MATHEMATICAL MODELING OF THE IMPACT OF YOGA ON COMMUNICABLE DISEASE TRANSMISSION DYNAMICS

A DISSERTATION SUBMITTED TO

RESEARCH CENTER

NEPAL SANSKRIT UNIVERSITY

BELJHUNDI, DANG, NEPAL

FOR THE AWARD OF

DOCTOR OF PHILOSOPHY

IN MATHEMATICS

SUBMITTED BY:

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NOVEMBER 2025 AD

Declaration

Dissertation entitled **"Mathematical Modeling of the Impact of Yoga on Communicable Disease Transmission Dynamics"** which is being submitted to the Research Centre, Nepal Sanskrit University, for the award of the degree of Doctor of Philosophy (PhD), is a research work carried out by me under the supervision of Prof. Dr. Dinesh Panthi, Department of Mathematics, Valmeeki Vidyapeeth, Nepal Sanskrit University, and co-supervised by Prof. Dr. Chet Raj Bhatta, Central Department of Mathematics, Tribhuvan University, and Dr. Samir Shrestha, Department of Mathematics, School of Science, Kathmandu University, Nepal.

I hereby declare that the research work contained herein is entirely my own original work, except where states otherwise by reference or acknowledgment, and has not been submitted earlier in part or full in this or any other form to any university or institute, here or elsewhere, for the requirement for any other degree or professional qualification. Literature, data or works done by others and cited within this dissertation has been given due acknowledgment and listed in the reference section.

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Raghu Bir Bhatta

Recommendation of Supervisor

This is to recommend that Mr. Raghu Bir Bhatta has carried out research entitled **"Mathematical Modeling of the Impact of Yoga on Communicable Disease Transmission Dynamics"** for the award of Doctor of Philosophy (PhD) in Mathematics under my supervision. To my knowledge, this work has not been submitted for any other degree.

He has fulfilled all the requirements laid down by the Nepal Sanskrit University, for the submission of the dissertation for the award of PhD degree in Mathematics. So I forward it to the Research Centre, Nepal Sanskrit University for further evaluation.

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Date: November, 2025

Recommendation

This is to recommend that this dissertation entitled **"Mathematical Modeling of the Impact of Yoga on Communicable Disease Transmission Dynamics"** by Mr. Raghu Bir Bhatta, submitted in fulfillment of the requirement for the degree of Doctor of Philosophy in Mathematics under rules and regulation of Nepal Sanskrit University, Research Center during the period prescribed by the university, is a original work carried out under my guidance and supervision.

To the best of my knowledge, the matter embodied in this dissertation has not been submitted to any other institution for the award of any degree.

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Date: November, 2025

Recommendation

This is to recommend that this dissertation entitled **"Mathematical Modeling of the Impact of Yoga on Communicable Disease Transmission Dynamics"** by Mr. Raghu Bir Bhatta, submitted in fulfillment of the requirement for the Degree of Doctor of Philosophy in Mathematics under Nepal Sanskrit University, Research Center during the period prescribed by the university, is his own original work carried out under my guidance and supervision.

He has fulfilled all the requirements of the University. To the best of my knowledge, the matter embodied in this dissertation has not been submitted to any other institution for the award of any degree.

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Acknowledgements

The Almighty has been the source of my success, granting me the strength to achieve my goals. My PhD dissertation reflects his guidance, and I pledge to uphold its integrity always.

I owe my profound gratitude to my supervisor, Prof. Dr. Dinesh Panthi, Department of Mathematics, Valmiki Vidyapeeth, Nepal Sanskrit University, for his invaluable guidance, inspiration, meticulous suggestions, and constructive criticism throughout the course of this dissertation. The completion of this work would not have been possible without his dedicated efforts in steering me toward academic excellence. Beyond the dissertation, I have also benefited from his teachings on time management, rational problem-solving, diligence, humility, and sincerity, for which I shall remain eternally indebted.

This dissertation would not have been possible without the guidance and support of my research expert, Prof. Dr. Chet Raj Bhatta, Central Department of Mathematics, Tribhuvan University, Nepal, whose mentorship not only assisted this work but also inspired and motivated me to pursue it with integrity. His guidance fostered my academic engagement and perseverance.

I am also deeply indebted to my co-supervisor, Dr. Samir Shrestha, Department of Mathematics, School of Science, Kathmandu University, Nepal, for his valuable guidance, constructive criticism, and technical support in data modeling. His patience, encouragement, and insightful suggestions were instrumental in shaping this dissertation.

I would like to express my sincere gratitude to Prof. Dr. Sudhan Kumar Poudel, Executive Director of the Research Centre, Nepal Sanskrit University, for his constant support and assistance. I am also grateful to the executive members and the entire academic team of the Research Centre for their guidance and help throughout my coursework and beyond.

I am deeply grateful to my parents, Shree Jay Dev Bhatta and Late Mother Nirmala Bhatta, whose prayers and blessings were a constant source of strength. I also sincerely thank my wife, Saraswati; son Bigyan; daughter-in-law Purnima; daughters Durga and Asmita; brothers Keshab and Madan; and brothers-in-law Manamati and Nisha Joshi for their unwavering support and encouragement throughout this journey. I extend my gratitude to my friends, staff, and Nepal Mathematical Society members for their motivation and support.

Raghu Bir Bhatta

Place: Dhangadhi

Date: November ,2025

Abstract

The study of the impact of Yoga on epidemiology represents a multidisciplinary domain, integrating mathematics, biology, and Yoga philosophy. Despite significant advances in medical science, communicable diseases continue to pose a substantial burden on global populations, with far-reaching economic consequences. In many regions, pharmaceutical interventions are not sufficiently available to prevent disease spread or significantly reduce mortality. Consequently, investigating the role of Yoga in influencing the transmission dynamics of communicable diseases is both timely and essential. Motivated by Yoga philosophy, this research develops modified SIR and SIS models that incorporate Yoga as a preventive strategy. The primary objective of this work is to formulate mathematical models that capture the effects of Yoga on disease transmission dynamics.

