$$

**Proof.** For the case where  $\zeta_j \geq 1$ , define the function  $U(S, I, R, j) = \ln(1 - S + I + R)$ . By applying Itô's lemma, we obtain

$$\begin{aligned} dU(S, I, R, j) &= \frac{1}{1 - S + I + R} (-\mu_\xi(1 - S) + 2\beta_\xi S^{\rho_\xi} I^{\zeta_\xi} - \mu_\xi I - (\mu_\xi + 2\eta_\xi)R) dt \\ &\quad - 2\sigma_\xi^2 \left( \frac{S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} \right)^2 dt + \frac{2\sigma_\xi S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} dW. \end{aligned} \quad (4.24)$$

Given that

$$\frac{1}{1 - S + I + R} (-\mu_\xi(1 - S) - \mu_\xi I - (\mu_\xi + 2\eta_\xi)R) \leq -\mu_\xi. \quad (4.25)$$

From (4.24) and (4.25), we deduce that

$$\begin{aligned} dU(S, I, R, j) &\leq \left[ -\mu_\xi + 2\beta_\xi \left( \frac{S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} \right) - 2\sigma_\xi^2 \left( \frac{S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} \right)^2 \right] dt \\ &\quad + \frac{2\sigma_\xi S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} dW \\ &= \tilde{\Psi}_\xi \left( \frac{S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} \right) dt + \frac{2\sigma_\xi S^{\rho_\xi} I^{\zeta_\xi}}{1 - S + I + R} dW. \end{aligned} \quad (4.26)$$

By integrating (4.26), we obtain

$$U(t) \leq U(0) + \int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{S^{\rho_{\xi(s)}} I^{\zeta_{\xi(s)}}}{1 - S(s) + I(s) + R(s)} \right) ds + M_t, \quad (4.27)$$

where

$$M_t = \int_0^t \frac{2\sigma_{\xi(s)}(S(s))^{\rho_{\xi(s)}}(I(s))^{\zeta_{\xi(s)}}}{1 - S(s) + I(s) + R(s)} dW(s). \quad (4.28)$$

Since  $S, I, R \in (0, 1)$ ,  $I \leq 1 - S$  and  $1 - S + I + R = 2(1 - S)$ , then by Lemma 4.2, it follows that

$$\begin{aligned} \frac{S^{\rho_{\xi}} I^{\zeta_{\xi}}}{1 - S + I + R} &\leq \frac{1}{2} S^{\rho_{\xi}} (1 - S)^{\zeta_{\xi} - 1} \\ &\leq \frac{\rho_{\xi}^{\rho_{\xi}} (\zeta_{\xi} - 1)^{(\zeta_{\xi} - 1)}}{2(\rho_{\xi} + \zeta_{\xi} - 1)^{\rho_{\xi} + \zeta_{\xi} - 1}}. \end{aligned} \quad (4.29)$$

According to (4.28) and (4.29), we obtain

$$\begin{aligned} [M]_t &= \int_0^t \left( \frac{2\sigma_{\xi(s)}(S(s))^{\rho_{\xi(s)}}(I(s))^{\zeta_{\xi(s)}}}{1 - S(s) + I(s) + R(s)} \right)^2 ds \\ &\leq \left[ \max_{j \in \mathcal{E}} \sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right] t. \end{aligned} \quad (4.30)$$

Thus, applying the law of large numbers for martingales (see [179]), we obtain

$$\limsup_{t \rightarrow \infty} \frac{M_t}{t} = 0 \quad \text{a.s..} \quad (4.31)$$

(i) If  $\frac{\beta_j}{2\sigma_j^2} > \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}}$ , then the function  $\tilde{\Psi}_j$  is increasing on  $[0, \frac{\beta_j}{2\sigma_j^2}]$ . From (4.29), it follows that

$$\tilde{\Psi}_{\xi} \left( \frac{S^{\rho_{\xi}} I^{\zeta_{\xi}}}{1 - S + I + R} \right) \leq \tilde{\Psi}_{\xi} \left( \frac{\rho_{\xi}^{\rho_{\xi}} (\zeta_{\xi} - 1)^{(\zeta_{\xi} - 1)}}{2(\rho_{\xi} + \zeta_{\xi} - 1)^{\rho_{\xi} + \zeta_{\xi} - 1}} \right). \quad (4.32)$$

Therefore, from (4.27) and (4.32), we get

$$U(t) \leq U(0) + \int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{\rho_{\xi(s)}^{\rho_{\xi(s)}} (\zeta_{\xi(s)} - 1)^{(\zeta_{\xi(s)} - 1)}}{2(\rho_{\xi(s)} + \zeta_{\xi(s)} - 1)^{\rho_{\xi(s)} + \zeta_{\xi(s)} - 1}} \right) ds + M_t. \quad (4.33)$$

Applying Birkhoff's Ergodic Theorem yields

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{1}{t} \int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{\rho_{\xi(s)}^{\rho_{\xi(s)}} (\zeta_{\xi(s)} - 1)^{(\zeta_{\xi(s)} - 1)}}{2(\rho_{\xi(s)} + \zeta_{\xi(s)} - 1)^{\rho_{\xi(s)} + \zeta_{\xi(s)} - 1}} \right) ds \\ = \sum_{j=1}^r \pi_j \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right) \quad \text{a.s..} \end{aligned} \quad (4.34)$$

If (4.22) holds, then, by (4.31), (4.33), and (4.34), we obtain

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{1}{t} \ln [1 - S(t) + I(t) + R(t)] &\leq \sum_{j=1}^r \pi_j \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right) \\ &< 0 \quad \text{a.s..} \end{aligned} \quad (4.35)$$

**(ii)** If  $\frac{\beta_j}{2\sigma_j^2} \leq \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}}$ , then from (4.27) and the fact that the function  $\tilde{\Psi}_j$  attains its maximum at  $\frac{\beta_j}{2\sigma_j^2}$ , one can easily verify

$$\begin{aligned} U(t) &\leq U(0) + \int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{\beta_{\xi(s)}}{2\sigma_{\xi(s)}^2} \right) ds \\ &\quad + \int_0^t \frac{2\sigma_{\xi(s)} S^{\rho_{\xi(s)}} I^{\zeta_{\xi(s)}}}{1 - S(s) + I(s) + R(s)} dW(s), \end{aligned} \quad (4.36)$$

where

$$\tilde{\Psi}_{\xi(s)} \left( \frac{\beta_{\xi(s)}}{2\sigma_{\xi(s)}^2} \right) = \frac{\beta_{\xi(s)}^2 - 2\mu_{\xi(s)}\sigma_{\xi(s)}^2}{2\sigma_{\xi(s)}^2}.$$

Similarly to the proof (i), if (4.23) holds, then

$$\begin{aligned} \limsup_{t \rightarrow \infty} \frac{1}{t} \ln [1 - S(t) + I(t) + R(t)] &\leq \sum_{j=1}^r \pi_j \left( \frac{\beta_j^2 - 2\mu_j\sigma_j^2}{2\sigma_j^2} \right) \\ &< 0 \quad \text{a.s..} \end{aligned} \quad (4.37)$$

Using (4.35) and (4.37), we derive

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln [1 - S(t) + I(t) + R(t)] < 0, \quad \text{a.s..}$$

This completes the proof. ■

### 4.3.3 Exponential Moment Stability

We present the following theorem, which establishes sufficient conditions to ensure the exponential stability of the  $q$ -th moment of the disease-free equilibrium state  $E_0$  for the system (4.3).

**Theorem 4.6.** *Let  $q > 0$ . The solution  $(S(t), I(t), R(t), \xi(t))$  of the stochastic differential system (4.3) is said to be exponentially stable in the  $q$ -th moment if one of the following conditions holds:*

**i)** *If  $\zeta_j \geq 1$ ,  $\frac{\beta_j}{2\sigma_j^2} > \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}}$ , for all  $j \in \mathcal{E}$ , and*

$$\sum_{j=1}^r \pi_j \left[ -\mu_j + 2\beta_j \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} + 2(q-1)\sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right] < 0. \quad (4.38)$$

**ii)** If  $\zeta_j \geq 1$ ,  $\frac{\beta_j}{2\sigma_j^2} \leq \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ , for all  $j \in \mathcal{E}$ , and

$$\sum_{j=1}^r \pi_j \left( \frac{\beta_j^2 - 2\mu_j \sigma_j^2}{2\sigma_j^2} + \frac{1}{2} q \sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right) < 0. \quad (4.39)$$

**Proof.**

(i) For the case where  $\zeta_j \geq 1$  and  $\frac{\beta_j}{2\sigma_j^2} > \frac{\rho_j^{\rho_j}(\zeta_j-1)^{\zeta_j-1}}{2(\rho_j+\zeta_j-1)^{\rho_j+\zeta_j-1}}$ , one can easily derive from (4.33)

$$\begin{aligned} \ln [(1 - S(t) + I(t) + R(t))^q] &\leq \ln [(1 - S_0 + I_0 + R_0)^q] + qM_t \\ &\quad + q \int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{\rho_{\xi(s)}^{\rho_{\xi(s)}} (\zeta_{\xi(s)} - 1)^{(\zeta_{\xi(s)} - 1)}}{2(\rho_{\xi(s)} + \zeta_{\xi(s)} - 1)^{\rho_{\xi(s)} + \zeta_{\xi(s)} - 1}} \right) ds. \end{aligned} \quad (4.40)$$

By Birkhoff's Ergodic Theorem, for any  $\varepsilon > 0$  and for sufficiently large values of  $t$ , we have

$$\begin{aligned} &\int_0^t \tilde{\Psi}_{\xi(s)} \left( \frac{\rho_{\xi(s)}^{\rho_{\xi(s)}} (\zeta_{\xi(s)} - 1)^{(\zeta_{\xi(s)} - 1)}}{2(\rho_{\xi(s)} + \zeta_{\xi(s)} - 1)^{\rho_{\xi(s)} + \zeta_{\xi(s)} - 1}} \right) ds \\ &\leq \left[ \sum_{j=1}^r \pi_j \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right) + \frac{\varepsilon}{2q} \right] t. \end{aligned} \quad (4.41)$$

By combining (4.40) and (4.41), we obtain

$$\begin{aligned} \mathbb{E} \left[ \frac{(1 - S(t) + I(t) + R(t))^q}{(1 - S_0 + I_0 + R_0)^q} \right] &\leq e^{\left( q \left[ \sum_{j=1}^r \pi_j \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right) + \frac{\varepsilon}{2q} \right] t \right)} \\ &\quad \times \mathbb{E} [e^{qM_t}]. \end{aligned} \quad (4.42)$$

On one hand,  $qM_t$  is a continuous real-valued martingale with the initial condition  $qM_0 = 0$ . Moreover, according to (4.29) and (4.30), the quadratic variation obeys

$$[qM]_t \leq 4q^2 \int_0^t \sigma_{\xi(s)}^2 \left( \frac{\rho_{\xi(s)}^{\rho_{\xi(s)}} (\zeta_{\xi(s)} - 1)^{(\zeta_{\xi(s)} - 1)}}{2(\rho_{\xi(s)} + \zeta_{\xi(s)} - 1)^{\rho_{\xi(s)} + \zeta_{\xi(s)} - 1}} \right)^2 ds. \quad (4.43)$$

By the ergodic property of Markov chain  $\xi(t)$  and (4.43), it follows that for sufficiently large  $t$ , we have

$$[qM]_t \leq q^2 \left[ \sum_{j=1}^r 4\pi_j \sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 + \frac{\varepsilon}{q^2} \right] t.$$

On the other hand,  $\exp(qM_t - \frac{1}{2}[qM]_t)$  is a martingale. Consequently, for sufficiently large values of  $t$ , we deduce that

$$\begin{aligned} \mathbb{E} [e^{qM_t}] &= \mathbb{E} \left[ e^{\frac{1}{2}[qM]_t} \right] \\ &\leq e^{\left( q^2 \left[ \sum_{j=1}^r 2\pi_j \sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 + \frac{\varepsilon}{2q^2} \right] t \right)}. \end{aligned} \quad (4.44)$$

By combining (4.42) and (4.44), we get

$$\begin{aligned}
 & \mathbb{E} \left[ \frac{(1 - S(t) + I(t) + R(t))^q}{(1 - S_0 + I_0 + R_0)^q} \right] \\
 & \leq e \left( q \sum_{j=1}^r \pi_j \left[ \tilde{\Psi}_j \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right) + 2q\sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right] t + \varepsilon t \right) \\
 & = e \left( q \sum_{j=1}^r \pi_j \left[ -\mu_j + 2\beta_j \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} + 2(q-1)\sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{(\zeta_j - 1)}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right] t + \varepsilon t \right). 
 \end{aligned} \tag{4.45}$$

Thus, if (4.38) holds, then by letting  $t \rightarrow +\infty$  and  $\varepsilon \rightarrow 0$ , the desired assertion is established.

(ii) Now, for the case where  $\zeta_j \geq 1$  and  $\frac{\beta_j}{2\sigma_j^2} \leq \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{2(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}}$ , it follows from (4.36)

$$\begin{aligned}
 \ln [(1 - S(t) + I(t) + R(t))^q] & \leq \ln [(1 - S_0 + I_0 + R_0)^q] + qM_t \\
 & \quad + q \int_0^t \left( \frac{\beta_{\xi(s)}^2 - 2\mu_{\xi(s)}\sigma_{\xi(s)}^2}{2\sigma_{\xi(s)}^2} \right) ds.
 \end{aligned}$$

Similarly to the proof (i) and using (4.39), we get

$$\begin{aligned}
 & \limsup_{t \rightarrow \infty} \frac{1}{t} \ln \mathbb{E} [(1 - S(t) + I(t) + R(t))^q] \\
 & \leq q \sum_{j=1}^r \pi_j \left( \frac{\beta_j^2 - 2\mu_j\sigma_j^2}{2\sigma_j^2} + \frac{q}{2}\sigma_j^2 \left( \frac{\rho_j^{\rho_j} (\zeta_j - 1)^{\zeta_j - 1}}{(\rho_j + \zeta_j - 1)^{\rho_j + \zeta_j - 1}} \right)^2 \right) < 0. 
 \end{aligned} \tag{4.46}$$

This completes the proof. ■

## 4.4 Stochastic Persistence

Stochastic persistence criteria are derived in terms of model parameters. The proof relies on the following lemma.

**Lemma 4.7.** *Let  $X$  be a random variable and let  $\alpha_0 > 0$  and  $C_1 > 0$  be positive constants. Assume that*

$$\mathbb{E}[\exp(\alpha_0 X)] + \mathbb{E}[\exp(-\alpha_0 X)] \leq C_1.$$

*Then, the logarithmic Laplace transform  $\varphi(\alpha) = \ln \mathbb{E}[\exp(\alpha X)]$  is twice differentiable on  $[0, \frac{\alpha_0}{2}]$ , and there exists  $C_2 > 0$  such that*

$$\varphi(\alpha) \leq \alpha \mathbb{E}[X] + \alpha^2 C_2, \quad \text{for } \alpha \in \left[0, \frac{\alpha_0}{2}\right].$$

**Proof.** The proof of this lemma can be found in [180]. ■

Using Lemma 4.7 and the methodologies described in [180, 181], we proceed to demonstrate the stochastic persistence of the disease when  $\Lambda > 0$ .