In Chapter 3, an $SYIRS$ model is proposed by introducing a *Yoga Sadhaka* compartment (Y) into the classical SIR framework. Chapter 4 presents an improved SIR model incorporating Yoga awareness, wherein the disease transmission rate decreases as the population of Yoga-aware individuals increases, with the transmission rate modeled as a decreasing

function of Yoga-aware infected individuals. Chapter 5 formulates and analyzes an $SIQS$ model with a quarantine compartment under a saturated incidence rate. Recognizing that disease control measures may not benefit all individuals equally, Chapter 6 extends the analysis to account for hyper-susceptibility within populations. Stability analyses of these models employ mathematical techniques such as the Routh–Hurwitz criteria, Lyapunov's theorem, and the Poincaré–Bendixson theorem. Both simulated and real-world epidemiological data are analyzed using *Mathematica*.

Reproduction numbers are derived using the next-generation matrix method, and corresponding threshold conditions are formulated in terms of the Yoga reproduction number (R_e), Yoga awareness reproduction number (R_a), quarantine reproduction number (R_q), and hyper-susceptible reproduction number (R_h) in Chapters 3–6, respectively. The local and global stability properties of the model equilibria are examined, and sensitivity analyses are conducted to investigate the relationships among reproduction numbers, state variables, and model parameters. Numerical simulations are presented to illustrate the dynamical behaviors of the proposed models.

The results indicate that a disease-free equilibrium exists when the reproduction numbers are below one, whereas an endemic equilibrium arises when they exceed one. As the population of Yoga Sadhakas or Yoga-aware individuals increases, disease transmission progressively declines, ultimately leading to disease elimination. These preventive mea-

sures promote behavioral changes that reduce both susceptibility and infectivity, thereby contributing to disease control. Numerical simulations further confirm that the theoretical predictions closely align with data-based outcomes. Overall, the findings demonstrate that Yoga exerts a positive influence on limiting disease transmission and provide a foundation for designing novel disease-prevention policies with potential economic benefits.

List of Abbreviations

SIR	Susceptible Infected Recovered
SIRS	Susceptible Infected Recovered Susceptible
SYIRS	Susceptible Yoga Sadhaka Infected Recovered Susceptible
SIS	Susceptible Infected Susceptible
SIQS	Susceptible Infected Quarantine Susceptible
SI	Susceptible Infected
SEIV	Susceptible Expected Infected Vaccinated
SIQR	Susceptible Infected Quarantine Recovered
WHO	World Health Organization
AVAV	Ahar Vihar Achar Vichar
SEIR	Susceptible Expected Infected Recovered
SAIR	Susceptible Aware Infected Recovered
AIDS	Acquired Immuno Deficiency Syndrome
HIV	Human Immuno Deficiency Virus
H1N1	Swine Flu
NPIs	Non-pharmaceutical Interventions
ODEs	Ordinary Differential Equations
PDEs	Partial Differential Equations
BMI	Body Mass Index
BFM	Body Fat Mass
ATT	Anti-Tuberculosis Treatment
DNA	Deoxyribonucleic acid
RNA	Ribonucleic acid
DFE	Disease Free Equilibrium

EE	Endemic Equilibrium
COPD	Chronic Obstructive Pulmonary Disorder
COVID-19	Corona Virus Disease of 2019
SARS-CoV-2	Severe Acute Respiratory Syndrome-2
SWEIQR	Susceptible Aware Exposed Infected Quarentine Recovered
RT-PCR	Reverse Transcription Polymerase Chain Reaction

List of Symbols

R_0	Basic reproduction number
R_e	Yoga reproduction number
R_a	Yoga aware reproduction number
R_q	Quarantine reproduction number
R_h	Reproduction number with hyper-susceptibility
R^n	n-dimensional space
Λ	Recruitment rate
β	Disease transmission rate
ρ_1	Disease transmission rate in Yoga Sadhak class
β_1	Disease transmission rate in Yoga aware class
σ	Yoga Pranayam efficacy
m	Rate of increase of Yoga Sadhak individuals
γ	Recovery rate
ρ_2	Rate of waning immunity induced by Yoga Pranayama
μ	Natural death rate
c	Yoga awareness coverage level
λ	Rate of waning disease induced immunity or rate of flow from class I to Q
θ	Rate of flow from class Q to S or from S to H
α	Preventive measures taken for epidemic control(Yoga awareness)
A	Growth rate of susceptible
p	Proportion of A who are hyper-susceptible
π	Level of protection of susceptible individuals by better immunity
δ	Rate at which infected individuals are improved
d	Death rate due to disease

List of Tables

List of Figures

Contents

Chapter 1

General Introduction

Preamble

Communicable diseases pose a constant threat to humans worldwide. Every person can be affected by some form of communicable disease during their lifetime, making these diseases a global concern. It is essential to understand the transmission dynamics of these diseases and to develop effective prevention strategies, such as awareness campaigns and vaccines.

This work explores the transmission dynamics of communicable diseases using mathematical modeling, an interdisciplinary approach. Mathematical modeling is a powerful tool for understanding and analyzing disease transmission, playing a crucial role in medical sciences. There are various types of epidemic models, such as the SIS and SIR models, as well as more advanced models tailored to specific diseases. The SIR model, developed by Kermack et al. (1927), is particularly important in epidemiology. It describes the different stages of disease transmission in human populations and interprets them in terms of variables and parameters. These parameters provide information about communi-

cable diseases such as influenza, swine flu, COVID-19, and others. Various mathematical techniques are employed to determine the reproduction number, perform stability analysis, and calculate the force of infection.

This chapter presents the transmission mechanisms of communicable diseases along with some basic information about them. It also provides a brief introduction to biomathematics, as well as discussions on *Pranayama*, Yoga awareness, quarantine, and hyper susceptibility. Additionally, the chapter includes a review of the literature, research questions, objectives, and the problem statement.

Introduction to Biomathematics

Biology is a natural science that encompasses the life, physical, and chemical structures of organisms, their development, and evolution. It provides a study of various scientific aspects of humans, plants, and animals. The study of human biology involves genetics, epidemiology, physiology, ecology, nutrition, and other related domains.

Mathematics has always benefited from its involvement with developing sciences. Each successive interaction regenerates and enhances its field. It is the leading science of the predictable future. It is evident that if mathematicians do not become involved in the biosciences, they will not be part of what are likely to be the most important and exciting scientific discoveries of all time. Mathematical biology is a fast-growing, well-established, and highly stimulating application of mathematics. The increasing use of mathematics in biology is predictable as biology be-

comes more quantitative and makes interdisciplinary involvement essential. Biology opens up new and exciting branches for mathematicians, while for biologists it offers another research tool commensurate with a new, powerful laboratory technique, which is considered useful, interesting, and biologically relevant (J. Murray, 2004; Zill, 2012). From a mathematical point of view, the art of good modeling of biological phenomena relies on:

a. A sound understanding and appreciation of the biological problem.

b. A realistic mathematical representation of the important biological phenomena.

c. Finding useful (preferably quantitative) solutions and interpreting them biologically in terms of insights and predictions.

Microbiology investigates the transmission dynamics of a wide range of communicable diseases, including COVID-19, malaria, swine flu, influenza, and measles. These diseases are caused by bacteria, viruses, fungi, and parasites. These agents cause illnesses in humans and can be harmful to the body in certain situations. Certain illnesses are infectious and can be transmitted from an infected individual to a healthy one. Susceptibility level, latent period, incubation time, transmission mode, and the infectious agent are factors influencing infection (Martcheva, 2015). Furthermore, viruses possess the following abilities that enable them to spread diseases (Elezkurtaj et al., 2021):

a. Ability to multiply inside a host cell.

b. Ability to cause disease.

c. Possession of nucleic acids, proteins, enzymes, and related components.

d. Ability to undergo mutation.

The characteristics of any virus causing a particular disease are difficult to recognize within a large population. During winter, viral infections occur in many regions. In the human population, the epidemiological system often follows a pattern in which the rate of infectious disease increases over time and, after completing the infection cycle, decreases gradually, as shown in Figure 1.1. In epidemiology, the term "epidemic"

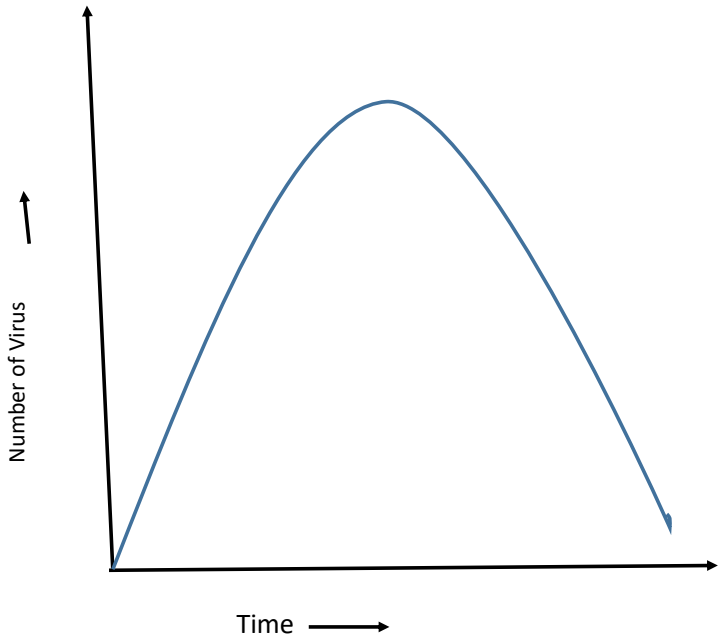


Figure 1.1: Graphic presentation of an acute viral infection.

refers to a sudden outbreak of an infectious disease that spreads rapidly through a population, affecting a large proportion of individuals. If it per-

sists for a long time, it becomes “endemic.” The spread of common communicable diseases is a major global issue that must be addressed through various public health programs. The spread of communicable diseases, their key characteristics, and their impact on human populations can be described through mathematical modeling, which incorporates possible risk factors, treatment modalities, health policies, and new opportunities for prevention, planning, and treatment. The emerging and increasingly essential application of biomathematics makes biology more comprehensive. Biomathematics opens up new paradigms of research, where mathematicians and biologists collaborate to provide suitable solutions to practitioners working in the medical domain through mathematical modeling. These biomathematical models are based on assumptions derived from real-life observations to ensure that the model functions in the desired direction. This interdisciplinary approach plays a significant role in analyzing the various complexities of biological systems.

Epidemic parameters, such as the basic reproduction number and certain threshold conditions, play a vital role in determining the extent of a disease. Many researchers have conducted studies on different types of infectious diseases. Some have focused on epidemiology, environment-based human infections, and other related diseases. They have proposed various techniques, such as the evaluation of the immune system, analysis of disease dynamics, assessment of vaccination and awareness effects, and the influence of environmental factors on the emergence and persistence of diseases. However, the epidemic analysis of communicable dis-

eases in relation to *yoga* and *yoga awareness* remains unexplored. Various techniques and mathematical models are used to measure the cause, transmission rate, reproduction rate, and effectiveness of prescribed treatments for communicable diseases. These models assist in studying the various stages of diseases under specific conditions for better decision-making in disease prevention. In this research work, extended forms of the SIR and SIS models are proposed. Mathematical modeling in this research consists of the following steps:

1. Model formulation
2. Evaluation of parameters
3. Investigation of sensitivity and stability of the model
4. Verification and simulation

Introduction to Mathematical Modeling

Mathematical modeling is a powerful tool of the scientific method in which the real world (external world) and the conceptual world (the world of the mind) are identified. The conceptual world is the realm of the mind in which we live when we try to understand what is happening in the real (external) world. The conceptual world has three stages: observation, modeling, and prediction (Dym, 2004). Observation measures what is happening in the real world; it may be direct (using our senses) or indirect. Mathematical models describe the behavior or results observed

and explain why those behaviors and results occurred, or they allow us to predict future behaviors or results that are yet unseen or unmeasured.