**Theorem 4.8.** *If  $\Lambda > 0$ , the disease persists stochastically, meaning that for any  $\varepsilon > 0$ , there exists a constant  $v > 0$  such that*

$$\liminf_{t \rightarrow \infty} \mathbf{P}_{X_0, j}(I(t) \geq v) > 1 - \varepsilon, \quad \text{for all } (X_0, j) \in \Delta \times \mathcal{E}.$$

**Proof.** Consider the function  $V_\alpha(S, I, R, j) = I^\alpha$ , where  $\alpha \in (0, 1)$  is a real constant. The infinitesimal generator  $\mathcal{L}V_\alpha$  is given by

$$\begin{aligned} \mathcal{L}V_\alpha &= \alpha I^{\alpha-1} [ -(\mu_j + \delta_j) I + \beta_j S^{\rho_j} I^{\zeta_j} ] + \frac{1}{2} \alpha(\alpha-1) I^{\alpha-2} (\sigma_j S^{\rho_j} I^{\zeta_j})^2 \\ &= \alpha \left[ -(\mu_j + \delta_j) + \beta_j S^{\rho_j} I^{\zeta_j-1} + \frac{\alpha-1}{2} \sigma_j^2 S^{2\rho_j} I^{2(\zeta_j-1)} \right] I^\alpha. \end{aligned} \quad (4.47)$$

We define

$$M_\alpha = \sup_{(S, I, R, j) \in \Delta \times \mathcal{E}} \left\{ \alpha \left( -(\mu_j + \delta_j) + \beta_j S^{\rho_j} I^{\zeta_j-1} + \frac{\alpha-1}{2} \sigma_j^2 S^{2\rho_j} I^{2(\zeta_j-1)} \right) \right\}. \quad (4.48)$$

Combining (4.47) and (4.48), we obtain

$$\mathcal{L}V_\alpha \leq M_\alpha V_\alpha. \quad (4.49)$$

For any  $(X_0, j) = (S_0, I_0, R_0, j) \in \Delta \times \mathcal{E}$ , applying Dynkin's formula in conjunction with (4.49), we get

$$\mathbb{E}^{X_0, j}[I^\alpha(t)] \leq I_0^\alpha + M_\alpha \int_0^t \mathbb{E}^{X_0, j}[I^\alpha(s)] ds.$$

Using Gronwall's inequality, we can establish that

$$\mathbb{E}^{X_0, j}[I^\alpha(t)] \leq I_0^\alpha e^{M_\alpha t}, \quad \text{for } (S_0, I_0, R_0, j) \in \Delta \times \mathcal{E}. \quad (4.50)$$

Analogously, for any  $t \geq pL$  where  $p \in \mathbb{N}$  and  $L > 0$ , we have

$$\mathbb{E}^{X_0, j}[I^\alpha(t)] \leq \mathbb{E}^{X_0, j}[I^\alpha(pL)] e^{M_\alpha(t-pL)}. \quad (4.51)$$

On the other hand, we have

$$\begin{aligned} \ln(I_0) - \ln(I(t)) &= - \int_0^t \Theta_{\xi(s)} ((S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)}-1}) ds \\ &\quad - \int_0^t \sigma_{\xi(s)} (S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)}-1} dW(s). \end{aligned} \quad (4.52)$$

From the ergodicity of the Markov chain  $\xi(s)$ , we derive

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \Theta_{\xi(s)} ((S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)}-1}) ds = \Lambda. \quad (4.53)$$

By applying the dominated convergence theorem and (4.53), one can easily verify

$$\lim_{t \rightarrow \infty} \mathbb{E} \left[ \frac{1}{t} \int_0^t \Theta_{\xi(s)} ((S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)} - 1}) ds \right] = \Lambda,$$

this implies that there exists  $T > 0$  such that

$$\mathbb{E} \left[ \frac{1}{T} \int_0^T \Theta_{\xi(s)} ((S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)} - 1}) ds \right] \geq \frac{\Lambda}{2}. \quad (4.54)$$

By (4.52) and (4.54), we have

$$\begin{aligned} \mathbb{E}^{X_0,j} \left[ \ln \left( \frac{I_0}{I(T)} \right) \right] &= -\mathbb{E}^{X_0,j} \left[ \int_0^T \Theta_{\xi(s)} ((S(s))^{\rho_{\xi(s)}} (I(s))^{\zeta_{\xi(s)} - 1}) ds \right] \\ &\leq -\frac{\Lambda}{2} T. \end{aligned} \quad (4.55)$$

From (4.50), we obtain

$$\begin{aligned} \mathbb{E}^{X_0,j} \left[ e^{\ln \left( \frac{I_0}{I(T)} \right)} + e^{\ln \left( \frac{I(T)}{I_0} \right)} \right] &\leq e^{M_{-1}T} + e^{M_1T} \\ &< \infty, \quad \text{for any } T \geq 0. \end{aligned}$$

Applying Lemma 4.7 and (4.55), we deduce

$$\ln \left( \mathbb{E}^{X_0,j} \left[ e^{\alpha \ln \left( \frac{I_0}{I(T)} \right)} \right] \right) \leq -\frac{\Lambda \alpha}{2} T + \widehat{M} \alpha^2, \quad \text{for } \alpha \in [0, 0.5]. \quad (4.56)$$

where  $\widehat{M}$  is a constant depending on  $T$ ,  $M_{-1}$ , and  $M_1$ . For sufficiently small  $\alpha$ , inequality (4.56), for  $I_0 > \varrho$ ,  $j \in \mathcal{E}$  yields

$$\begin{aligned} \mathbb{E}^{X_0,j} \left[ \frac{I_0^\alpha}{I^\alpha(T)} \right] &= \mathbb{E}^{X_0,j} \left[ e^{\alpha \ln \left( \frac{I_0}{I(T)} \right)} \right] \\ &\leq e^{\left( -\frac{\Lambda \alpha}{2} T + \widehat{M} \alpha^2 \right)} \\ &\leq e^{\left( -\frac{\Lambda \alpha}{4} T \right)}. \end{aligned}$$

For a sufficiently small  $\varrho > 0$  such that  $I_0 > \varrho$ , we have

$$\mathbb{E}^{X_0,j} [I^{-\alpha}(T)] \leq \kappa I_0^{-\alpha}, \quad \text{where } \kappa = \exp \left( -\frac{\Lambda \alpha}{4} T \right). \quad (4.57)$$

Inequalities (4.50) and (4.57) imply that

$$\begin{aligned} \mathbb{E}^{X_0,j} [I^{-\alpha}(T)] &\leq \kappa I_0^{-\alpha} + C \\ &= \kappa \mathbb{E}^{X_0,j} [I_0^{-\alpha}] + C, \end{aligned} \quad (4.58)$$

where  $C = \varrho^{-\alpha} \exp(M_{-\alpha}T)$ . By applying the Markov property and (4.58), we conclude that

$$\mathbb{E}^{X_0,j} [I^{-\alpha} \{(p+1)T\}] \leq \kappa \mathbb{E}^{X_0,j} [I^{-\alpha}(pT)] + C, \quad \text{for } p \in \mathbb{N}.$$

By iterating the previous inequality, we derive

$$\mathbb{E}^{X_0,j} [I^{-\alpha}(nT)] \leq \kappa^n I_0^{-\alpha} + \frac{C(1 - \kappa^n)}{1 - \kappa}, \quad \text{for } n \in \mathbb{N}. \quad (4.59)$$

From (4.51) and (4.59), we have

$$\mathbb{E}^{X_0,j} [I^{-\alpha}(t)] \leq \left( \kappa^n I_0^{-\alpha} + \frac{C(1-\kappa^n)}{1-\kappa} \right) e^{M_{-\alpha} T}, \quad \text{for } t \in [nT, (n+1)T]. \quad (4.60)$$

Since  $\kappa < 1$ , taking  $n \rightarrow +\infty$  in (4.60), yields

$$\begin{aligned} \limsup_{t \rightarrow +\infty} \mathbb{E}^{X_0,j} [I^{-\alpha}(t)] &= \frac{C}{1-\kappa} e^{M_{-\alpha} T} \\ &\stackrel{\Delta}{=} \Upsilon. \end{aligned}$$

Applying Chebyshev's inequality, we obtain

$$\begin{aligned} \mathbf{P} (|I(t)| < v) &= \mathbf{P} \left( \frac{1}{|I(t)|^\alpha} > \frac{1}{v^\alpha} \right) \\ &\leq v^\alpha \mathbb{E} [|I^{-\alpha}(t)|], \end{aligned}$$

for  $v = (\frac{\epsilon}{\Upsilon})^{\frac{1}{\alpha}}$  and an arbitrarily small constant  $\epsilon > 0$ , we get

$$\begin{aligned} \limsup_{t \rightarrow \infty} \mathbf{P} (|I(t)| < v) &\leq v^\alpha \Upsilon \\ &= \epsilon. \end{aligned}$$

Thus,  $\liminf_{t \rightarrow \infty} \mathbf{P} (I(t) \geq v) > 1 - \epsilon$ . The proof is completed. ■

## 4.5 Computer Simulations

We analyze the influence of key parameters on the behavior of systems (4.3). Two examples of two-state Markov chains are presented to illustrate our results. The simulations were implemented in Python, using the Spyder IDE, enabling a comprehensive numerical analysis of the phenomena under consideration. According to the higher-order numerical method developed using the Euler-Maruyama method [182], let  $k$  denote the time index and let  $\varsigma$  represent the discrete time step. The discretized system is given by

$$\begin{cases} S_{k+1} = S_k + \left[ \mu_k - \mu_k S_k - \beta_k S_k^{\rho_k} I_k^{\zeta_k} + \eta_k R_k \right] \varsigma - \sigma_k S_k^{\rho_k} I_k^{\zeta_k} \Delta W_k, \\ I_{k+1} = I_k + \left[ -(\mu_k + \delta_k) I_k + \beta_k S_k^{\rho_k} I_k^{\zeta_k} \right] \varsigma + \sigma_k S_k^{\rho_k} I_k^{\zeta_k} \Delta W_k, \\ R_{k+1} = R_k + [-(\mu_k + \eta_k) R_k + \delta_k I_k] \varsigma, \end{cases}$$

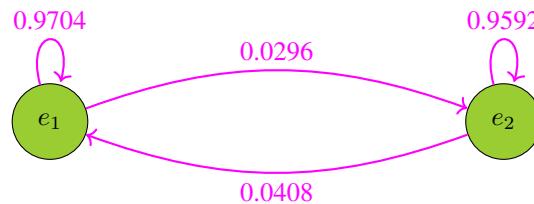
where  $\Delta W_k \sim \mathcal{N}(0, \varsigma)$  represents the increment of the Wiener process.

### 4.5.1 Two-State Markov Chains

Consider a continuous-time Markov chain  $(\xi(t))_{t \geq 0}$  with the state space  $\mathcal{E} = \{e_1, e_2\}$ , the generator matrix  $\Gamma$  and the stationary distribution  $\pi$ . For a small time step  $\varsigma > 0$ , the transition matrix  $A$  can be approximated as  $A = e^{\varsigma \Gamma}$ . Subsequently, we consider the two following Markov chains.

**Example 4.9.** Setting  $\varsigma = 0.01$  and  $\Gamma = \begin{pmatrix} -3 & 3 \\ 4 & -4 \end{pmatrix}$ , yields

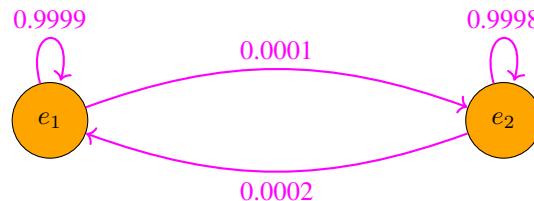
$$A = \begin{pmatrix} 0.9704 & 0.0296 \\ 0.0408 & 0.9592 \end{pmatrix}, \quad \pi = (\pi_1, \pi_2) = \left( \frac{4}{7}, \frac{3}{7} \right) \approx (0.5714, 0.4286).$$



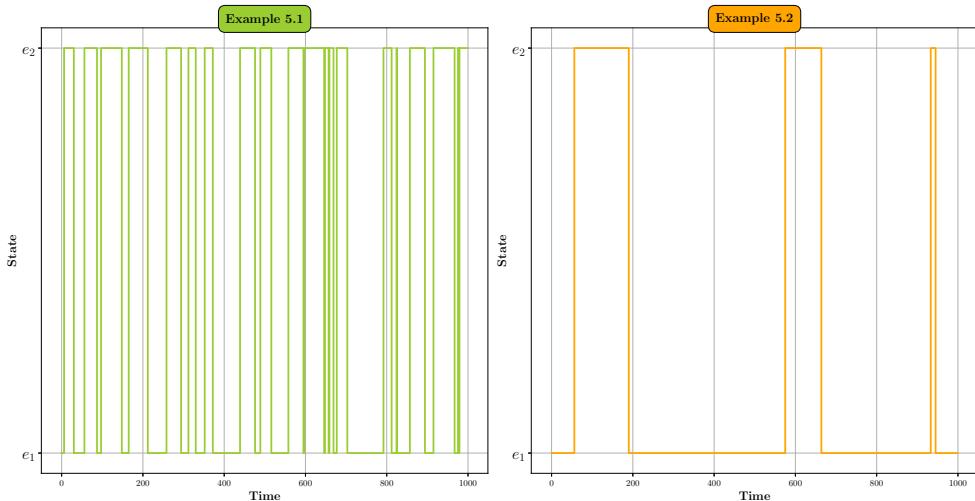
**Figure 4.2.** Two-State Markov Chain Diagram of Example 4.9

**Example 4.10.** Setting  $\varsigma = 0.0001$  and  $\Gamma = \begin{pmatrix} -1 & 1 \\ 2 & -2 \end{pmatrix}$ , yields

$$A = \begin{pmatrix} 0.9999 & 0.0001 \\ 0.0002 & 0.9998 \end{pmatrix}, \quad \pi = (\pi_1, \pi_2) = (0.6667, 0.3333).$$



**Figure 4.3.** Two-State Markov Chain Diagram of Example 4.10



**Figure 4.4.** Transition dynamics of Markov chains.

## 4.5.2 Extinction ( $\Lambda < 0$ )

#### 4.5.2.1 Case: $\tilde{\Lambda}_1 < 0, \tilde{\Lambda}_2 < 0, \bar{\Lambda}_1 < 0$ , and $\bar{\Lambda}_2 < 0$

(a) Let

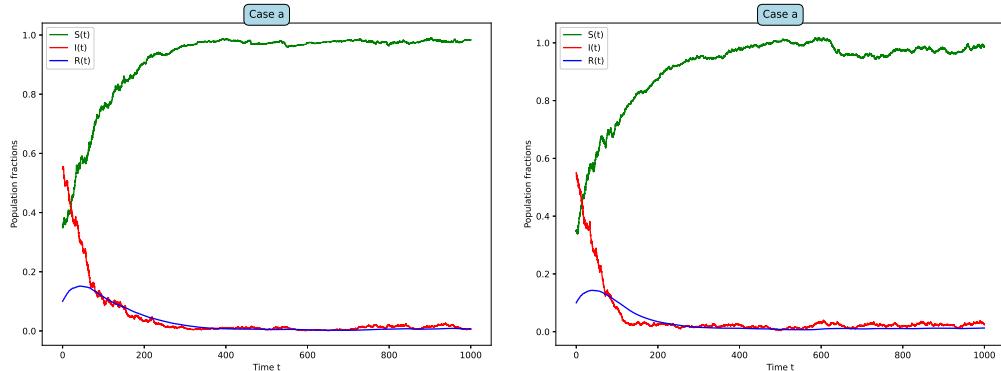
$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (0.5, 0.3), \quad (\rho_1, \rho_2) = (0.5, 0.5), \\ (\mu_1, \mu_2) = (0.999, 0.887), \quad (\beta_1, \beta_2) = (0.3, 0.4), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.9899, 0.988), \quad (\sigma_1, \sigma_2) = (0.9999, 0.8899).$$

Thus,

$$\tilde{\Lambda}_1 \approx -1.8989 < 0, \quad \tilde{\Lambda}_2 \approx -1.875 < 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \tilde{\Lambda}_j \approx \begin{cases} -1.85681 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -1.84213 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.5.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (a)

(b) Let

$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.1), \quad (\rho_1, \rho_2) = (0.5, 0.7), \\ (\mu_1, \mu_2) = (0.9999, 0.61), \quad (\beta_1, \beta_2) = (0.1, 0.3), \quad (\eta_1, \eta_2) = (0.5, 0.3), \\ (\delta_1, \delta_2) = (0.5, 0.3), \quad (\sigma_1, \sigma_2) = (0.8, 0.9).$$

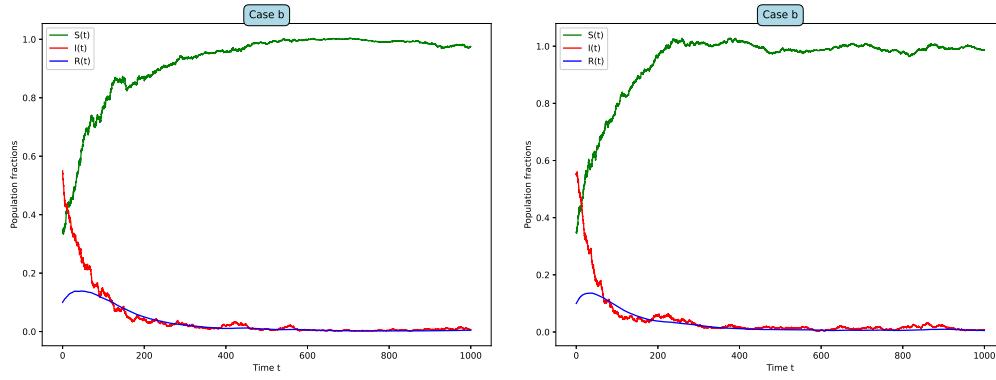
Thus,

$$\bar{\Lambda}_1 \approx -1.4921 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1} (\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx -0.3438 < 0, \\ \bar{\Lambda}_2 \approx -0.8544 < 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2} (\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx -0.3568 < 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} -1.3327 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -1.2796 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$

#### 4.5. COMPUTER SIMULATIONS



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.6.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (b)

(c) Let

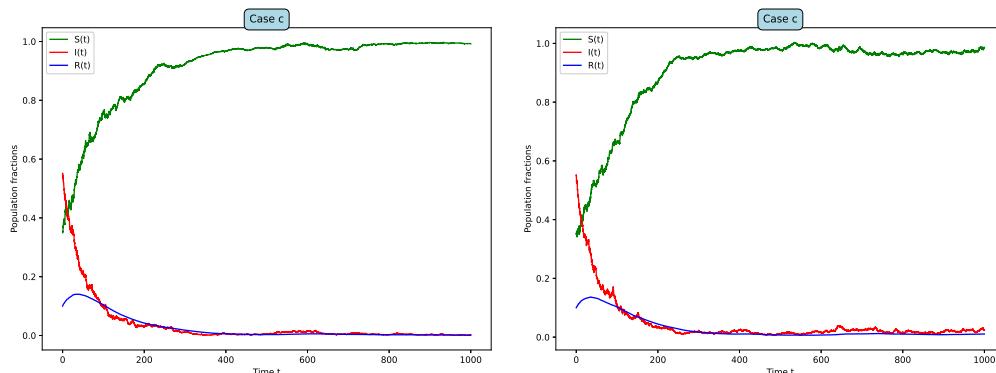
$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (1.2, 1.5), \quad (\rho_1, \rho_2) = (0.6, 0.8), \\ (\mu_1, \mu_2) = (2, 2.5), \quad (\beta_1, \beta_2) = (2.75, 2.5), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (3, 3), \quad (\sigma_1, \sigma_2) = (0.003, 0.001).$$