Mathematical modeling of any real-life problem provides the theoretical and numerical analysis of the problem, which assists in decision making and in understanding the problem in the language of the real world. The construction of any model is a process based on a set of observations and experiments. Mathematical modeling for a particular problem is determined by collecting information about the problem, presenting it in numerical form, and finally analyzing it in relation to the given problem.

The primary aim of a modeler is to quantify the abstract behavior of natural phenomena and to describe real-world problems. Direct experimentation on all real-world problems is not always possible, as such experiments may be time-consuming, expensive, dangerous, or even impossible in some cases. Mathematical modeling is used to describe such situations. The challenge in mathematical modeling is not to produce the most comprehensive descriptive model but to develop the simplest possible model that captures the major features of the phenomenon of interest. Mathematical modeling is a research tool that converts sophisticated mathematical ideas into detailed programs of experimental design and establishes the theoretical framework of a system by considering all relevant premises (N. Britton, 2005; Ma, Zhou, & Wu, 2009). In biomathematical modeling, the creation of a model is often grounded in the functionality of human life. In this work, the process of model development

is governed by the following steps:

1. Selection of a communicable disease as the real-world problem.
2. Formulation of a mathematical model with a certain number of variables and parameters based on defined assumptions.
3. Establishing relationships between variables and parameters using appropriate mathematical ideas and methods. The significant contributions of the study are discussed in this step, which provides new relationships between the obtained mathematical results and the situation being studied.
4. Interpretation of the results based on the mathematical framework, representing the practical world. Ideally, the results obtained through this mathematical modeling effort should be consistent with those of previous studies.

The complete cycle of the model-building process involves multiple refinements and compensations in successive steps until an acceptable model is achieved, as shown in Figure 1.2. The final model-building process demonstrates the formation, development, and evaluation of a mathematical model. The main purpose of model building is that the process should be both significant and straightforward, with the goal of producing predictions and conclusions based on a specific mathematical model. In short, mathematical modeling translates human problems, experiences, and phenomena into the precise and concise language of mathematics. Mathematics has the potential to provide results that describe a

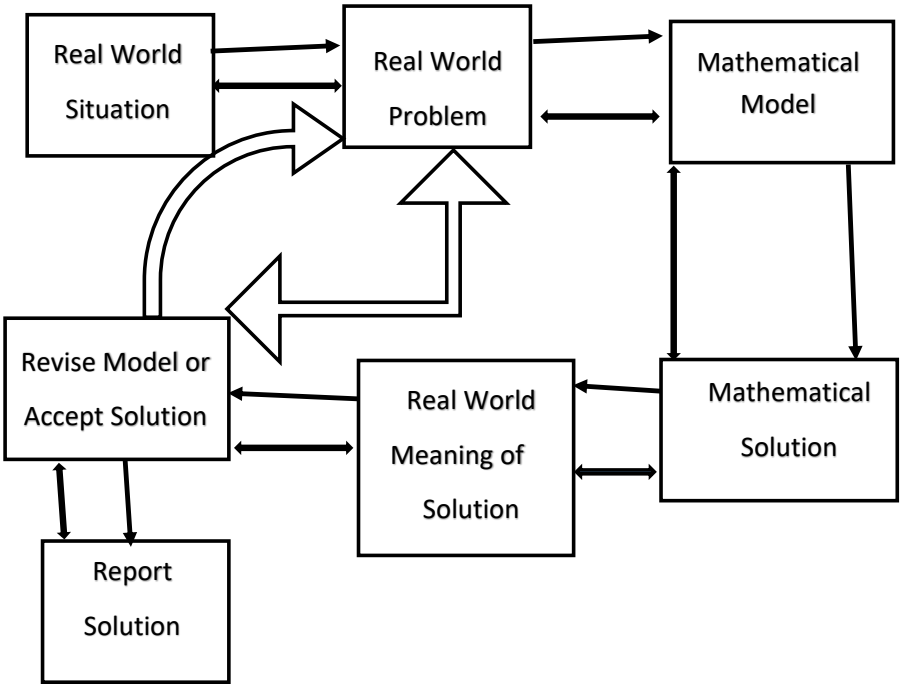


Figure 1.2: Process of constructing an appropriate mathematical model.

given situation, and these results depend on the form of the governing equations used. It fosters scientific understanding through the qualitative and quantitative expression of the current knowledge of a system (Dym, 2004; J. D. Murray, 2001). In this dissertation, mathematical models are based on ordinary differential equations with a definite number of variables and parameters.

Types of Models

The term *model* frequently refers to a pictorial representation of real-life situations in everyday conversation. Models are designed according to specific situations and are generally standard in nature. Some models are physical, while others are theoretical, biological, mathematical, sta-

tistical, or logical, as defined by Allman, Allman, and Rhodes (2004); Banerjee (2021); N. Britton (2005).

1. **Physical Models:** These models are intended for study, display, experimentation, or evaluation. For example, a map or globe is a physical model of the Earth. It is a miniature representation of a real object.

2. **Theoretical Models:** These models form the baseline for studying applications or practical situations. They are commonly associated with scientific theories from various fields and are used to explain observed phenomena by generating hypothetical or conceptual mechanisms.

3. **Logical Models:** Logical models are abstract in nature and serve as concrete representations of abstract systems. They are constructed from undefined statements, definitions, axioms, and theorems within a mathematical framework.

4. **Mathematical Models:** Mathematical models are closely associated with theoretical models and are constructed using logical structures. They attempt to reveal facts and aspects of reality through mathematical theories and solution techniques based on defined assumptions. Chapter 2 discusses four types of mathematical models.

Philosophical Background of the Proposed Models

Modeling with Pranayama

Pranayama, a yogic practice that emphasizes controlled breathing, can influence physiological parameters such as oxygen intake and immune response. Modeling with Pranayama involves defining variables and parameters that incorporate these physiological effects into disease transmission dynamics to evaluate its potential impact on susceptibility, infectivity and recovery rates.