Thus,

$$\bar{\Lambda}_1 \approx -3.6313 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1}(\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx 305554.92 > 0, \\ \bar{\Lambda}_2 \approx -4.3057 < 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2}(\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx 2499999.58 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} -3.7999 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -3.8561 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases} \quad (4.61)$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.7.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (c)

**4.5.2.2 Case:  $\tilde{\Lambda}_1 < 0, \tilde{\Lambda}_2 > 0, \bar{\Lambda}_1 < 0$ , and  $\bar{\Lambda}_2 > 0$**

**(d)** Let

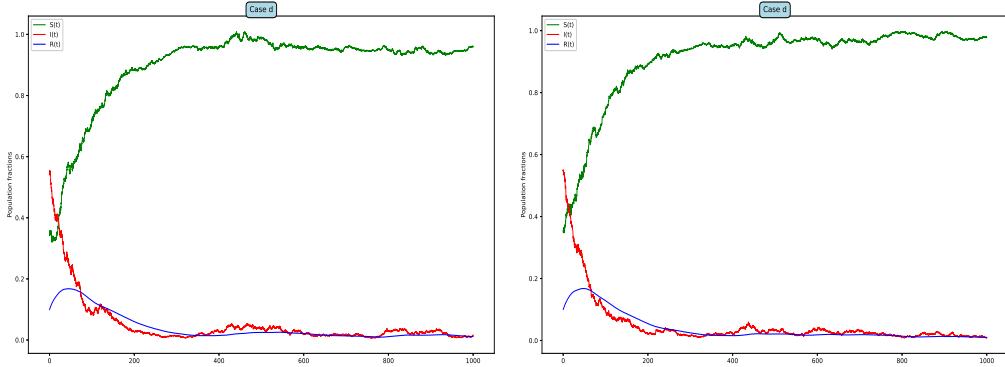
$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (0.5, 0.3), \quad (\rho_1, \rho_2) = (0.5, 0.5), \\ (\mu_1, \mu_2) = (0.9998, 0.05), \quad (\beta_1, \beta_2) = (0.45, 0.4), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.9989, 0.006), \quad (\sigma_1, \sigma_2) = (0.99999, 0.99999).$$

Thus,

$$\tilde{\Lambda}_1 \approx -1.7961 < 0, \quad \tilde{\Lambda}_2 \approx 0.0583 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \tilde{\Lambda}_j \approx \begin{cases} -1.33257 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -1.178096 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



**Figure 4.8.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (d)

**(e)** Let

$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.1), \quad (\rho_1, \rho_2) = (0.5, 0.7), \\ (\mu_1, \mu_2) = (0.9999, 0.0001), \quad (\beta_1, \beta_2) = (0.1, 0.3), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.99999, 0.0003), \quad (\sigma_1, \sigma_2) = (0.8, 0.9).$$

Thus,

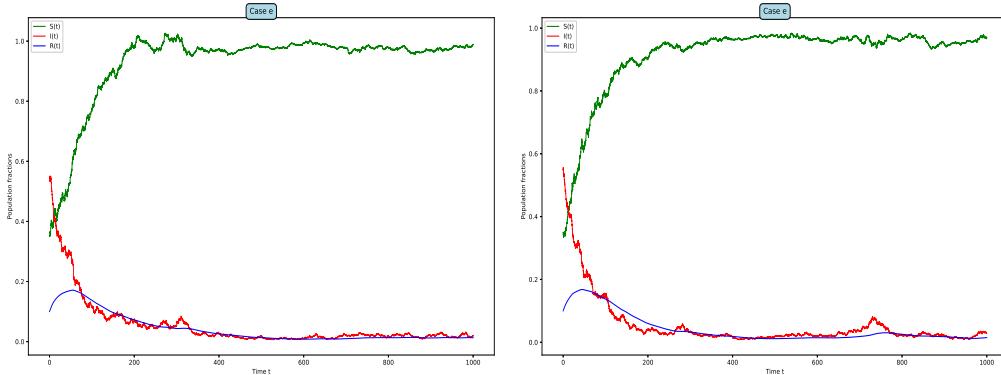
$$\bar{\Lambda}_1 \approx -1.9920 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1} (\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx -0.09375 < 0, \\ \bar{\Lambda}_2 \approx 0.0552 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2} (\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx -0.36863 < 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} -1.4803 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -1.3097 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$

## 4.5. COMPUTER SIMULATIONS

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With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.9.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (e)

(f) Let

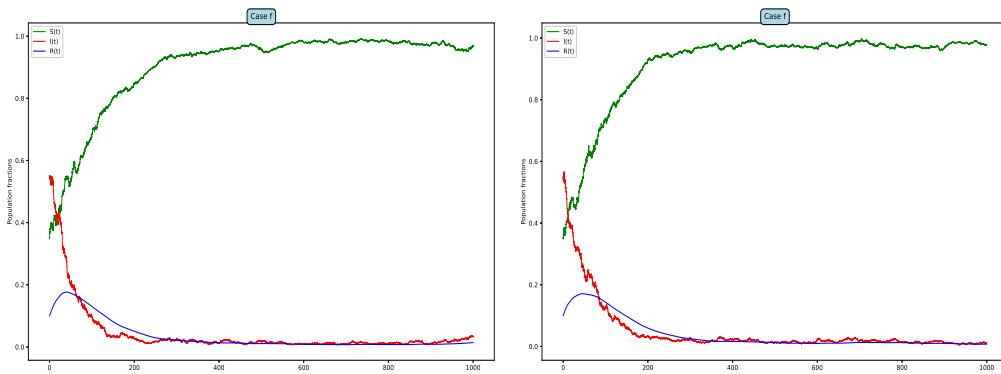
$$(S_0, I_0, R_0) = (0.35, 0.55, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.2), \quad (\rho_1, \rho_2) = (0.8, 0.6), \\ (\mu_1, \mu_2) = (0.9999, 0.23), \quad (\beta_1, \beta_2) = (0.9999, 0.9), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.99998, 0.34), \quad (\sigma_1, \sigma_2) = (0.001, 0.003).$$

Thus,

$$\bar{\Lambda}_1 \approx -1.5794 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1}(\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx 999899.57 > 0, \\ \bar{\Lambda}_2 \approx 0.0039 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2}(\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx 99999.36 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} -0.3919 < 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ -0.5238 < 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.10.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (f)

### 4.5.3 Persistence ( $\Lambda > 0$ )

#### 4.5.3.1 Case: $\tilde{\Lambda}_1 > 0, \tilde{\Lambda}_2 > 0, \bar{\Lambda}_1 > 0$ , and $\bar{\Lambda}_2 > 0$

(a) Let

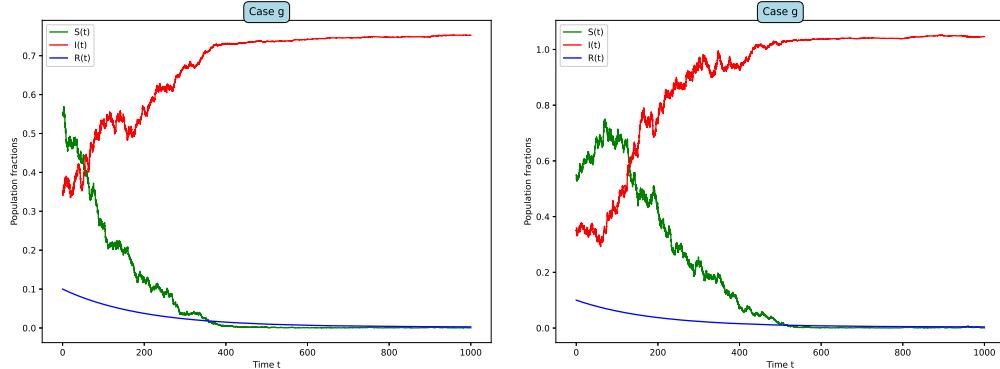
$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (0.25, 0.15), \quad (\rho_1, \rho_2) = (0.5, 0.5), \\ (\mu_1, \mu_2) = (0.001, 0.005), \quad (\beta_1, \beta_2) = (0.88, 0.99), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.001, 0.006), \quad (\sigma_1, \sigma_2) = (0.26, 0.67).$$

Thus,

$$\tilde{\Lambda}_1 \approx 0.2423 > 0, \quad \tilde{\Lambda}_2 \approx 0.1827 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \tilde{\Lambda}_j \approx \begin{cases} 0.2274 > 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ 0.2224 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.11.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (g)

(b) Let

$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.1), \quad (\rho_1, \rho_2) = (0.5, 0.7), \\ (\mu_1, \mu_2) = (0.001, 0.006), \quad (\beta_1, \beta_2) = (0.28, 0.35), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.002, 0.003), \quad (\sigma_1, \sigma_2) = (0.75, 0.7).$$

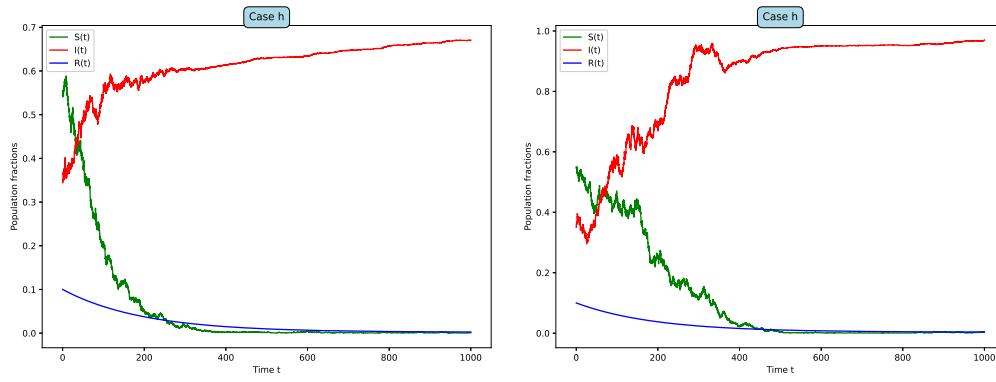
Thus,

$$\bar{\Lambda}_1 \approx 0.0666 > 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1} (\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx -0.0022 < 0, \\ \bar{\Lambda}_2 \approx 0.1159 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2} (\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx -0.0254 < 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} 0.079 > 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ 0.0831 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$

## 4.5. COMPUTER SIMULATIONS



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.12.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (h)

(c) Let

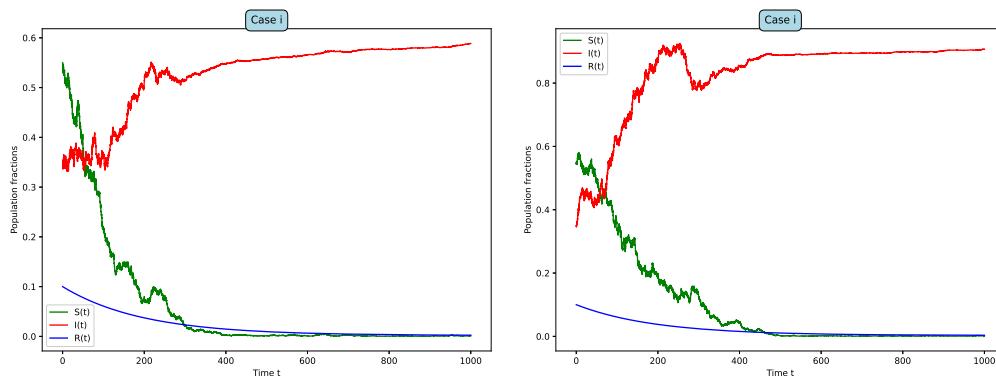
$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (1.2, 1.5), \quad (\rho_1, \rho_2) = (0.6, 0.8), \\ (\mu_1, \mu_2) = (0.002, 0.003), \quad (\beta_1, \beta_2) = (0.999, 0.999), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.001, 0.002), \quad (\sigma_1, \sigma_2) = (0.003, 0.001).$$

Thus,

$$\bar{\Lambda}_1 \approx 0.6341 > 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1}(\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx 110999.36 > 0, \\ \bar{\Lambda}_2 \approx 0.0679 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2}(\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx 9989.57 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} 0.4925 > 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ 0.4454 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.13.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (i)

**4.5.3.2 Case:  $\tilde{\Lambda}_1 < 0, \tilde{\Lambda}_2 > 0, \bar{\Lambda}_1 < 0$ , and  $\bar{\Lambda}_2 > 0$**

(d) Let

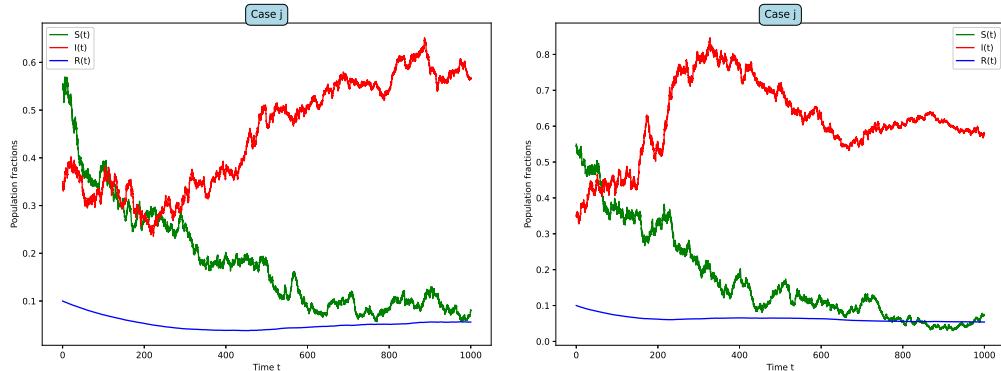
$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (0.5, 0.3), \quad (\rho_1, \rho_2) = (0.5, 0.5), \\ (\mu_1, \mu_2) = (0.112, 0.05), \quad (\beta_1, \beta_2) = (0.45, 0.69), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.0906, 0.006), \quad (\sigma_1, \sigma_2) = (0.99999, 0.999).$$

Thus,

$$\tilde{\Lambda}_1 \approx -0.0001 < 0, \quad \tilde{\Lambda}_2 \approx 0.2847 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} 0.07112 > 0, & \text{if } \pi = (\frac{4}{7}, \frac{3}{7}) \approx (0.5714, 0.4286), \\ 0.09484 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.14.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (j)

(e) Let

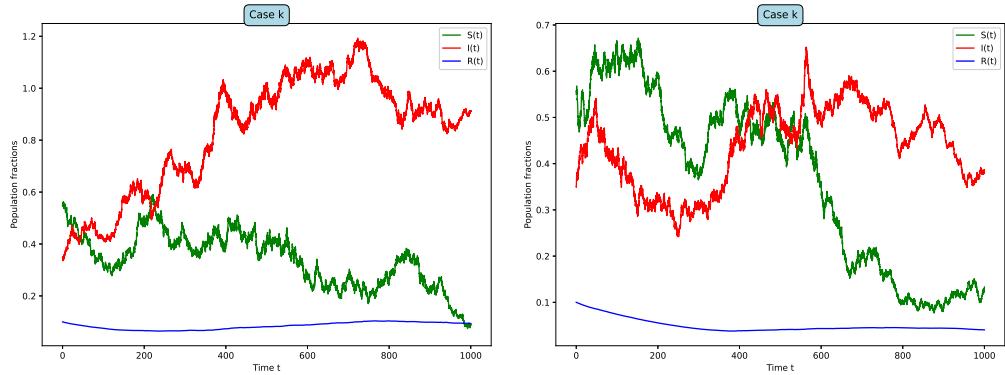
$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.1), \quad (\rho_1, \rho_2) = (0.5, 0.2), \\ (\mu_1, \mu_2) = (0.021, 0.061), \quad (\beta_1, \beta_2) = (0.075, 0.402), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.01234, 0.0061), \quad (\sigma_1, \sigma_2) = (0.8, 0.7).$$

Thus,

$$\bar{\Lambda}_1 \approx -0.0289 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1}(\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx -0.3828 < 0, \\ \bar{\Lambda}_2 \approx 0.0978 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2}(\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx -0.0057 < 0.$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} 0.0027 > 0, & \text{if } \pi = (\frac{4}{7}, \frac{3}{7}) \approx (0.5714, 0.4286), \\ 0.0132 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.15.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (k)