The term *yoga* is derived from the Sanskrit root *"yuj"*, which means contemplation (*Samadhi*, trance) and is characterized by the mind pervading all its planes. It is a psycho-somatic-spiritual discipline aimed at achieving union and harmony between the mind (*Purusha*) and the body (*Prakriti*), and ultimately the union of individual consciousness with universal consciousness. It represents a mind–body connection that involves relaxation, meditation, and a set of physical exercises performed in synchrony with breathing. Yoga serves as a means of attaining physical, mental, social, and spiritual well-being for practitioners. This can be achieved through the systematic and disciplined practice of *Ashtanga* (eight-limbed) Yoga, as described by the sage Patanjali. Patanjali identifies eight components (limbs) of Yoga (Maehle, 2007; Miller et al., 1996; Vivekananda, 2019):

- *Yama* (abstinences),

- *Niyama* (observances),
- *Asana* (postures),
- *Pranayama* (breath control),
- *Pratyahara* (withdrawal of the senses),
- *Dharana* (concentration),
- *Dhyana* (meditation), and
- *Samadhi* (absorption).

The first two limbs of Ashtanga Yoga, yama and niyama, constitute the ethical code and personal discipline essential for the development of moral, spiritual, and social well-being. The third and fourth limbs, asana and pranayama, contribute to physical development and the improvement of physiological functions. The fifth and sixth limbs, pratyahara and dharana, focus on controlling the senses and cultivating a calm, alert, and one-pointed mind. The final two limbs, dhyana and samadhi, lead to inner peace, ecstasy, a higher level of consciousness, and the ultimate union of individual mindfulness with universal consciousness, culminating in the realization of the power of the universe (Eggleston, 2015). The result is the unfolding of a unique spiritual personality that benefits all of humanity. Yoga fosters the development of an integrated and holistic personality.

Prana and Pranayama are both philosophical concepts and practical disciplines. The fourth limb of Ashtanga Yoga, Pranayama, is an

exact science that regulates breath and controls Prana by temporarily restraining inhalation and exhalation. A healthy life may be considered a by-product of practicing yogic techniques, as yoga practitioners have been observed to be physically and mentally healthier and better able to cope with stress than the general population. Yoga is widely practiced and globally accepted; hence, it can be effectively integrated as a health-promoting tool in society.

Both healthy individuals and patients may seek medical consultation regarding yoga. If medical professionals develop an interest in yoga and practice it themselves, it may bridge traditional yogic heritage with modern medical science. Documented scientific evidence strongly indicates that yoga possesses promotive, preventive, and curative potential. It can serve as an effective lifestyle adjunct to medical treatment, helping to reduce drug dosage and improve patients' quality of life. It should be emphasized that yoga is highly effective for the prevention and management of widespread health problems and health-related disorders. Modern medicine is particularly effective in controlling infections, performing surgeries, and managing diseases. However, it has limited efficacy in addressing infectious diseases caused by viruses, stress-related conditions, chronic degenerative disorders, age-related ailments, and lifestyle diseases — the prevailing challenges of modern society. Yoga has demonstrated remarkable benefits in such cases.

Our public health delivery system is often understaffed, underfunded, and burdened by severe economic strain. Knowledge of inex-

pensive, effective, and easily administrable yogic techniques can significantly aid in achieving the WHO's goal of ensuring "physical, mental, spiritual, and social health" for all. There are numerous enzymes in the human body, and yoga helps regulate them, strengthen immunity, and maintain balance among the three bodily elements: *vata*, *pitta*, and *kapha* (Ramdev, 2009; Sivananda, 2019). This model is mathematically described in detail in Chapter 3.

Modeling with Yoga Awareness

WHO's definition of health states that health is a state of physical, mental, and social well-being. Yoga follows the same principle. People practice yoga for health, harmony, and endorphin-rich happiness. Yoga awareness helps individuals live an easy and healthy life. It is said that yoga is not only *asana* and pranayama, but a way of life (Eggleston, 2015). Good health depends on a long-term commitment, and the foundation for that commitment is built on four important pillars of a healthy lifestyle: *Aahar* (food), *Vihar* (recreation), *Aachar* (routine), and *Vichar* (thoughts). These are collectively termed yoga awareness (also called *Yogacchara*) in this dissertation.

Aahar is the first pillar of Yoga awareness, which refers to food. It is said that "Annam Brahma," meaning food is Brahman (the Lord of creation). An old saying states, "As the food, so the mind; as the mind, so the man" (Maehle, 2007, 2012; Vivekananda, 2019). According to Ayurveda, food is categorized into three types: *Sattvika*, *Rajasika*, and

Tamasika.

Vihar is the second pillar of Yoga awareness, which refers to recreation. Its literal meaning is moving or walking. Stress is a common factor in modern life, and daily stressors vary widely. Recreation, relaxation, and spending time with oneself or with family help regenerate the body and mind. Recreation acts as a natural antidepressant: it relieves depression and anxiety, elevates mood, and fosters a sense of well-being. Engaging in active creative hobbies, such as gardening, painting, or playing musical instruments, stimulates the senses, releases pent-up emotions, and recharges the mind. Playing sports is another effective way to relax both body and mind. Relaxation is the cornerstone of a well-balanced personality, helping maintain a positive attitude and cultivate the art of living. Regular exercise and relaxation keep the body fit and prevent disorders (Eggleston, 2015).

The third pillar of Yoga awareness is Aachar (routine), which is essential for mental health. Regularity and sincerity are two major components of a good routine. Aachar incorporates all necessary tasks related to self-care, work, food, recreation, and sleep. Many difficulties in life can be mitigated by establishing proper habits and routines. As Dr. Abdul Kalam stated, “We cannot change our future, but we can change our habits, and our habits will surely change our future” (Kalam & Rajan, 2002). It also includes hygiene practices such as isolation, hand washing, and face masks.