**(f)** Let

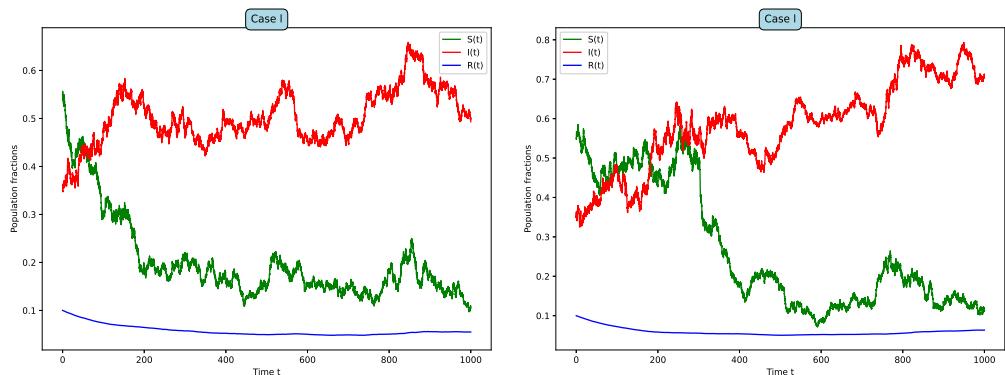
$$(S_0, I_0, R_0) = (0.55, 0.35, 0.1), \quad (\zeta_1, \zeta_2) = (1.5, 1.2), \quad (\rho_1, \rho_2) = (0.8, 0.6), \\ (\mu_1, \mu_2) = (0.19, 0.002), \quad (\beta_1, \beta_2) = (0.999, 0.999), \quad (\eta_1, \eta_2) = (0.5, 0.5), \\ (\delta_1, \delta_2) = (0.29998, 0.001), \quad (\sigma_1, \sigma_2) = (0.001, 0.003).$$

Thus,

$$\bar{\Lambda}_1 \approx -0.0698 < 0, \quad \frac{\beta_1}{\sigma_1^2} - \frac{\rho_1^{\rho_1}(\zeta_1 - 1)^{\zeta_1 - 1}}{(\rho_1 + \zeta_1 - 1)^{\rho_1 + \zeta_1 - 1}} \approx 998999.57 > 0, \\ \bar{\Lambda}_2 \approx 0.6341 > 0, \quad \frac{\beta_2}{\sigma_2^2} - \frac{\rho_2^{\rho_2}(\zeta_2 - 1)^{\zeta_2 - 1}}{(\rho_2 + \zeta_2 - 1)^{\rho_2 + \zeta_2 - 1}} \approx 110999.36 > 0,$$

and,

$$\Lambda = \sum_{j=1}^2 \pi_j \bar{\Lambda}_j \approx \begin{cases} 0.4581 > 0, & \text{if } \pi = \left(\frac{4}{7}, \frac{3}{7}\right) \approx (0.5714, 0.4286), \\ 0.3995 > 0, & \text{if } \pi = (0.6667, 0.3333). \end{cases}$$



With the Markov chain from Example 4.9

With the Markov chain from Example 4.10

**Figure 4.16.** Evolution of  $S_t$ ,  $I_t$ , and  $R_t$  in Case (l)

## 4.6 Conclusion

This study provides a comprehensive analysis of a stochastic SIRS epidemic model with hybrid dynamics, governed by a finite-state Markov chain and incorporating nonlinear transmission rates. The introduction of switched exponents in the bilinear incidence term  $S^{\rho_{\xi(t)}} I^{\zeta_{\xi(t)}}$  allows a more realistic representation of the dynamics of the epidemic under variable environmental conditions. A key contribution of this work is the derivation of necessary and sufficient conditions for the extinction and persistence of diseases, addressing a significant gap in the existing literature. The threshold parameter  $\Lambda$ , which depends on the switching exponents  $\rho_{\xi(t)}$  and  $\zeta_{\xi(t)}$ , serves as a critical determinant of the long-term behavior of the system. Specifically, if  $\Lambda > 0$ , the disease persists stochastically, while  $\Lambda < 0$  ensures the global asymptotic stability of the disease-free equilibrium in probability, leading to eventual eradication. These results generalize classical thresholds by incorporating the effects of regime switching on the bilinear incidence rate  $S^{\rho_{\xi(t)}} I^{\zeta_{\xi(t)}}$ , thus extending their applicability to systems influenced by environmental fluctuations.

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# General Conclusion

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## 1. Relevance of SIRS Models to Epidemiological Modelling

Mathematical modeling of infectious diseases has a growing role to play in understanding the prediction and control of global health risks. In the SIRS model, in particular, which allows individuals to transition from susceptible to infected to recovered, then become susceptible again, is pivotal because it captures the reality of transient immunity. It has also historically helped to account for the cyclical nature of many epidemics and underpins most contemporary strategies to combat the resurgence of diseases such as influenza or whooping cough. The success of the SIRS model is, indeed, to combine in a parsimonious manner various drivers of the complexity of living systems: demography, the regenerating crop of susceptible individuals, deductibility of social contacts, treatments or vaccines. It offers a logical methodology for estimation of "epidemic levels" (such as the basic reproduction number) as part of an informed process for preparedness and guidance of prevention policies. For example, this tool helps decision makers identify the target vaccination coverage and forecast the transition to endemicity. But the fact of epidemics tells us that their dynamics are never perfectly smooth, predictable, or fully under our control. They are characterized by fluctuations, shocks, unexpected environmental or social perturbations, and population heterogeneity. SIRS model itself has developed to recognize this observation: the introduction of stochastic disturbances like Brownian motion, Lévy jumps or regime switching endows the SIRS model with superior relevance and ability to describe phenomena.

In this thesis, this generalization is based on a very tangible concept. On the other hand, it permits to study latent mechanisms of resurgences or extinctions that are missed by deterministic analysis: such as an insight into the reason why a disease can unexpectedly make a comeback after several months of silence, or why an isolated fluctuation is enough to remove it. On the other hand, the stochastic version of the SIRS model can be used to make the concept of risk more concrete: "What are the odds that the disease will disappear within a year? How great is the danger of a new spike in cases due to mutations or unexpected gatherings?" Finally, the SIRS model and, in particular, its rich versions that are analyzed in this paper come as an interdisciplinary crossroad, where mathematics, data science, biology, sociology, and public health join efforts to benefit both from fundamental research, health decisions, and public education. With the rapid growth of global health challenges, perfection and in-depth understanding of this model fall within the realm of scientific needs.

## 2. Novelty of the Two Approaches Proposed in the Thesis

This thesis is notable for two great scientific advances in epidemic dynamic studies. To begin with, the incorporation of Lévy jumps in the SIRS model (i.e., leaping over the SIRS structural

network), which model sudden, rare events with high impact, such as a super spreading event, a key mutation, or an abrupt shift in environmental conditions, gives us a new analytical framework to analyze epidemics such as ours, which happen in a way that cannot be predicted a priori. In contrast to traditional Gaussian noise, Lévy flights mathematically model extreme events, and their occurrence heavily impacts the course of an epidemic. Second, ad-hoc switching of model parameters inspired by a Markov chain aims to mitigate the "all-else-being-equal" syndrome in static models; a pathogen does not consider a static environment, it experiences seasonal variation (e.g., filtering in swine 1 I see range change laboratories, a change in public health response- policy, managing the infected, public behavior change). This methodological novelty captures the cumulative effect of policy sequences or the susceptibility to fast switches and clarifies a number of empirical cases which have remained obscure so far.

By combining both approaches, the thesis offers a proposal for a new generation of models able to reproduce the variety of real-world endemicity, successive waves, abrupt extinction, etc., as well as their unpredictability, essential in situations of emergency health or under information deficit to enlighten decision makers.

### **3. Theoretical Results Comparison for Each Approach**

The theoretical findings of the thesis mark significant progress in mathematical soundness and in solving real-life problems. In contrast to the SIRS model with Lévy jumps the global existence and uniqueness of solutions have already been proved, such that the system is well posed in the probability space, and we are able to perform a more detailed analysis of long-term behaviors. On top of this groundwork, the thesis suggests bespoke Lyapunov functions for the case of stochasticity, one for the case of quasi-certain extinction and the other for the case of endemic persistence. These conditions are given in terms of thresholds that are explicitly dependent not only on the intrinsic parameters of the disease but also on the space and weight of the jumps. So, for the first time ever, a measure has been established for how a big shock can move the dynamic of the collective. The chapter on Markov-switching is also definitive. Of particular interest is the analysis of the Markov chain describing parameter shifts yielding sufficient conditions for stability, extinction, or resurgence, not as a mean trend but by aggregating the exact form of regime alternations. An interesting observation is that, iii in these small "unfavorable" intervals are already enough to revert the final result of the global dynamics, to the point of the emergence of "stop-and-go" profiles very similar to those of real epidemics. Beyond the existence of thresholds itself, the thesis also addresses convergence rates, transients, and beyond threshold results survivability against parametric changes or lack of system information crucial for the practical application of the theoretical results.

### **4. Summary of Numerical Simulations**

The numerical simulations presented in this thesis are not only examples, but a real numerical laboratory in which the theoretical validity, the robustness, and the practical applicability of the proposed models are tested. For Lévy jump models, numerical results illustrate the fact that, for parameters close to threshold, one or several large shocks can completely change the fate of the epidemic: a system close to extinction can be tipped in an unexpected wave, or, conversely, an outbreak may disappear due to a lucky series of factors. The imitation of random oscillations, premature extinctions, or endemic resurgences agrees well with theoretical expectations. For Markovian switching, timing skeletons appear as harmonic waves: blocks of calm and outbursts, in a way which is well consistent with real epidemic data. Sensitivity to the times spent in the two regimes, as well as to the switching rate, provides pseudo-experimental verifications of

#### **4.6. CONCLUSION**

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our predictions showing, questionably, that averages are not sufficient to understand 1d global dynamics. On the technical side, the derivation of some adaptive numerical algorithms that are sensitive to shocks or undergo switching guaranteed the reliability of the results. Multiple sets of parameters, variance reduction methods, and long simulations were used for a stability analysis linked to quantified, comparable scenarios, useful to decision makers for horizon scanning for epidemics.

### **5. Open Questions Not Addressed**

Reading this thesis opens up many research questions to be considered. Methodologically, it is crucial to have access to the true parameters : how to estimate the actual distribution of Lévy jumps or the transition law of the Markov-switching process from surveillance data which are often heterogeneous or with gaps? The challenge of spatial extension, of mobility heterogeneity, or even interactions between subpopulations has only barely been touched upon, despite the current need to understand how diseases spread in space or clusters emerge in space.

Moreover, the study of the adaptive optimal control problem in a case where the epidemic dynamics (the shock term and the switching term) is driven by both stochastic components has not been considered. Lastly, interindividual transmission questions at network level considering memory or time-delay effects are a straightforward extension along the lines of recent advances in applied stochastic process theory.

### **6. Perspectives for Future Work**

There are many promising and open perspectives: If the combination of Lévy jumps and Markov switching were to be taken to the third stage in structured systems or with multiple interactions, in theory, we would be even closer to capturing world's complexity. Methodological axes, to work on generic extinction/persistence criteria for interdependent networks, filtering methods for real-time calibration like the one offered by Bayesian statistics, or the asymmetrical coupling, delay and memory effects of transmission, hold the promise of rich insights. In applications, these models become broader the classical epidemiology: addition, collective rumors, social movements, economic, or ecological crises have similar dynamics. The application of the hybrid stochastic SIRS model for these domains allows us to interpret the specific mechanisms of contagion, disruption, or resilience within each system. Finally, the increasing role of applied mathematics in public decision based on the issue of outreach and accessibility is: how open simulation platforms are to be developed, how a meaningful dialogue between researchers, practitioners, decision makers, and citizens can be fostered, how shared data and risk culture can be encouraged. Modeling will realize its promise as an operational tool that becomes part of the collective, adaptive response to crisis: the challenge and ambition of this ablest thesis.

# APPENDIX A

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## Python Programs

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### A.1 Introduction

This appendix provides a comprehensive presentation of the Python programs developed to generate the figures and results discussed in this thesis. These programs serve as essential tools for illustrating the key concepts, mathematical models, and simulations explored in the research. Each section corresponds to a specific chapter that details the computational methodologies employed and ensures the reproducibility of the results. By structuring the code in a modular and well-documented manner, this chapter aims to facilitate understanding and future adaptations for similar scientific inquiries.

### A.2 Python Programs: Chapter 2

**Listing A.1.** *Python implementation for visualizing the time evolution of  $x(t)$ ,  $y(t)$ , and  $z(t)$  in the Lorenz system*

```
1 # Importing necessary libraries
2 import numpy as np
3 import matplotlib.pyplot as plt
4 from scipy.integrate import solve_ivp
5
6 # Definition of the Lorenz system parameters
7 sigma = 10.0
8 rho = 30.0
9 beta = 8.0 / 3.0
10
11 # Definition of the Lorenz differential equations
12 def lorenz(t, state):
13     x, y, z = state
14     dxdt = sigma * (y - x)
15     dydt = x * (rho - z) - y
16     dzdt = x * y - beta * z
17     return [dxdt, dydt, dzdt]
18
19 # Initial conditions
20 initial_state = [5.0, 21.0, 10.0]
21
22 # Time interval
23 t_span = (0, 100)
```

```
24 t_eval = np.linspace(0, 100, 10000)
25
26 # Solving the system of differential equations
27 sol = solve_ivp(lorenz, t_span, initial_state, t_eval=t_eval)
28
29 # Extracting the solutions
30 x = sol.y[0]
31 y = sol.y[1]
32 z = sol.y[2]
33 t = sol.t
34
35 # Plotting the time series of x(t), y(t), and z(t)
36 plt.figure(figsize=(10, 6))
37
38 plt.subplot(3, 1, 1)
39 plt.plot(t, x, label=r'$x(t)$', color='r')
40 plt.xlabel('Time $t$')
41 plt.ylabel(r'$x(t)$')
42 plt.title('Time Series of $x(t)$')
43 plt.legend()
44 plt.grid()
45
46 plt.subplot(3, 1, 2)
47 plt.plot(t, y, label=r'$y(t)$', color='g')
48 plt.xlabel('Time $t$')
49 plt.ylabel(r'$y(t)$')
50 plt.title('Time Series of $y(t)$')
51 plt.legend()
52 plt.grid()
53
54 plt.subplot(3, 1, 3)
55 plt.plot(t, z, label=r'$z(t)$', color='b')
56 plt.xlabel('Time $t$')
57 plt.ylabel(r'$z(t)$')
58 plt.title('Time Series of $z(t)$')
59 plt.legend()
60 plt.grid()
61
62 plt.tight_layout()
63
64 # Save the figure as an EPS file
65 plt.savefig('lorenz_time_series.eps', format='eps', dpi=1000)
66 plt.show()
```

**Listing A.2.** Simulation of the Lorenz system and plotting of parametric curves

```
1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.integrate import solve_ivp
4
5 # Definition of the Lorenz system parameters
6 sigma = 10.0
7 rho = 30.0
8 beta = 8.0 / 3.0
9
```

```

10 # Definition of the Lorenz differential equations
11 def lorenz(t, state):
12     x, y, z = state
13     dxdt = sigma * (y - x)
14     dydt = x * (rho - z) - y
15     dzdt = x * y - beta * z
16     return [dxdt, dydt, dzdt]
17
18 # Initial conditions
19 initial_state = [5.0, 21.0, 10.0]
20
21 # Time interval
22 t_span = (0, 100)
23 t_eval = np.linspace(0, 100, 10000)
24
25 # Solving the system of differential equations
26 sol = solve_ivp(lorenz, t_span, initial_state, t_eval=t_eval)
27
28 # Extracting the solutions
29 x, y, z = sol.y
30
31 # Plotting parametric curves
32 plt.figure(figsize=(15, 5))
33
34 plt.subplot(1, 3, 1)
35 plt.plot(x, y, color='b')
36 plt.xlabel('x(t)')
37 plt.ylabel('y(t)')
38 plt.title('Parametric Curve (x(t), y(t))')
39 plt.grid()
40
41 plt.subplot(1, 3, 2)
42 plt.plot(x, z, color='r')
43 plt.xlabel('x(t)')
44 plt.ylabel('z(t)')
45 plt.title('Parametric Curve (x(t), z(t))')
46 plt.grid()
47
48 plt.subplot(1, 3, 3)
49 plt.plot(y, z, color='g')
50 plt.xlabel('y(t)')
51 plt.ylabel('z(t)')
52 plt.title('Parametric Curve (y(t), z(t))')
53 plt.grid()
54
55 plt.tight_layout()
56 plt.savefig('lorenz_parametric_curves.eps', format='eps', dpi=1000)
57 plt.show()

```

**Listing A.3.** Python implementation for visualizing the 3D trajectory of the Lorenz system

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.integrate import solve_ivp
4 from mpl_toolkits.mplot3d import Axes3D # Required for 3D plotting

```