Intellectual health depends on the quality of our thoughts. Vichar,

or thoughts, represent the fourth pillar of Yoga awareness and serve as food for the mind. Buddha taught that based on our thinking, we become the person we are: thinking weakness results in weakness, while thinking strength fosters strength. Positive thought processes can be cultivated through reading good books and scriptures, attending *Satsanga* (religious discourses), reciting mantras, observing rituals, recalling positive experiences, and maintaining a positive mindset in all situations (Raj, 2010; Ramdev, 2009; Vidyapeeth, 2015; Warah, 2022). The Yoga awareness model is described in detail in Chapters 4 and 5.

Modeling with Quarantine and Yogachara at Saturated Incidence Rate

There is no doubt that communicable diseases pose significant threats to populations, and the simple provision of health care is insufficient to control their spread. Over the past century, public health has developed a series of population-level strategies to mitigate the transmission of communicable diseases. The mode of transmission for most communicable diseases is well understood; therefore, population-based interventions, such as contact tracing and isolation, are frequently employed in situations such as tuberculosis. However, there are circumstances in which communicable diseases threaten populations despite these measures, and a broader public health strategy may be required. The susceptible–infectious–quarantine–susceptible ($SIQS$) model is designed to address such situations by introducing a quarantine (Q) compartment to the existing SIS model, with Yogachara incorporated as a psycho-

logical preventive measure. Quarantine is an important component of communicable disease control, but it is by no means the sole method for controlling an outbreak (Brauer, Castillo-Chavez, Feng, et al., 2019; Rothman, Greenland, Lash, et al., 2008).

It is also important to distinguish clearly between quarantine and isolation. Quarantine refers to the separation of exposed individuals who are not yet symptomatic for a defined period (usually corresponding to the known incubation period of the suspected pathogen) to determine whether they will develop symptoms. Quarantine achieves two main objectives. First, it interrupts the chain of transmission, since individuals who are not circulating socially are less likely to infect others. Second, it allows individuals under surveillance to be identified and directed toward appropriate care if they become symptomatic. This is particularly critical for diseases with pre-symptomatic viral shedding. Isolation, on the other hand, involves separating symptomatic individuals from the general population (Ali, Shah, Imran, & Khan, 2020; Sánchez, 2021).

Mathematical Modeling of Hyper-Susceptibility in a Population

A hyper-susceptible group is defined by characteristics that increase the probability that its members are more susceptible than others to a particular disease. These characteristics are known as risk factors and may arise from both genetic and life-history-related factors. Genetic factors are inherited traits that determine the fundamental biochemical machinery with which an individual can respond to environmental insults. These

factors include sex, the capacity for enzymatic detoxification and elimination of harmful chemicals, and immunologic competence. In addition, certain developmental and metabolic abnormalities, as well as predispositions to specific diseases, can be inherited. Life-history factors are nongenetic forces that act over time to modify the body's ability to cope with environmental insults. These factors include age, illness, nutritional status, practice of Yoga, drug use, socioeconomic status, behavioral traits, *Yoga awareness*, and occupational or environmental exposures to harmful agents (Bingham, 1986).

Immunologic, metabolic, and other genetic factors governing hypersusceptibility are not static but change throughout a person's life (Khalsa, Cohen, McCall, & Telles, 2016; Raj, 2010). Yoga practice supports the development of a metabolic detoxification system, which may reduce the population's susceptibility to infectious diseases. Many infectious diseases also enhance immunity against reinfection. Conversely, harmful behavioral traits such as smoking and habitual use of alcohol or other drugs increase susceptibility to infectious agents and reduce the body's tolerance to disease. In underdeveloped nations, nutrition is a critical determinant of hyper-susceptibility. Insufficient intake of key nutrients, including vitamins C and E, magnesium, riboflavin, and protein, can significantly increase vulnerability.

Well-being is a multifaceted concept in the context of human welfare. Eastern philosophy adopts a holistic approach to well-being, situating individuals within a natural context. Maharishi Patanjali's *Yoga Su-*

tras provide a comprehensive, multilevel toolkit of principles and practices leading to union with pure consciousness. The eight limbs of the Yoga Sutras promote well-being at both the individual and societal levels.

Historical examples illustrate the use of these practices for collective well-being: Mahatma Gandhi promoted moral well-being through the practice of Yamas and Niyamas; Swami Ramdev has enhanced the mental and physical well-being of millions using Asanas and Pranayama; and Maharishi Mahesh Yogi has relieved stress and anxiety for millions, particularly in the Western world, through Dhyana and Samadhi, fostering a taste of blissful living. This dissertation explores how existing and novel tools based on the *Yoga Sutras* can unite humanity and address complex challenges to promote universal flourishing (Maheshwari & Werd, 2020). A mathematical model for the dynamics of a hypersusceptible population relative to a susceptible population, including preventive measures such as Yogachara, is developed in Chapter 6.

Motivation and Challenges

Mathematical modeling has some of the most exciting applications in the field of communicable diseases. It is an interdisciplinary approach applied across various domains of research and can be viewed as the process of describing phenomena in terms of mathematical equations. Mathematical modeling is a central methodology in bio-mathematical research, offering advanced practical applications, numerous interesting

insights, and productive inferences (Dym, 2004; J. Murray, 2004).

Motivated by the significant applications of mathematical modeling, the Eastern philosophy of yoga, and the recent COVID-19 pandemic, this study develops new models by incorporating a yoga Sadhaka compartment into the classical SIR model and a modified SIR model with yoga awareness. Furthermore, a susceptible–infectious–quarantine–susceptible ($SIQS$) model is designed to address disease control strategies by integrating Yogachara as a preventive measure and introducing a quarantine (Q) compartment with a saturated incidence rate into the existing SIS framework. It has also been observed that susceptibility varies among individuals, depending on factors such as Aahar, Vihar, Aachar, and Vichar. Not all individuals have the same probability of becoming infected. genetic, behavioral, and cultural factors, as well as regularity in yoga practice, influence the likelihood of becoming infectious. Consequently, the dynamics of a hyper-susceptible population relative to a susceptible population is considered as one of the models in this study (Khalsa et al., 2016; Raj, 2010).