```

5 # Definition of the Lorenz system parameters
6 sigma = 10.0
7 rho = 30.0
8 beta = 8.0 / 3.0
9
10
11 # Definition of the Lorenz differential equations
12 def lorenz(t, state):
13     x, y, z = state
14     dxdt = sigma * (y - x)
15     dydt = x * (rho - z) - y
16     dzdt = x * y - beta * z
17     return [dxdt, dydt, dzdt]
18
19 # Initial conditions
20 initial_state = [5.0, 21.0, 10.0]
21
22 # Time interval
23 t_span = (0, 100)
24 t_eval = np.linspace(0, 100, 10000)
25
26 # Solving the system of differential equations
27 sol = solve_ivp(lorenz, t_span, initial_state, t_eval=t_eval)
28
29 # Extracting the solutions
30 x = sol.y[0]
31 y = sol.y[1]
32 z = sol.y[2]
33
34 # Creating a 3D plot
35 fig = plt.figure(figsize=(10, 8))
36 ax = fig.add_subplot(111, projection='3d') # Create a 3D axis
37
38 # Plotting the 3D parametric curve
39 ax.plot(x, y, z, color='b', lw=0.5)
40 ax.set_xlabel(r'$x(t)$')
41 ax.set_ylabel(r'$y(t)$')
42 ax.set_zlabel(r'$z(t)$')
43 ax.set_title(r'3D Trajectory of the Lorenz System')
44
45 # Save the figure as an EPS file
46 plt.savefig('lorenz_3d_curve.eps', format='eps', dpi=1000)
47
48 # Display the plot
49 plt.show()

```

**Listing A.4.** Comparison of logistic and exponential models in Python

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.optimize import least_squares
4
5 # Model functions
6 def logistic(t, alpha, mu, k):
7     """Logistic model (saturated growth)"""

```

```
8     return (mu * np.exp(k * t)) / (alpha * np.exp(k * t) + mu -
9         ↪ alpha)
10
11 def exponential(t, C, k):
12     """Exponential model (unbounded growth)"""
13     return C * np.exp(k * t)
14
15 # Manually extracted data (t, y) for the logistic curve (blue)
16 logistic_data = np.array([
17     [0, 1.0],
18     [1, 2.4],
19     [2, 6.0],
20     [3, 10.0],
21     [4, 15.0],
22     [5, 18.0],
23     [6, 19.5],
24     [7, 19.8]
25 ])
26
27 # Manually extracted data (t, y) for the exponential curve (red)
28 exponential_data = np.array([
29     [0, 1.0],
30     [0.5, 1.65],
31     [1, 2.7],
32     [1.5, 4.4],
33     [2, 7.2],
34     [2.3, 10.0],
35     [2.6, 14.5]
36 ])
37
38 # Error functions for parameter optimization
39 def logistic_error(params):
40     alpha, mu, k = params
41     y_model = logistic(logistic_data[:, 0], alpha, mu, k)
42     return y_model - logistic_data[:, 1]
43
44 def exponential_error(params):
45     C, k = params
46     y_model = exponential(exponential_data[:, 0], C, k)
47     return y_model - exponential_data[:, 1]
48
49 # Initial parameter estimates
50 init_logistic = [0.2, 1.0, 1.0] # alpha, mu, k
51 init_exponential = [1.0, 1.0] # C, k
52
53 # Parameter optimization
54 logistic_result = least_squares(logistic_error, init_logistic,
55     ↪ bounds=(0, np.inf))
56 exponential_result = least_squares(exponential_error,
57     ↪ init_exponential, bounds=(0, np.inf))
58
59 # Optimized parameters
60 alpha_opt, mu_opt, k_opt_log = logistic_result.x
61 C_opt, k_opt_exp = exponential_result.x
```

## A.2. PYTHON PROGRAMS: CHAPTER 2

---

```
59 # Display results
60 print("Optimized parameters:")
61 print(f"Logistic: alpha = {alpha_opt:.4f}, mu = {mu_opt:.4f}, k = {
62     ↪ k_opt_log:.4f}")
63 print(f"Exponential: C = {C_opt:.4f}, k = {k_opt_exp:.4f}")
64
65 # Create a finite domain for plotting the curves
66 t_fine = np.linspace(0, 7, 500)
67
68 # Calculate optimized curves
69 y_log_opt = logistic(t_fine, alpha_opt, mu_opt, k_opt_log)
70 y_exp_opt = exponential(t_fine, C_opt, k_opt_exp)
71
72 # Plot the curves
73 plt.figure(figsize=(8, 5))
74 plt.plot(t_fine, y_log_opt, color='#0000FF', linewidth=3,
75           label=r'Logistic: $y(t) = \frac{\mu e^{kt}}{\alpha e^{kt}}
76           ↪ + \mu - \alpha}$')
77 plt.plot(t_fine, y_exp_opt, color='#FF0000', linestyle='--',
78           ↪ linewidth=2,
79           label=r'Exponential: $y(t) = Ce^{kt}$')
80
81 plt.xlim(0, 7)
82 plt.ylim(0, 22)
83 plt.xlabel('Time (t)')
84 plt.ylabel('y(t)')
85 plt.grid(True, linestyle=':', color='gray', alpha=0.5)
86 plt.legend(loc='upper left', fontsize=10)
87 plt.tight_layout()
88 plt.show()
```

**Listing A.5.** Python program generating the Predator-Prey model evolution laws

```
1 # Importing necessary libraries
2 import numpy as np
3 import matplotlib.pyplot as plt
4 from scipy.integrate import odeint
5
6 carrying_capacity = np.array([300, 200])
7 alpha = np.array([400, 100])
8 signs = np.array([1, -1])
9
10 def predator_prey_system(y, t, carrying_capacity, signs):
11     dydt = signs * (1 - np.flipud(y / carrying_capacity)) * y
12     return dydt
13
14 initial_conditions = [100, 50]
15 time_span = np.linspace(0, 20, 1000)
16
17 solution = odeint(predator_prey_system, initial_conditions,
18     ↪ time_span, args=(carrying_capacity, signs))
19
20 plt.figure(figsize=(10, 6))
21 plt.plot(time_span, solution[:, 0], 'bo-', label='Prey', linewidth
```

```

1   ↪ =2)
2 plt.plot(time_span, solution[:, 1], 'go-', label='Predators',
2   ↪ linewidth=2)
3
4 plt.xlabel('Time (t)')
5 plt.ylabel('Population')
6 plt.legend()
7 plt.title('Predator-Prey Model Evolution Laws')
8 plt.grid(True)
9
10 plt.show()

```

**Listing A.6.** Python code for modeling diffusion using a random walk

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3
4 # Parameter definition
5 num_steps = 50000 # Number of steps in the random walk
6 x = np.zeros(num_steps + 1) # Adding one point to avoid errors
7 y = np.zeros(num_steps + 1)
8
9 # Generate random steps
10 theta = np.random.uniform(0, 2*np.pi, num_steps) # Random angles
11 x[1:] = np.cumsum(np.cos(theta))
12 y[1:] = np.cumsum(np.sin(theta))
13
14 # Create the figure
15 plt.figure(figsize=(8, 6))
16 plt.plot(x, y, linewidth=0.7, alpha=0.7, color='black') #
16   ↪ Trajectory
17 plt.scatter(0, 0, color='red', label='Start') # Starting point
18 plt.scatter(x[-1], y[-1], color='blue', label='End') # Final point
19 plt.axis('equal')
20 plt.title("Diffusion Modeling with a Random Walk")
21 plt.legend()
22 plt.show()

```

## A.3 Python Programs: Chapter 3

**Listing A.7.** Python program generating the cumulative evolution plot of stochastic data

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3
4 num_curves = 10
5
6 colors = ['#1f77b4', '#ff7f0e', '#2ca02c', '#d62728', '#9467bd', ,
6   ↪ '#8c564b', '#e377c2', '#7f7f7f', '#bcbd22', '#17becf']
7
8 for i in range(num_curves):
9     color = colors[i % len(colors)]
10    plt.plot(np.cumsum(np.random.randn(1000)), color=color,
10      ↪ linewidth=1)

```

### A.3. PYTHON PROGRAMS: CHAPTER 3

```
11 plt.xlabel('Time Steps') # Label for the x-axis
12 plt.ylabel('Cumulative Sum') # Label for the y-axis
13 plt.axis('off')
14 plt.show()
```

**Listing A.8.** Python program generating Girko's circular law using SageMathCell

```
1 import numpy as np
2 import matplotlib.pyplot as plt
3
4 n = 66
5 r = 66
6
7 colors = ['blue', 'green', 'red', 'cyan', 'magenta', 'yellow', '
    ↪ black', 'orange', 'purple', 'brown']
8
9 for i in range(1, r):
10     M = np.sign(np.random.randn(n, n)) / np.sqrt(n)
11     S = np.linalg.eigvals(M)
12     plt.plot(S.real, S.imag, '.', color=colors[i % len(colors)],
    ↪ markersize=3)
13
14 plt.axis('off')
15 plt.title("Circular Law")
16 plt.show()
```

**Listing A.9.** Python code for simulating and analyzing the Ornstein-Uhlenbeck process.

```
1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.stats import norm
4
5 # Parameters for the Ornstein-Uhlenbeck process
6 mu = 1.0      # Mean reversion strength
7 sigma = 1.0    # Volatility
8 X0 = 0.0       # Initial value
9 T = 10         # Total time
10 dt = 0.01      # Time step
11 N = int(T / dt) # Number of time steps
12 t = np.linspace(0, T, N) # Time array
13
14 # Simulate the Ornstein-Uhlenbeck process
15 np.random.seed(42) # For reproducibility
16 dB = np.random.normal(0, np.sqrt(dt), N) # Brownian increments
17 X = np.zeros(N) # Initialize process
18 X[0] = X0
19 for i in range(1, N):
20     X[i] = X[i-1] - mu * X[i-1] * dt + sigma * dB[i]
21
22 # Theoretical mean and variance
23 mean_theoretical = X0 * np.exp(-mu * t) # Theoretical mean
24 var_theoretical = (sigma**2 / (2 * mu)) * (1 - np.exp(-2 * mu * t))
    ↪ # Theoretical variance
25
```

```

26 # Generate additional data for comparison
27 num_simulations = 100 # Number of simulations for ensemble
28     ↪ analysis
29 X_ensemble = np.zeros((num_simulations, N))
30 for j in range(num_simulations):
31     dB_ensemble = np.random.normal(0, np.sqrt(dt), N)
32     X_ensemble[j, 0] = X0
33     for i in range(1, N):
34         X_ensemble[j, i] = X_ensemble[j, i-1] - mu * X_ensemble[j,
35             ↪ i-1] * dt + sigma * dB_ensemble[i]
36
37 # Create the figure with 4 subplots
38 fig, axs = plt.subplots(2, 2, figsize=(14, 10))
39 fig.suptitle('Ornstein-Uhlenbeck Process: Simulation and Analysis',
40             ↪ fontsize=16, y=1.02)
41
42 # Subplot 1: Simulated process
43 axs[0, 0].plot(t, X, label='Simulated Path', color='blue',
44                 ↪ linewidth=1.5)
45 axs[0, 0].set_title('Simulated Ornstein-Uhlenbeck Process')
46 axs[0, 0].set_xlabel('Time (t)')
47 axs[0, 0].set_ylabel('X(t)')
48 axs[0, 0].legend(loc='upper right')
49 axs[0, 0].grid(True, linestyle='--', alpha=0.6)
50
51 # Subplot 2: Theoretical mean vs simulated mean
52 axs[0, 1].plot(t, mean_theoretical, label='Theoretical Mean', color
53                 ↪ ='green', linestyle='--', linewidth=2)
54 axs[0, 1].plot(t, np.mean(X_ensemble, axis=0), label='Simulated
55                 ↪ Mean', color='orange', linestyle='-', linewidth=1.5)
56 axs[0, 1].set_title('Theoretical vs Simulated Mean')
57 axs[0, 1].set_xlabel('Time (t)')
58 axs[0, 1].set_ylabel('Mean')
59 axs[0, 1].legend(loc='upper right')
60 axs[0, 1].grid(True, linestyle='--', alpha=0.6)
61
62 # Subplot 3: Theoretical variance vs simulated variance
63 axs[1, 0].plot(t, var_theoretical, label='Theoretical Variance',
64                 ↪ color='red', linestyle='--', linewidth=2)
65 axs[1, 0].plot(t, np.var(X_ensemble, axis=0), label='Simulated
66                 ↪ Variance', color='purple', linestyle='-', linewidth=1.5)
67 axs[1, 0].set_title('Theoretical vs Simulated Variance')
68 axs[1, 0].set_xlabel('Time (t)')
69 axs[1, 0].set_ylabel('Variance')
70 axs[1, 0].legend(loc='upper right')
71 axs[1, 0].grid(True, linestyle='--', alpha=0.6)
72
73 # Subplot 4: Distribution of X(t) at a specific time (e.g., t = T
74     ↪ /2)
75 t_half = int(N / 2) # Time index at t = T/2
76 x_values = np.linspace(min(X_ensemble[:, t_half]), max(X_ensemble
77     ↪ [:, t_half]), 1000)
78 pdf_theoretical = norm.pdf(x_values, mean_theoretical[t_half], np.
79     ↪ sqrt(var_theoretical[t_half]))

```

```
69 axs[1, 1].hist(X_ensemble[:, t_half], bins=30, density=True, color=
    ↪ 'skyblue', alpha=0.7, label='Simulated Distribution')
70 axs[1, 1].plot(x_values, pdf_theoretical, label='Theoretical
    ↪ Distribution', color='darkblue', linestyle='--', linewidth
    ↪ =2)
71 axs[1, 1].set_title(f'Distribution of X(t) at t = {t[t_half]:.2f}')
72 axs[1, 1].set_xlabel('X(t)')
73 axs[1, 1].set_ylabel('Probability Density')
74 axs[1, 1].legend(loc='upper right')
75 axs[1, 1].grid(True, linestyle='--', alpha=0.6)
76
77 # Adjust layout and spacing
78 plt.tight_layout()
79
80 # Save the figure as an EPS file
81 output_filename = 'ornstein_uhlenbeck_analysis.eps'
82 plt.savefig(output_filename, format='eps', dpi=300, bbox_inches =
    ↪ tight)
83 print(f"Figure saved as {output_filename}")
84
85 # Show the figure
86 plt.show()
```

## A.4 Python Programs: Chapter 4

**Listing A.10.** *Stochastic Simulation of the SIRS Model - Persistence Case*

```
1 import numpy as np
2 import matplotlib.pyplot as plt
3
4 # Define a new command to modify the citation number color
5 def display_number(number):
6     precision = 9
7     format_str = "{:. " + str(precision) + "f}"
8     return format_str.format(number)
9
10 def simulate_and_plot_SIRS(ax, t, rho, alpha, lambda_val, eta,
11     ↪ sigma, initial_S, initial_I, initial_R):
12     """
13         Simulate the SIRS model with stochasticity and plot the results
14         .
15
16     Parameters:
17         ax (matplotlib.axes.Axes): Axes object to plot on.
18         t (numpy.ndarray): Array of time values.
19         rho (float): Rate of transmission.
20         alpha (float): Rate of infection.
21         lambda_val (float): Rate of recovery.
22         eta (float): Rate of reintroduction into the susceptible
23             ↪ population.
24         sigma (float): Volatility parameter.
25         initial_S (float): Initial proportion of susceptible
26             ↪ individuals.
```

```

23     initial_I (float): Initial proportion of infected
24         ↪ individuals.
25     initial_R (float): Initial proportion of recovered
26         ↪ individuals.
27
28     Returns:
29         None
30     """
31
32     # Initialize arrays to store the results
33     susceptible = np.zeros_like(t)
34     infected = np.zeros_like(t)
35     recovered = np.zeros_like(t)
36
37     # Set initial values
38     susceptible[0], infected[0], recovered[0] = initial_S,
39         ↪ initial_I, initial_R
40
41     # Perform Euler-Maruyama integration
42     for i in range(1, len(t)):
43         dW_S = np.random.normal(0, np.sqrt(dt))
44         dW_I = np.random.normal(0, np.sqrt(dt))
45         dSdt = rho * (1 - susceptible[i-1]) + eta * recovered[i-1]
46             ↪ - alpha * susceptible[i-1] * infected[i-1]
47         dIdt = alpha * susceptible[i-1] * infected[i-1] - (rho +
48             ↪ lambda_val) * infected[i-1]
49         dRdt = lambda_val * infected[i-1] - (rho + eta) * recovered
50             ↪ [i-1]
51         susceptible[i] = susceptible[i-1] + dSdt * dt + sigma *
52             ↪ susceptible[i-1] * infected[i-1] * dW_S
53         infected[i] = infected[i-1] + dIdt * dt + sigma *
54             ↪ susceptible[i-1] * infected[i-1] * dW_I
55         recovered[i] = recovered[i-1] + dRdt * dt
56
57     # Plot the results
58     ax.plot(t, susceptible, label='Susceptible', color='blue')
59     ax.plot(t, infected, label='Infected', color='red')
60     ax.plot(t, recovered, label='Recovered', color='green')
61     ax.set_xlabel('Time', color='magenta')
62     ax.set_ylabel('Proportion: S, I and R', color='lime')
63     ax.set_title(f'Stochastic Simulation of SIRS Model: Persistence
64         ↪ case\nInitial Values: (S={case["initial_S"]}, I={case
65             ↪ ["initial_I"]}, R={case["initial_R"]}), transform=ax.
66             ↪ transAxes, fontsize=10, verticalalignment='top',
67             ↪ horizontalalignment='center', color='olive')
68     ax.legend()
69     ax.grid(True)
70
71     # Calculate T1 and T2
72     T1 = alpha / (rho + lambda_val + (1/4) * sigma**2)
73     T2 = alpha / (rho + lambda_val + (1/2) * sigma**2)
74
75     # Add parameter values below the x-axis
76     parameter_offset = -0.25 # Offset in centimeters (1.5 cm $\
77         ↪ approx$ 0.59 inches)

```

```
64     t_values_offset = -0.15 # Offset in centimeters (1.5 cm $\
65         ↪ approx$ 0.59 inches)
66
66     ax.text(0.18, t_values_offset, f'$\\mathcal{{T}}^{1}={
67         ↪ display_number(T1)}$', color='Maroon', transform=ax.
67         ↪ transAxes, fontsize=10, verticalalignment='top')
67     ax.text(0.58, t_values_offset, f'$\\mathcal{{T}}^{2}={
68         ↪ display_number(T2)}$', color='Maroon', transform=ax.
68         ↪ transAxes, fontsize=10, verticalalignment='top')
69
69     ax.text(0.03, parameter_offset, f'$\\rho={display_number(rho)}$'
70         ↪ ', color='teal', transform=ax.transAxes, fontsize=10,
70         ↪ verticalalignment='top')
70     ax.text(0.38, parameter_offset, f'$\\alpha={display_number(
71         ↪ alpha)}$', color='teal', transform=ax.transAxes, fontsize
71         ↪ =10, verticalalignment='top')
71     ax.text(0.72, parameter_offset, f'$\\lambda={display_number(
72         ↪ lambda_val)}$', color='teal', transform=ax.transAxes,
72         ↪ fontsize=10, verticalalignment='top')
73
73     ax.text(0.18, parameter_offset - 0.1, f'$\\eta={display_number(
74         ↪ eta)}$', color='teal', transform=ax.transAxes, fontsize
74         ↪ =10, verticalalignment='top')
74     ax.text(0.58, parameter_offset - 0.1, f'$\\varsigma_{\\upsilon}={
75         ↪ display_number(sigma)}$', color='teal', transform=ax.
75         ↪ transAxes, fontsize=10, verticalalignment='top')
76
76 # Define the parameters for each case
77 cases = [
78     {"rho": 0.000026, "alpha": 0.99999999, "lambda_val": 0.000011,
78         ↪ "eta": 0.05, "sigma": 0.99999999, "initial_S": 0.35, "
78         ↪ initial_I": 0.45, "initial_R": 0.2},
79     {"rho": 0.31, "alpha": 0.99, "lambda_val": 0.39, "eta": 0.05, "
79         ↪ sigma": 0.99999999, "initial_S": 0.35, "initial_I":
79         ↪ 0.45, "initial_R": 0.2},
80     {"rho": 0.99999999, "alpha": 0.28, "lambda_val": 0.99999999, "
80         ↪ eta": 0.05, "sigma": 0.87, "initial_S": 0.35, "initial_I
80         ↪ ": 0.45, "initial_R": 0.2},
81     {"rho": 0.00011, "alpha": 0.96, "lambda_val": 0.00015, "eta": "
81         ↪ 0.05, "sigma": 0.053, "initial_S": 0.35, "initial_I": "
81         ↪ 0.45, "initial_R": 0.2}
82 ]
83 try:
84     # Iterate over each case and plot the SIRS model
85     for i, case in enumerate(cases):
86         print(f"Case {i+1}:")
87         fig, ax = plt.subplots()
88         t_min, t_max = 1000, 1020
89         dt = 0.001 # You may need to adjust the step size for
89             ↪ higher precision
90         num_steps = int((t_max - t_min) / dt)
91         t = np.linspace(t_min, t_max, num_steps)
92         simulate_and_plot_SIRS(ax, t, **case)
93         plt.tight_layout() # Adjust layout for better spacing
```

```

94     # Save the graph in EPS format
95     filename = f"graph_case_{i+1}.eps"
96     plt.savefig(filename, format='eps')
97     print(f"Saved graph to {filename}")
98     plt.show()

99
100 except Exception as e:
101     print("An error occurred during execution:")
102     print(e)

```

**Listing A.11.** Stochastic Simulation of the SIRS Model - Extinction Case

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3
4 # Define a new command to modify the citation number color
5 precision = 9
6
7 def display_number(number):
8     format_str = "{:.{}f}".format(precision)
9     return format_str.format(number)
10
11 def simulate_and_plot_SIRS(ax, t, rho, alpha, lambda_val, eta,
12     ↪ sigma, initial_S, initial_I, initial_R):
13     """
14     Simulate the SIRS model with stochasticity and plot the results
15     ↪ .
16
17     Parameters:
18         ax (matplotlib.axes.Axes): Axes object to plot on.
19         t (numpy.ndarray): Array of time values.
20         rho (float): Rate of transmission.
21         alpha (float): Rate of infection.
22         lambda_val (float): Rate of recovery.
23         eta (float): Rate of reintroduction into the susceptible
24             ↪ population.
25         sigma (float): Volatility parameter.
26
27     Returns:
28         None
29     """
30
31     # Initialize arrays to store the results
32     susceptible = np.zeros_like(t)
33     infected = np.zeros_like(t)
34     recovered = np.zeros_like(t)
35
36     # Set initial values
37     susceptible[0], infected[0], recovered[0] = initial_S,
38     ↪ initial_I, initial_R
39
40     # Perform Euler-Maruyama integration
41     for i in range(1, len(t)):
42         dW_S = np.random.normal(0, np.sqrt(dt))
43         dW_I = np.random.normal(0, np.sqrt(dt))

```

```

40     dSdt = rho * (1 - susceptible[i-1]) + eta * recovered[i-1]
41         ↪ - alpha * susceptible[i-1] * infected[i-1]
42     dIdt = alpha * susceptible[i-1] * infected[i-1] - (rho +
43         ↪ lambda_val) * infected[i-1]
44     dRdt = lambda_val * infected[i-1] - (rho + eta) * recovered
45         ↪ [i-1]
46     susceptible[i] = susceptible[i-1] + dSdt * dt + sigma *
47         ↪ susceptible[i-1] * infected[i-1] * dW_S
48     infected[i] = infected[i-1] + dIdt * dt + sigma *
49         ↪ susceptible[i-1] * infected[i-1] * dW_I
50     recovered[i] = recovered[i-1] + dRdt * dt
51
52 # Plot the results
53 ax.plot(t, susceptible, label='Susceptible', color='blue')
54 ax.plot(t, infected, label='Infected', color='red')
55 ax.plot(t, recovered, label='Recovered', color='green')
56 ax.set_xlabel('Time', color='magenta')
57 ax.set_ylabel('Proportion: S, I and R', color='lime')
58 ax.set_title(f'Stochastic Simulation of SIRS Model: Extinction
59     ↪ case\nInitial Values: (S={case["initial_S"]}, I={case[
60         ↪ initial_I"]}, R={case["initial_R"]})', transform=ax.
61         ↪ transAxes, fontsize=10, verticalalignment='top',
62         ↪ horizontalalignment='center', color='olive')
63 ax.legend()
64 ax.legend()
65 ax.grid(True)
66
67 # Calculate T3 and T4
68 T3 = alpha / (rho + (1/4) * sigma**2)
69 T4 = ((1/2) * sigma**2)
70
71 # Add parameter values below the x-axis
72 parameter_offset = -0.18 # Offset in centimeters (1.5 cm $\\
73     ↪ approx$ 0.59 inches)
74 t_values_offset = -0.1 # Offset in centimeters (1.5 cm $\\
75     ↪ approx$ 0.59 inches)
76
77 ax.text(0.03, parameter_offset, f'$\\rho={display_number(rho)}$'
78     ↪ ', color='teal', transform=ax.transAxes, fontsize=10,
79     ↪ verticalalignment='top')
80 ax.text(0.38, parameter_offset, f'$\\alpha={display_number(
81         ↪ alpha)}$', color='teal', transform=ax.transAxes,
82         ↪ fontsize=10, verticalalignment='top')
83 ax.text(0.72, parameter_offset, f'$\\lambda={display_number(
84         ↪ lambda_val)}$', color='teal', transform=ax.transAxes,
85         ↪ fontsize=10, verticalalignment='top')
86
87 ax.text(0.18, parameter_offset-0.07, f'$\\eta={display_number(
88         ↪ eta)}$', color='teal', transform=ax.transAxes, fontsize
89         ↪ =10, verticalalignment='top')
90 ax.text(0.58, parameter_offset-0.07, f'$\\sigma_{\{\\upsilon
91         ↪ \}}={display_number(sigma)}$', color='teal', transform=ax
92         ↪ .transAxes, fontsize=10, verticalalignment='top')

```

```

73     ax.text(0.18, t_values_offset, f'$\mathcal{T}^3$={
74         display_number(T3)}$', color='Maroon', transform=ax.
75         transAxes, fontsize=10, verticalalignment='top')
76     ax.text(0.58, t_values_offset, f'$\mathcal{T}^4$={
77         display_number(T4)}$', color='Maroon', transform=ax.
78         transAxes, fontsize=10, verticalalignment='top')

79 # Define the parameters for each case
80 cases = [
81     {"rho": 0.1, "alpha": 0.799999999, "lambda_val": 0.51, "eta": 0.05,
82      "sigma": 0.89, "initial_S": 0.35, "initial_I": 0.45, "initial_R": 0.2},
83     {"rho": 0.91, "alpha": 0.99, "lambda_val": 0.49, "eta": 0.05, "sigma": 0.599,
84      "initial_S": 0.35, "initial_I": 0.45, "initial_R": 0.2},
85     {"rho": 0.021, "alpha": 0.018, "lambda_val": 0.43, "eta": 0.05,
86      "sigma": 0.999999999, "initial_S": 0.35, "initial_I": 0.45, "initial_R": 0.2},
87     {"rho": 0.00011, "alpha": 0.46, "lambda_val": 0.00015, "eta": 0.05,
88      "sigma": 0.999999999, "initial_S": 0.35, "initial_I": 0.45, "initial_R": 0.2}
89 ]
90
91 try:
92     # Iterate over each case and plot the SIRS model
93     for i, case in enumerate(cases):
94         print(f"Case {i+1}:")
95         fig, ax = plt.subplots()
96         t_min, t_max = 1000, 1020
97         dt = 0.001 # You may need to adjust the step size for
98             # higher precision
99         num_steps = int((t_max - t_min) / dt)
100        t = np.linspace(t_min, t_max, num_steps)
101        simulate_and_plot_SIRS(ax, t, **case)
102        plt.tight_layout() # Adjust layout for better spacing
103        # Save the graph in EPS format
104        filename = f"graph_case_{i+1}.eps"
105        plt.savefig(filename, format='eps')
106        print(f"Saved graph to {filename}")
107        plt.show()

108 except Exception as e:
109     print("An error occurred during execution:")
110     print(e)

```

## A.5 Python Programs: Chapter 5

*Listing A.12. Python code for simulating Markov chains.*

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.linalg import expm
4

```

```
5 # Enable LaTeX in matplotlib
6 plt.rc('text', usetex=True)
7 plt.rc('font', family='serif')
8
9 # Parameters for the first Markov chain
10 Gamma1 = np.array([[[-3, 3],
11                    [4, -4]])
12 varsigma1 = 0.01
13 A1 = expm(Gamma1 * varsigma1)
14 pi1 = np.array([0.75, 0.25])
15 n_steps1 = 1000
16 initial_state1 = 0
17
18 # Parameters for the second Markov chain
19 Gamma2 = np.array([[[-1, 1],
20                    [2, -2]])
21 varsigma2 = 0.005
22 A2 = expm(Gamma2 * varsigma2)
23 pi2 = np.array([0.6667, 0.3333])
24 n_steps2 = 1000
25 initial_state2 = 0
26
27 # Function to simulate a Markov chain
28 def simulate_markov_chain(transition_matrix, initial_state, n_steps
29   ↪ ):
30   """Simulate a Markov chain given its transition matrix.
31
32   Args:
33     transition_matrix: 2D array representing state transition
34       ↪ probabilities
35     initial_state: Starting state of the chain
36     n_steps: Number of steps to simulate
37
38   Returns:
39     Array of states visited during the simulation
40   """
41
42   states = np.zeros(n_steps, dtype=int)
43   states[0] = initial_state
44   for t in range(1, n_steps):
45     current_state = states[t - 1]
46     states[t] = np.random.choice([0, 1], p=transition_matrix[
47       ↪ current_state])
48   return states
49
50 # Simulate both Markov chains
51 states1 = simulate_markov_chain(A1, initial_state1, n_steps1)
52 states2 = simulate_markov_chain(A2, initial_state2, n_steps2)
53
54 # Create the plots
55 fig, axes = plt.subplots(1, 2, figsize=(16, 8))
56
57 # Plot for the first Markov chain
58 axes[0].step(range(n_steps1), states1 + 1, where='post', linewidth
59   ↪ =2, color='#9ACD32') # LimeGreen
```

```

55 axes[0].set_yticks([1, 2])
56 axes[0].set_yticklabels([r'$e_1$', r'$e_2$'], fontsize=18)
57 axes[0].set_xlabel(r'\textbf{Time}', fontsize=14)
58 axes[0].set_ylabel(r'\textbf{State}', fontsize=14)
59 axes[0].set_title(r'\textbf{\Large{Example 5.1}}', fontsize=16,
60                   bbox=dict(facecolor='#9ACD32', edgecolor='black',
61                               boxstyle='round, pad=0.5'))
62 axes[0].grid()
63
64 # Plot for the second Markov chain
65 axes[1].step(range(n_steps2), states2 + 1, where='post', linewidth
66               =2, color='#FFA500') # Orange
67 axes[1].set_yticks([1, 2])
68 axes[1].set_yticklabels([r'$e_1$', r'$e_2$'], fontsize=18)
69 axes[1].set_xlabel(r'\textbf{Time}', fontsize=14)
70 axes[1].set_ylabel(r'\textbf{State}', fontsize=14)
71 axes[1].set_title(r'\textbf{\Large{Example 5.2}}', fontsize=16,
72                   bbox=dict(facecolor='#FFA500', edgecolor='black',
73                               boxstyle='round, pad=0.5'))
74 axes[1].grid()
75
76 # Adjust layout to avoid overlap
77 plt.tight_layout()
78
79 # Save the figure in EPS format
80 plt.savefig("markov_chain_comparison.eps", format='eps')
81
82 # Display the plot
83 plt.show()

```

**Listing A.13.** Stochastic SIRS Model with Switching Based on the Markov Chain from Example

5.1—Cases a, b, and c

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.integrate import solve_ivp
4 from scipy.linalg import expm
5
6 # Generator matrix Gamma and transition matrix A
7 Gamma = np.array([[-3, 3], [4, -4]]) # Generator matrix
8 varsigma = 0.01 # Time step
9 A = expm(Gamma * varsigma) # Matrix exponential
10 A = A / A.sum(axis=1, keepdims=True) # Normalization to ensure
11     # rows sum to 1
12 assert np.allclose(A.sum(axis=1), 1), "Transition matrix rows must
13     # sum to 1"
14 pi = np.array([0.75, 0.25]) # Stationary distribution
15 states = [1, 2] # Possible states
16
17 def simulate_markov_chain(t_max, dt=0.01):
18     """Simulate a continuous-time Markov chain.
19
20     Args:

```

```
21     t_max: Maximum simulation time
22     dt: Time step size
23
24     Returns:
25         List of states visited during simulation
26     """
27
28     n_steps = int(t_max / dt)
29     state = np.random.choice(states, p=pi) # Initial state from
30         ↪ stationary distribution
31     trajectory = [state]
32     for _ in range(n_steps):
33         next_state_prob = A[states.index(state)] # Transition
34             ↪ probabilities
35         next_state = np.random.choice(states, p=next_state_prob)
36         trajectory.append(next_state)
37         state = next_state
38     return trajectory
39
40
41 # Parameter sets for three different cases
42 params_dict = {
43     1: { # Case a
44         'zeta1': 0.5, 'rho1': 0.5, 'mu1': 0.999, 'beta1': 0.3,
45             ↪ eta1': 0.5, 'delta1': 0.9899, 'sigma1': 0.9999,
46         'zeta2': 0.3, 'rho2': 0.5, 'mu2': 0.887, 'beta2': 0.4,
47             ↪ eta2': 0.5, 'delta2': 0.988, 'sigma2': 0.8899
48     },
49     2: { # Case b
50         'zeta1': 1.5, 'rho1': 0.5, 'mu1': 0.9999, 'beta1': 0.1,
51             ↪ eta1': 0.5, 'delta1': 0.5, 'sigma1': 0.8,
52         'zeta2': 1.1, 'rho2': 0.7, 'mu2': 0.61, 'beta2': 0.3, 'eta2':
53             ↪ : 0.3, 'delta2': 0.3, 'sigma2': 0.9
54     },
55     3: { # Case c
56         'zeta1': 1.2, 'rho1': 0.6, 'mu1': 2, 'beta1': 2.75, 'eta1':
57             ↪ 0.5, 'delta1': 3, 'sigma1': 0.003,
58         'zeta2': 1.5, 'rho2': 0.8, 'mu2': 2.5, 'beta2': 2.5, 'eta2':
59             ↪ : 0.5, 'delta2': 3, 'sigma2': 0.001
60     }
61 }
62
63 def model(t, y, params):
64     """Differential equations for the SIRS model with switching.
65
66     Args:
67         t: Current time
68         y: Current state [S, I, R]
69         params: Dictionary containing parameters and Markov chain
70             ↪ state
71
72     Returns:
73         List of derivatives [dS, dI, dR]
74     """
75
76     S, I, R = y
77     state_index = min(len(params['state']) - 1, int(t / params['dt']
```