Mathematical modeling is crucial for constructing, representing, and interpreting real-life problems or complex situations in various contexts. These models are applied to analyze problems, interpret numerical data, explore systems, and make predictions. Through modeling activities, researchers are expected to construct models and describe the factors influencing the phenomena under study. With emerging opportunities, biomathematical modeling has demonstrated significant appli-

cations. It plays an efficient and inspiring role in research and must be planned and organized commensurate with the importance of its contribution. Modeling directs research toward new opportunities in science, technology, Eastern philosophy, or other domains. Mathematical modeling of communicable diseases enables quantitative characterization of infectious processes. In this sense, the present research not only supports and advances the study of communicable diseases but also opens avenues for integrating Eastern philosophy with modern scientific approaches, potentially generating new insights, inventions, and discoveries to benefit humanity.

Unlike modeling of physical systems, where equations can accurately describe reality, mathematical modeling in biological problems, such as epidemiology, is more complex. This complexity arises from the evolutionary nature of living systems and the multiple spatial and temporal scales on which biological processes occur. The multi-scale nature of biological systems makes the mathematical investigation of disease transmission dynamics more challenging, requiring sophisticated approaches to capture the real-world behavior of infectious diseases and the effectiveness of control strategies.

Review of Literature

The purpose of the review of literature is to evaluate the performance of existing theories and identify their limitations. It aims to determine what has already been accomplished in the field and to what extent the topic

has been investigated. The main objectives of this chapter are:

- Identify the methodologies employed in previous research.
- Examine the techniques used for solving nonlinear ordinary differential equations.
- Highlight the core research areas within the field.
- Identify gaps in existing knowledge and determine potential contributions of the current research.
- Review prior theories and technologies to improve existing models, to develop new ones, or to propose novel methodologies for model formulation in this domain.

The relevant literature is organized into thematic sections.

Mathematical Modeling on Communicable Diseases

The general study of the causes of epidemic outbreaks and the spread of epidemics has led to the development of a variety of mathematical models, such as susceptible-infected susceptible (SIS), susceptible–infected–recovered (SIR), susceptible–infected–recovered–susceptible ($SIRS$),susceptible–aware infected–recovered ($SAIR$), susceptible–exposed–infected–recovered ($SEIR$). These models are highly useful in describing the dynamics of infectious diseases and in predicting how a disease can be contained or eradicated from a given geographical region. In a deterministic model, every set of variable values is uniquely determined by the parameters in the model

and the initial state of the variables. Deterministic compartmental mathematical models are valuable tools in epidemiology for investigating transmission characteristics and predicting future outbreaks at the community level. Various types of deterministic compartmental models have been developed by researchers, scientists, and mathematicians (Allman et al., 2004; N. Britton, 2005; Martcheva, 2015; Zill, 2012).

In compartmental disease transmission models, the population is divided into compartments, and assumptions are made regarding the nature and rate of transfer from one compartment to another. For example, in an SIR model, the population is divided into three classes: $S = S(t)$ denotes the number of susceptible individuals who have not yet been infected, $I = I(t)$ denotes the number of infected individuals, assumed to be infectious and capable of spreading the disease to susceptibles, and $R = R(t)$ denotes the number of individuals who have been infected and removed from the possibility of reinfection or spreading the infection. For many diseases, infectives return to the susceptible class upon recovery, as these diseases confer no immunity against reinfection. Such models are appropriate for most diseases transmitted by bacterial and for most sexually transmitted infections, but not diseases such as AIDS, from which there is no recovery. The terminology SIS is used to describe a disease with no immunity against reinfection, indicating that individuals move from the susceptible class to the infective class and then back to the susceptible class. In contrast, diseases caused by viruses are typically modeled as SIR type. In addition to the basic distinction between

diseases for which recovery confers immunity and diseases for which recovered individuals are susceptible, there are intermediate possibilities, such as temporary immunity, which are represented by $SIRS$ models. More complex compartmental models, such as $SEIR$ and $SEIS$, incorporate an exposed period between infection and becoming infectious (Allman et al., 2004; N. Britton, 2005; Martcheva, 2015; Zill, 2012).

Epidemiology, defined as the study of the distribution and determinants of health-related states in populations, has developed from early descriptive observations to a modern, evidence-based, and interdisciplinary science (Susser, 1973). Its origins lie in ancient Greece, where Hippocrates emphasized the influence of environmental and lifestyle factors on disease, introducing the concepts of epidemic and endemic (Hippocrates, 1923), while Galen highlighted population-level observations and the role of environmental conditions (Galen, 1968). During the medieval and early modern periods, public health measures such as quarantine were used to control plague outbreaks (Cohn, 2002), and thinkers like Fracastoro, Graunt, and Ramazzini contributed early ideas on contagion, mortality analysis, and occupational health (Fracastoro, 1546; Graunt, 1662; Ramazzini, 1964). Some historical perspectives on epidemic analysis of communicable diseases are as follows: (Martcheva, 2015):

Historical Perspectives on Epidemic Modeling

The history of epidemic modeling reflects the evolution of quantitative reasoning, empirical observation, and computational advances in understanding infectious disease dynamics. From early statistical records to contemporary network-based simulations, the discipline has progressively integrated mathematical rigor, behavioral insights, and preventive paradigms.

Early Statistical Foundations (17th–18th Century). Graunt (1662) introduced quantitative analysis in public health through the *Bills of Mortality*, marking the beginning of demographic and epidemiological statistics. Building on this foundation, Bernoulli (1760) developed one of the earliest mathematical models of smallpox inoculation, demonstrating the benefits of preventive interventions using differential calculus. These contributions established the statistical and analytical basis for later developments in epidemiological modeling.

Development of Vital Statistics and Empirical Epidemiology (19th Century). Farr (1838) advanced the systematic collection and interpretation of mortality and morbidity data, establishing quantitative relationships among environmental conditions, population density, and disease occurrence. His work emphasized that epidemic phenomena followed predictable, law-like patterns, thereby preparing the conceptual groundwork for formal mathematical modeling of disease spread.

Birth of Modern Mathematical Epidemiology (Early 20th Century). Kermack and McKendrick (1927) developed the classical *susceptible–infectious–recovered (SIR)* model, a deterministic nonlinear differential equation framework that explained epidemic waves and threshold behavior. During this period, the concept of the basic reproduction number (R_0) also emerged as a main epidemiological metric for determining the epidemic potential of an infectious agent and identifying conditions for its control.