```

    ↪ ])) # Prevent index overflow
66 state = params['state'][state_index]
67 p = params_dict[state]
68
69 # Extract parameters for current state
70 zeta, rho, mu, beta, eta, delta, sigma = (
71     p[f'zeta{state}'], p[f'rho{state}'], p[f'mu{state}'],
72     p[f'beta{state}'], p[f'eta{state}'], p[f'delta{state}'], p[
73         ↪ f'sigma{state}']
74 )
75
76 # Stochastic differential equations
77 dS = (mu - mu * S - beta * S**rho * I**zeta + eta * R) * params
78     ↪ ['dt'] \
79     - sigma * S**rho * I**zeta * np.sqrt(params['dt']) * np.
80         ↪ random.randn()
81 dI = (- (mu + delta) * I + beta * S**rho * I**zeta) * params[
82     ↪ dt'] \
83     + sigma * S**rho * I**zeta * np.sqrt(params['dt']) * np.
84         ↪ random.randn()
85 dR = (- (mu + eta) * R + delta * I) * params['dt']
86
87     return [dS, dI, dR]
88
89 def solve_system(params, t_max=1000, dt=0.01):
90     """Solve the SIRS system with Markov switching.
91
92     Args:
93         params: Parameters dictionary
94         t_max: Maximum simulation time
95         dt: Time step size
96
97     Returns:
98         Tuple of time points and solution array
99     """
100
101 t = np.linspace(0, t_max, int(t_max / dt) + 1)
102 y0 = [0.35, 0.55, 0.1] # Initial conditions [S, I, R]
103 state_trajectory = simulate_markov_chain(t_max, dt)
104 params['state'] = state_trajectory
105 sol = solve_ivp(lambda t, y: model(t, y, params), [0, t_max],
106     ↪ y0, t_eval=t)
107 return sol.t, sol.y
108
109 def plot_results():
110     """Run simulations and plot results for all three cases."""
111     cases = ['a', 'b', 'c']
112
113     for i, case in enumerate(cases):
114         plt.figure(figsize=(8, 6))
115         params = {'dt': 0.01}
116         params['state'] = simulate_markov_chain(1000, dt=0.01)
117         t, y = solve_system(params, t_max=1000, dt=0.01)
118
119         plt.plot(t, y[0], 'g', label='Susceptible (S)')

```

```

113     plt.plot(t, y[1], 'r', label='Infected (I)')
114     plt.plot(t, y[2], 'b', label='Recovered (R)')
115     plt.title(f'Case {case} - SIRS Model with Markov Switching'
116               , bbox=dict(facecolor='lightblue', edgecolor='black',
117                           boxstyle='round, pad=0.5'))
117     plt.xlabel('Time')
118     plt.ylabel('Population Proportion')
119     plt.legend()
120     plt.grid(True)
121     plt.tight_layout()
122     plt.savefig(f'sirs_markov_case_{case}.eps', format='eps')
123     plt.show()
124
125 # Execute the simulation and visualization
126 plot_results()

```

**Listing A.14.** Stochastic SIRS Model with Markov Chain Switching from Example 5.2 — Cases a, b, and c

```

1 import numpy as np
2 import matplotlib.pyplot as plt
3 from scipy.integrate import solve_ivp
4 from scipy.linalg import expm
5
6 # Markov chain parameters (generator and transition matrix)
7 Gamma = np.array([[-1, 1], [2, -2]]) # Updated generator matrix
8 dt = 0.0001
9 A = np.linalg.matrix_power(np.exp(Gamma * dt), 1) # Transition
10    ↪ matrix for timestep 0.0001
11 pi = np.array([0.6667, 0.3333]) # Stationary distribution
12 states = [1, 2] # Markov chain states
13
14 # Markov chain simulation function
15 def simulate_markov_chain(t_max, dt=0.01):
16     """
17         Simulates the Markov chain over a given period with step dt.
18
19         t_max : float : Maximum simulation time
20         dt : float : Simulation timestep (default 0.01)
21
22         returns : list : State trajectory over time [1, 2]
23     """
24     n_steps = int(t_max / dt)
25     state = np.random.choice(states, p=pi) # Initial state sampled
26         ↪ from stationary distribution
27     trajectory = [state]
28
29     for _ in range(n_steps):
30         next_state_prob = A[state - 1] # Transition probabilities
31             ↪ for current state
32
33         # Normalize probabilities if necessary
34         if not np.isclose(next_state_prob.sum(), 1):
35             next_state_prob /= next_state_prob.sum()

```

```

34     next_state = np.random.choice(states, p=next_state_prob)
35     trajectory.append(next_state)
36     state = next_state
37
38     return trajectory
39
40 # Parameter sets for different cases
41 params_dict = {
42     1: { # Case a
43         'zeta1': 0.5, 'rho1': 0.5, 'mu1': 0.999, 'beta1': 0.3, '
44             ↪ eta1': 0.5, 'delta1': 0.9899, 'sigma1': 0.9999,
45         'zeta2': 0.3, 'rho2': 0.5, 'mu2': 0.887, 'beta2': 0.4, '
46             ↪ eta2': 0.5, 'delta2': 0.988, 'sigma2': 0.8899
47     },
48     2: { # Case b
49         'zeta1': 1.5, 'rho1': 0.5, 'mu1': 0.9999, 'beta1': 0.1, '
50             ↪ eta1': 0.5, 'delta1': 0.5, 'sigma1': 0.8,
51         'zeta2': 1.1, 'rho2': 0.7, 'mu2': 0.61, 'beta2': 0.3, 'eta2'
52             ↪ ': 0.3, 'delta2': 0.3, 'sigma2': 0.9
53     },
54     3: { # Case c
55         'zeta1': 1.2, 'rho1': 0.6, 'mu1': 2, 'beta1': 2.75, 'eta1':
56             ↪ 0.5, 'delta1': 3, 'sigma1': 0.003,
57         'zeta2': 1.5, 'rho2': 0.8, 'mu2': 2.5, 'beta2': 2.5, 'eta2'
58             ↪ : 0.5, 'delta2': 3, 'sigma2': 0.001
59     }
60 }
61
62 # SIRS model function
63 def model(t, y, params):
64     S, I, R = y
65     state_index = min(len(params['state']) - 1, int(t / params['dt']
66             ↪ ])) # Avoid index overflow
67     state = params['state'][state_index]
68     p = params_dict[state]
69
70     zeta1, rho1, mu1, beta1, eta1, delta1, sigma1 = (
71         p['zeta1'], p['rho1'], p['mu1'], p['beta1'], p['eta1'], p[
72             ↪ 'delta1'], p['sigma1']
73     )
74
75     dS = (mu1 - mu1 * S - beta1 * S**rho1 * I**zeta1 + eta1 * R) *
76             ↪ params['dt'] \
77             - sigma1 * S**rho1 * I**zeta1 * np.sqrt(params['dt']) * np
78                 ↪ .random.randn()
79     dI = (- (mu1 + delta1) * I + beta1 * S**rho1 * I**zeta1) *
80             ↪ params['dt'] \
81             + sigma1 * S**rho1 * I**zeta1 * np.sqrt(params['dt']) * np
82                 ↪ .random.randn()
83     dR = (- (mu1 + eta1) * R + delta1 * I) * params['dt']
84
85     return [dS, dI, dR]
86
87 # System solver

```

```

76 def solve_system(params, t_max=1000, dt=0.01):
77     t = np.linspace(0, t_max, int(t_max / dt) + 1)
78     y0 = [0.35, 0.55, 0.1] # Initial conditions: S, I, R
79     state_trajectory = simulate_markov_chain(t_max, dt)
80     params['state'] = state_trajectory
81     sol = solve_ivp(lambda t, y: model(t, y, params), [0, t_max],
82                      y0, t_eval=t)
83     return sol.t, sol.y
84
85 # Plotting function
86 def plot_results():
87     cases = ['a', 'b', 'c']
88
89     for i, case in enumerate(cases):
90         plt.figure(figsize=(8, 6)) # Create new figure per case
91         params = {'dt': 0.01}
92         params['state'] = simulate_markov_chain(1000, dt=0.01)
93         t, y = solve_system(params, t_max=1000, dt=0.01)
94
95         plt.plot(t, y[0], 'g', label='S(t)') # Susceptible - green
96         plt.plot(t, y[1], 'r', label='I(t)') # Infected - red
97         plt.plot(t, y[2], 'b', label='R(t)') # Recovered - blue
98         plt.title(f'Case {case}', bbox=dict(facecolor='lightblue',
99                   edgecolor='black', boxstyle='round', pad=0.5))
100        plt.xlabel('Time t')
101        plt.ylabel('Population fractions')
102        plt.legend(loc='best')
103        plt.tight_layout()
104        plt.savefig(f'output_trajectory_case_{case}.eps', format='
105                      eps') # Save each plot separately
106        plt.show()

# Run simulation and plot
plot_results()

```

## A.6 Conclusion

This chapter has systematically outlined the Python implementations used throughout this thesis, offering detailed information on the numerical simulations, data visualizations, and computational techniques that support the theoretical findings. By maintaining clarity and rigor in the code structure and documentation, this work ensures transparency and reproducibility, which are fundamental in scientific research. The presented programs provide a solid foundation for further exploration, optimization, and extension of the studied models, reinforcing the importance of computational approaches in modern scientific investigations.

## APPENDIX B

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### Table of Common Stochastic Integrals

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This appendix presents a systematically organized collection of stochastic integral identities, carefully selected for their utility in probability theory and stochastic calculus. Although mathematically rich, the inherent complexity of these formulas makes complete memorization challenging.

Let  $a < b$  and  $0 < T$ . We then have:

1.  $\int_a^b dW_t = W_b - W_a;$
2.  $\int_0^T W_t dW_t = \frac{W_T^2}{2} - \frac{T}{2};$
3.  $\int_0^T (W_t^2 - t) dW_t = \frac{W_T^3}{3} - TW_T;$
4.  $\int_0^T t dW_t = TW_T - \int_0^T W_t dt, \quad 0 < T;$
5.  $\int_0^T W_t^2 dW_t = \frac{W_T^3}{3} - \int_0^T W_t dt;$
6.  $\int_0^T e^{\frac{t}{2}} \cos W_t dW_t = e^{\frac{T}{2}} \sin W_T;$
7.  $\int_0^T e^{\frac{t}{2}} \sin W_t dW_t = 1 - e^{\frac{T}{2}} \cos W_T;$
8.  $\int_0^T e^{-\frac{t}{2} + W_t} dW_t = e^{-\frac{T}{2} + W_T} - 1;$
9.  $\int_0^T e^{\frac{\lambda^2 t}{2}} \cos(\lambda W_t) dW_t = \frac{1}{\lambda} e^{\frac{\lambda^2 T}{2}} \sin(\lambda W_T);$
10.  $\int_0^T e^{\frac{\lambda^2 t}{2}} \sin(\lambda W_t) dW_t = \frac{1}{\lambda} \left( 1 - e^{\frac{\lambda^2 T}{2}} \cos(\lambda W_T) \right);$
11.  $\int_0^T e^{-\frac{\lambda^2 t}{2} + \lambda W_t} dW_t = \frac{1}{\pm \lambda} \left( e^{-\frac{\lambda^2 T}{2} + \lambda W_T} - 1 \right);$
12.  $\int_a^b t^{-\frac{3}{2}} W_t e^{-\frac{W_t^2}{2t}} dW_t = a^{-\frac{1}{2}} e^{-\frac{W_a^2}{2a}} - b^{-\frac{1}{2}} e^{-\frac{W_b^2}{2b}};$
13.  $\int_0^T \cos W_t dW_t = \sin W_T + \frac{1}{2} \int_0^T \sin W_t dt;$

- 
14.  $\int_0^T \sin W_t dW_t = 1 - \cos W_T - \frac{1}{2} \int_0^T \cos W_t dt;$
15.  $d\left(\int_a^t f(s, W_s) dW_s\right) = f(t, W_t) dW_t;$
16.  $\int_a^b Y_t dW_t = F_b - F_a$ , when  $Y_t dW_t = dF_t$ ;
17.  $\int_a^b f(t) dW_t = f(t)W_t|_a^b - \int_a^b f'(t)W_t dt;$
18.  $\int_a^b g'(W_t) dW_t = g(W_t)|_a^b - \frac{1}{2} \int_a^b g''(W_t) dt.$

# APPENDIX C

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## Chronology & Lexicon

### C.1 Historical Introduction to Stochastic Calculus Terminology

The terminology of stochastic calculus reflects its multidisciplinary origins, where physics, mathematics, and finance intersected to model randomness in continuous time. This chapter presents a dual perspective: the conceptual evolution through key terms (Subsection C.1.1) and the human lineage of discovery (Subsection C.1.2).

#### C.1.1 Lexical Foundations

The bilingual glossary reveals several historical layers.

- **Botanical Origins:** The term *Brownian motion* (1827) predates its rigorous mathematical formulation (Wiener, 1923) by nearly a century, honoring Robert Brown's observations of pollen particles.
- **Martingale Semantics:** The gambling term *martingale*, originating in 18th-century French, was formalized mathematically by Doob in the 1940s, while the concept of *local martingale* emerged from Itô's school in the 1950s.
- **Notational Variants:** The Stratonovich integral (1960s) retains its cyclical-origin spelling, contrasting with the Anglicized *Itô* and the Hepburn romanization of *Itô*'s lemma.

#### C.1.2 Chronological Paradigms

The timeline highlights three distinct eras.

**1900–1940:** Foundational contributions by Einstein, Wiener, and Kolmogorov established the physical and measure-theoretic bases.

**1940–1980:** Itô, Doob, and Meyer developed the modern stochastic calculus framework, alongside parallel contributions from the French (Lévy, Malliavin) and Soviet (Stratonovich, Skorokhod) schools.

**1980–present:** Application-driven expansions in finance (Heston model) and rough path theory (Lyons), as well as Hairer's regularity structures, characterize this period.

This lexicon and chronology provide both linguistic reference points and historical orientation for the technical vocabulary developed in this work. Particular attention has been paid to the following.

- Preserving original naming conventions (for example, *Doléans-Dade* instead of *Dade-Doléans*).
- Standardization abbreviations (CLT/TCL) in linguistic traditions.

- Recognizing the hybrid etymology of terms such as *Ornstein-Uhlenbeck* (Dutch-German physics heritage).

## C.2 Bilingual English/French Lexicon

English	French
<b>Law of Large Numbers (LLN)</b>	Loi des grands nombres (LGN)
<b>Central Limit Theorem (CLT)</b>	Théorème central limite (TCL)
<b>Brownian motion</b>	Mouvement brownien
<b>Itô integral</b>	Intégrale d'Itô
<b>Stratonovich integral</b>	Intégrale de Stratonovich
<b>Quadratic variation</b>	Variation quadratique
<b>Martingale</b>	Martingale
<b>Local martingale</b>	Martingale locale
<b>Semimartingale</b>	Semi-martingale
<b>Itô's lemma</b>	Lemme d'Itô
<b>Doléans-Dade exponential</b>	Exponentielle de Doléans-Dade
<b>Predictable process</b>	Processus prévisible
<b>Adapted process</b>	Processus adapté
<b>Stochastic differential equation (SDE)</b>	Équation différentielle stochastique (EDS)
<b>Drift coefficient</b>	Coefficient de dérive
<b>Diffusion coefficient</b>	Coefficient de diffusion
<b>Strong solution</b>	Solution forte
<b>Weak solution</b>	Solution faible
<b>Existence and uniqueness</b>	Existence et unicité
<b>Fokker-Planck equation</b>	Équation de Fokker-Planck
<b>Kolmogorov equations</b>	Équations de Kolmogorov
<b>Geometric Brownian motion</b>	Mouvement brownien géométrique
<b>Ornstein-Uhlenbeck process</b>	Processus d'Ornstein-Uhlenbeck
<b>SIR model</b>	Modèle SIR
<b>Heston model</b>	Modèle de Heston
<b>Dynamical system</b>	Système dynamique
<b>Ergodic theory</b>	Théorie ergodique
<b>Invariant measure</b>	Mesure invariante
<b>Lyapunov exponent</b>	Exposant de Lyapunov
<b>Stochastic flow</b>	Flot stochastique
<b>Random dynamical system</b>	Système dynamique aléatoire
<b>Stochastic stability</b>	Stabilité stochastique
<b>Bifurcation theory</b>	Théorie des bifurcations
<b>Attractor</b>	Attracteur
<b>Strange attractor</b>	Attracteur étrange

**Note:** Translations follow standard probability conventions. The acronyms in parentheses are internationally recognized abbreviations.

### C.3 Historical Figures in Brownian Motion and Stochastic Calculus

Dates	Scientist
1975 –	Martin Hairer
1968 –	Wendelin Werner
1959 –	Jean-François Le Gall
1955 –	Alain-Sol Sznitman
1954 –	Dominique Bakry
1953 –	Terry Lyons
1951 –	David Nualart
1949 – 2014	Marc Yor
1947 –	Shige Peng
1947 –	Étienne Pardoux
1944 –	Nicole El Karoui
1944 –	Jean Jacod
1942 – 2004	Catherine Doléans-Dade
1940 –	S. R. Srinivasa Varadhan
1940 –	Daniel W. Stroock
1938 –	Mark Iosifovich Freidlin
1938 – 1995	Fischer Black
1935 –	Shin'ichi Watanabe
1934 –	Albert Shiryaev
1934 – 2003	Paul-André Meyer
1930 –	Henry McKean
1930 – 2011	Anatoliy Skorokhod
1930 – 1997	Ruslan Stratonovich
1927 – 2013	Donald Burkholder
1925 – 2010	Paul Malliavin
1924 – 2014	Eugene Dynkin
1923 – 2020	Freeman Dyson
1916 – 2008	Gilbert Hunt
1915 – 2008	Kiyosi Itô (Itô)
1915 – 1940	Wolfgang Doeblin
1914 – 1984	Mark Kac
1911 – 2004	Shizuo Kakutani
1910 – 2004	Joseph Leo Doob
1908 – 1989	Robert Horton Cameron
1906 – 1970	William Feller
1903 – 1987	Andrey Kolmogorov
1900 – 1988	George Uhlenbeck
1896 – 1971	Paul Lévy
1894 – 1964	Norbert Wiener
1879 – 1955	Albert Einstein
1875 – 1941	Henri Lebesgue
1872 – 1946	Paul Langevin
1872 – 1917	Marian Smoluchowski
1871 – 1956	Émile Borel
1870 – 1942	Jean Perrin
1870 – 1946	Louis Bachelier
1856 – 1922	Andrey Markov
1856 – 1894	Thomas Stieltjes
1773 – 1858	Robert Brown

### C.3. HISTORICAL FIGURES IN BROWNIAN MOTION AND STOCHASTIC CALCULUS

**Historical Note:** This list includes key contributors to the theoretical development of Brownian motion and stochastic calculus, arranged in reverse chronological order. Names are presented according to their linguistic conventions (e.g., Itô/Itō respecting Japanese transcription).

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# Scientific Publications

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1. El Bakkioui, K., El Khalfi, Y., Boutouil, S., Harchaoui, B., El Idrissi, M., Settati, A., & Lahrouz, A. (2025). *Exploring the Impact of Jump Perturbations on Stochastic SIRS Dynamics*. **Applied Mathematics**, 19(3), 671–681. <https://www.scilit.com/publications/d0d8609e917fdabc8772ceccaab93322>
2. Esseroukh, M., Harchaoui, B., El Khatib, B., El Bakkioui, K., Aznague, S., Lahrouz, A., & Settati, A. (2025). *Logistic Growth and Relapse in the Stochastic Dynamics of SIRI Epidemics*. **Applied Mathematics**, 19(4), 739–750. <https://doi.org/10.1234/am.2025.19739>
3. Aznague, S., Settati, A., Lahrouz, A., Harchaoui, B., El Bakkioui, K., & Nait Brahim, A. (2025). *Threshold Dynamics of Stochastic SIS Epidemic Models with Logistic Recruitment Rate*. **New Mathematics and Natural Computation**, 1–24. World Scientific. <https://www.worldscientific.com/doi/10.1142/S1793005725500127>
4. El Khatib, B., Harchaoui, B., Esseroukh, M., Aznague, S., El Bakkioui, K., El Khalfi, Y., Settati, A., & Lahrouz, A. (2025). *Stochastic Analysis of COVID-19 Epidemics Under Quarantine Measures*. **New Mathematics and Natural Computation**. World Scientific. <https://doi.org/10.1142/S1793005727500244>
5. Settati, A., Caraballo, T., Harchaoui, B., Lahrouz, A., El Bakkioui, K., & El Haitami, A. (2025). *Impact of Scale-Free Network Structures on Stochastic SIRS Epidemics*. **Journal of Theoretical Biology**, 123456, 128745. <https://doi.org/10.1016/j.jtbi.2025.128745>
6. El Bakkioui, K., Settati, A., Harchaoui, B., Lahrouz, A., & Tridane, A. (2025). *Necessary and Sufficient Criteria for Stochastic SIRS Epidemic Models under Switched Transmission Rate Exponents*. **Results in Physics**. [Sous révision]

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# Scientific Communications

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- **JIAMA'24** (2024). 2nd Edition of the International Conference on Artificial Intelligence and Applied Mathematics. Faculty of Sciences and Technologies of Al Hoceima, Morocco, May 11, 2024. <https://jiama24.sciencesconf.org/>
- **SISFCA'24** (2024). The First International School on Fractional Calculus and Systems Theory. Meknes, Morocco, December 12–14, 2024. <https://sisfca24.sciencesconf.org/>
- **IWNAM: 3DTS25** (2025). International Workshop on Numerical Analysis and Modeling: Doctoral Training School (3rd Edition). Oral presentation: *Influence of Stochastic Jumps on the Dynamics of SIRS Systems*. Fez, Morocco, April 07–11, 2025. <https://idts25.sciencesconf.org/>
- **ICRAMCS 2025** (2025). 7th Edition of the International Conference on Research in Applied Mathematics and Computer Science. Presentation: *Study of the Effect of Jump Perturbations on the Stochastic Dynamics of SIRS Models*. Marrakech, Morocco, April 24–26, 2025. <https://icramcs2025.sciencesconf.org/data/>

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# Index of Notations and Abbreviations

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## A

- $A(x, j)$  diffusion matrix, 116
- $\alpha$  infection coefficient, 98
- $\alpha_t$  Markov chain, 79
- $\alpha_t$  infection rate, 24
- $a$  recovery rate, 26, 28, 35
- a.s. almost sure convergence, 47

## B

- $(b, \sigma^2, \nu)$  Lévy triplet , 57
- $B(t)$  Brownian motion, 68
- $B_t$  Brownian motion, 56, 57, 60, 79
- $B_t$  multidimensional Brownian motion, 59, 63
- $\beta$  disease transmission rate, 21
- $\beta$  transmission rate, 36, 38–40
- $\mathcal{B}$  Borel  $\sigma$ -algebra, 95
- $\mathcal{B}^d$  Borel sigma-algebra on  $\mathbb{R}^d$ , 44
- $b$  drift coefficient, 55
- $b$  infection rate, 26, 28, 30, 33, 35

## C

- $C_b$  bounded continuous functions, 96
- $C_b(D)$  space of bounded continuous functions on  $D$ , 88
- $\text{Cov}(X, Y)$  covariance, 45
- COVID-19, 20
- càdlàg right-continuous with left limits almost surely, 55

## D

- $\Delta t$  time increment, 26
- $\Delta$  probability simplex, 116

$\delta$  immunity loss rate, 40

$\delta_{ij}$  Kronecker delta, 59

$\xrightarrow{d}$  convergence in distribution, 47

$d$  death rate, 30

## E

- $\mathcal{E}$  state space, 116
- $\eta$  immunity loss rate, 98
- $\eta(u)$  Lévy exponent, 55
- $\mathbb{E}$  expectation, 69
- $\mathbb{E}(X)$  expectation, 45
- $\mathbb{E}(XY)$  expectation of product, 48
- $\mathbb{E}(X | \mathcal{G})$  conditional expectation, 49
- $\mathbb{E}(\mathbf{X} | \mathcal{G})$  vector conditional expectation, 50
- $e$  input rate, 30

## F

- $f_{(a,b)}$  function, 117
- $\mathcal{F}$  sigma-algebra, 44
- $\mathcal{F}_t^B$  natural filtration of Brownian motion, 57
- $\mathcal{F}_\tau$  stopped  $\sigma$ -algebra, 51
- $\overline{\mathcal{F}}_t$  completed filtration, 58
- $\overline{\mathcal{F}}$  completed  $\sigma$ -algebra, 45
- $f(S, I)$  mass incidence function, 33
- $f(x, t)$  drift coefficient, 66

## G

- $\Gamma$  generator matrix, 116
- $G, G^{-1}$  Bihari's function, 70
- $\gamma$  drift parameter, 79
- $\gamma$  recovery rate, 21, 36, 38–40

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 $g(x, t)$  diffusion coefficient, 66

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 $H(S)$ , 102

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 $I$  infectious individuals, 38, 39  
 $I(t)$  indefinite Itô integral, 62  
 $I(t)$  infected individuals, 36  
 $I(t)$  infected individuals at time  $t$ , 26, 28, 35  
 $I(t)$  infected population, 106  
 $I(t)$  local Itô integral, 64  
 $I(t)$  number of infected individuals at time  $t$ , 33  
 $I^{\zeta_{\xi(t)}}$ , 112  
 $S^{\rho_{\xi(t)}} I^{\zeta_{\xi(t)}}$  switched bilinear incidence term, 140  
 $\int g(t) dB_t$  Itô integral, 60  
 $\langle I \rangle_t$  quadratic variation, 62  
 $\mathbf{I}(\mathbf{t})$  infected individuals at time  $t$ , 2

J  
 $J$  jacobian matrix, 31

K  
 $\mathcal{K}$  class of comparison functions, 77  
 $k = \frac{1}{N - \frac{a}{b}}$  carrying capacity, 35

L  
 $\mathbf{L}^p$  space, 46  
 $L_t$  Lévy process, 79  
 $\Lambda$  Lambda, 136  
 $\Lambda$  threshold condition, 119  
 $\Lambda$  threshold parameter, 112, 119, 140  
 $\lambda$  Poisson intensity, 56  
 $\lambda$  Poisson rate, 79  
 $\lambda$  recovery rate, 98  
 $\lambda_t$  time-dependent intensity, 55  
 $\lambda_{\max}$  largest eigenvalue, 94  
 $\lambda_{\min}$  smallest eigenvalue, 92  
 $\limsup A_k$  limit superior of sets, 48  
 $\mathbf{L}^2$  Space of square-integrable random variables, 61  
 $\mathbf{L}^p$  space, 46  
 $\mathcal{L}$  generator, 90, 92, 93, 117  
 $\mathcal{L}$  generator of the process, 88  
 $\mathcal{L}$ -harmonic function, 87  
 $\mathcal{L}V(x, t)$ , 80

$\bar{\Lambda}_1, \bar{\Lambda}_2$  Lambda-bar, 132  
 $\tilde{\Lambda}_1, \tilde{\Lambda}_2$  Lambda-tilde, 132  
 $\xrightarrow{\mathbf{L}^p}$  convergence in  $\mathbf{L}^p$ , 47

M  
 $M_t$  martingale, 105  
 $\langle M \rangle_t$  Meyer angle bracket, 99  
 $\langle M \rangle_t$  quadratic variation, 53  
 $\langle M, N \rangle_t$  joint quadratic variation, 53  
 $\mathbb{M}^2$  Space of square-integrable processes, 60, 62  
 $\mathbb{M}^2$  matrix-valued integrable processes, 63  
 $\mathbb{M}_{\text{loc}}^2$  space of locally square-integrable processes, 64  
 $\mathbb{M}_0$  space of simple processes, 60  
 $\mu$  birth rate, 33  
 $\mu$  drift coefficient, 79  
 $\mu$  mortality rate, 36  
 $\mu_1$  mortality rate of susceptible individuals, 32  
 $\mu_2$  mortality rate of infected individuals, 32  
 $\mu_3$  mortality rate of recovered individuals, 32  
 $\mu_X$  distribution measure, 46

N  
 $N$  Poisson counting measure, 99  
 $N$  total population, 35, 36  
 $N$  total population size, 27  
 $N(dt, dx)$  Poisson random measure, 56  
 $N_t$  Poisson process, 55, 68, 79  
 $N_t$  counting process, 55  
 $\mathcal{N}(\mu, \sigma^2)$  normal distribution, 57  
 $\nu$  Lévy measure, 79  
 $\nu$  birth rate, 36  
 $\nu(dx)$  Lévy measure, 55  
 $\nu(dz, dt)$  compensated jump measure, 68  
 $\tilde{N}(dt, dz)$  compensated Poisson measure, 68  
 $\tilde{N}$  compensated Poisson measure, 99

O  
 $(\Omega, \mathcal{F}, \mathbf{P})$  probability space, 45  
 $\Omega$  sample space, 44  
ODE ordinary differential equation, 16, 17

P

- $\Pi(S)$ , 102  
 $\mathbf{P}$  probability measure, 45  
 $\mathbf{P}(A | B)$  conditional probability, 48  
 $\mathbf{P}_{x,\alpha}$  probability measure, 91  
 $\mathbf{P}_{x,i}$  probability measure for process starting at  $(x, i)$ , 89  
 $\pi$  Lévy measure, 99  
 $\xrightarrow{\text{P}}$  convergence in probability, 47  
 $p$  vaccination rate, 30, 40  
 $p_A$  asymptomatic probability, 24  
 $p_H$  hospitalization probability, 24  
 $p_c$  percolation threshold, 5
- Q**  
 $q$  fraction vaccinated, 40  
 $q_{ij}$  transition rates, 79
- R**  
 $R$  recovered, 109  
 $R$  recovered individuals, 38, 39  
 $R(t)$  recovered individuals, 36  
 $R(t)$  recovered individuals at time  $t$ , 2, 26  
 $R(t)$  recovered population, 106  
 $R_0$  basic reproduction number, 30, 31  
 $R_{\text{eff}}$  effective reproduction number, 4  
 $\mathbb{R}^d$  cartesian product of  $d$  copies of real numbers, 50  
 $\mathbb{R}_+^3$  positive real space, 116  
 $\rho$  birth/death rate, 98  
 $\rho(\cdot)$  spectral radius, 92  
 $\rho_{\xi(t)}$  switching exponent, 140  
 $r = bN - a$  growth rate, 35  
 $r$  loss of immunity rate, 32  
 $r$  rate of loss of immunity, 32
- S**  
 $S$  susceptible, 109  
 $S$  susceptible individuals, 38, 39  
 $S(t)$  number of susceptible individuals at time  $t$ , 33  
 $S(t)$  susceptible individuals, 36  
 $S(t)$  susceptible individuals at time  $t$ , 1, 26, 28, 35  
 $S(t)$  susceptible population, 106  
 $SIR$  susceptible-infected-recovered model, 40  
 $SIR/SIS/SIRS/SEIR/SVIS$  epidemiological models, 40  
 $S^{\rho_{\xi(t)}}$ , 112
- $S_h$  sphere of radius  $h$ , 77  
 $\sigma$  birth rate or immigration rate, 32  
 $\sigma$  diffusion coefficient, 55, 79  
 $\sigma$  incubation rate, 21, 36  
 $\sigma$  noise intensity, 41  
 $\sigma(X)$  sigma-algebra generated by  $X$ , 45  
 $\sigma(X_i \in I)$  sigma-algebra generated by a family, 45  
 $\sigma_D$  first hitting time of domain  $D$ , 88, 89  
 $\sigma_U$  first hitting time, 90  
 $\varsigma_v$  jump impact function, 99  
 $\text{scipy}$ , 16  
 $\text{solve_ivp}$ , 16  
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 $\text{SI}$  model, 33  
 $\text{SIR}$  model, 26–29, 32, 36, 98  
 $\text{SIRS}$  model, 33, 109  
 $\text{SIS}$  model, 34  
 $\text{SIR}$  model, 113  
 $\text{SIRS}$  epidemic model, 112  
 $\text{SIRS}$  model, 113
- T**  
 $\Theta_j(x)$  quadratic function, 119  
 $\mathcal{T}^1$  threshold, 106  
 $\mathcal{T}^1, \mathcal{T}^2$ , 102  
 $\mathcal{T}^2$  threshold, 106  
 $\mathcal{T}^3$  threshold, 104, 108  
 $\mathcal{T}^4$  threshold, 104, 108  
 $\tau_D$  first exit time from domain  $D$ , 87  
 $\tau_k$  jump times, 56  
 $\tau_t$  stopping time, 60  
 $t_h$  hospitalization time, 24  
 $t_i$  incubation time, 24  
 $t_p$  pre-symptomatic infectious period, 25  
 $t_s$  infectious period, 24  
 $t_{bh}$  time before hospitalization, 24
- V**  
 $V(S, I, R, j)$  Lyapunov function, 119  
 $V(X)$  variance, 45  
 $V_x$  partial derivatives, 66  
 $V_{xx}$  partial derivatives, 66  
 $\varrho, \varrho'$ , 102  
 $\varsigma_v$ , 102  
 $\vartheta$  birth/death rate, 40
- W**  
 $W(t)$  Brownian motion, 116
- X**

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$X(t)$ Markov process, 116	Y
$X_t$ Lévy process, 55	$Y_t$ compound Poisson process, 56
$X_t$ random walk process, 41	Z
$\xi_k$ jump sizes, 56	$\zeta_j$ exponent parameter, 119
$x(t)$ Itô process, 66	$\zeta_{\xi(t)}$ switching exponent, 140

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- analysis of persistence, 106
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