Model Extensions and Theoretical Refinements (Mid 20th Century). Subsequent developments extended the classical epidemic models to capture biological and demographic complexities. Compartmental models such as SIS and $SEIR$ incorporated recurrent and latent infections (H. W. Hethcote, 2000), while Bailey (1957) introduced stochastic formulations to account for random transmission processes. R. M. Anderson and May (1991) further integrated demographic structure, spatial heterogeneity, and immunity loss, solidifying the theoretical foundations of modern population level epidemiology.

Computational and Network-Based Modeling (Late 20th–21st Century). Advances in computational power and data availability enabled the emergence of agent-based and network-based models that captured individual heterogeneity, spatial structure, and complex contact patterns (Keeling & Eames, 2005; Pastor-Satorras & Vespignani, 2001). In parallel, the application of optimal control theory facilitated rigorous evaluation of vaccination, quarantine, and treatment strategies (Lenhart & Workman, 2007). These

approaches bridged classical theory with modern computational epidemiology.

Behavioral, Socio-Cultural, and Awareness Models (Contemporary Era). Recent research highlights the importance of behavioral and socio-cultural dynamics in shaping epidemic outcomes. Models increasingly incorporate awareness diffusion, misinformation, and adaptive social responses (Funk, Gilad, Watkins, & Jansen, 2010). Interdisciplinary frameworks have emerged that integrate lifestyle and cultural health practices — including yoga, pranayama, and meditation—as modifiers of susceptibility, recovery, and transmission parameters. These developments mark a paradigm shift from purely biological approaches to more holistic bio-psycho-social models of epidemic dynamics.

Recent Epidemiological Models (2020–2024). The COVID-19 pandemic catalyzed significant methodological advances in spatio-temporal and network-based epidemic modeling; means studying how diseases spread over time, across places, and through connections between people. Some authors proposed a multi-agent simulation on small-world networks to represent neighborhood-level and long-distance social interactions. Xue, Liu, Chen, and Liu (2024) developed a network-based meta-population model incorporating population movement and asymptomatic transmission, enhancing prediction accuracy. They applied a Generalised Network Autoregressive (GNAR) model to capture temporal and spatial dependencies across Irish counties under varying mobility restrictions. and in-

vestigated co-evolving multiplex networks linking information diffusion and disease spread, revealing nonlinear feedback between awareness and transmission. Additionally, Z. Liu, Wan, Prakash, Lau, and Jin (2024) reviewed applications of graph neural networks (GNNs) for epidemic forecasting, demonstrating improved spatial-temporal predictions through integration with compartmental models.

The study of mathematical modeling in epidemiology has a rich historical foundation that dates back to the 18th century. Daniel Bernoulli was one of the pioneers, using probabilistic reasoning in 1760 to demonstrate the benefits of smallpox inoculation, while Thomas Malthus applied mathematical concepts to population growth and carrying capacity, influencing later epidemiological models. In the 20th century, William O. Kermack and A.G. McKendrick developed the SIR model, which became a cornerstone for understanding infectious disease spread, complemented by William Farr's statistical analyses of mortality data that helped uncover the patterns of epidemic outbreaks. Modern approaches have evolved to include complex computational and multiscale models, integrating biological, environmental, and social factors to more accurately capture disease dynamics. Recently, there is growing interest in incorporating lifestyle interventions such as yoga into epidemiological models. Yoga has been shown to influence immune function, stress response, and overall well-being, which could affect disease susceptibility and progression. Integrating yoga into mathematical models could involve modifying parameters to reflect its effects on immunity and stress,

developing new compartments to represent yoga practitioners, or using stochastic approaches to capture variability. This interdisciplinary approach provides a holistic perspective, bridging traditional epidemiological modeling with preventive and health-promoting lifestyle interventions, thereby offering new insights into disease transmission dynamics.

Throughout history, human populations have been repeatedly affected by infectious diseases such as plague, influenza, and yellow fever. Limited understanding of epidemic dynamics has historically resulted in significant mortality. Over time, mathematical modeling has become a fundamental tool for analyzing and predicting disease transmission, evaluating control strategies, and supporting public health decision-making (Foppa, 2016; Martcheva, 2015; J. D. Murray, 2001). Owing to the complexity of epidemic processes, computer simulations are often employed to analyze interactions among model parameters and assess intervention effectiveness. Vaccination remains one of the most effective preventive measures against infectious diseases. However, for newly emerging pathogens, delays in vaccine development and distribution often coincide with ongoing outbreaks, as seen during the H1N1 (2009), Ebola (2013–2016), and COVID-19 (2019–2022) pandemics (Garira, 2013; Lin, Muthuraman, & Lawley, 2010; Muqbel & Röst, n.d.).

In such cases, non-pharmaceutical interventions—such as hygiene, awareness programs, and behavioral modifications—play crucial roles in mitigating transmission. Recent attention has also turned toward lifestyle-based strategies, including yoga, as complementary preventive measures.

Funk, Gilad, Watkins, and Jansen (2009) introduced a mathematical model linking population awareness to epidemiological dynamics, demonstrating that increased awareness can reduce outbreak size. Similarly, Q. Wu, Fu, Small, and Xu (2012) modeled awareness effects on scale-free networks, identifying that local and contact awareness elevate epidemic thresholds, while global awareness reduces disease prevalence. Samanta, Rana, Sharma, Misra, and Chattopadhyay (2013) examined media-driven awareness programs and showed that program intensity can significantly alter system dynamics, occasionally inducing sustained oscillations that complicate control efforts.

Building upon this, Kabir, Kuga, and Tanimoto (2019) proposed an $SIR - UA$ (Susceptible–Infected–Recovered with Unaware–Aware states) model, illustrating how awareness decreases susceptibility and infectivity while enhancing recovery. Canabarro, Linares, et al. (2020) developed an age-structured $SIRD$-like model for $COVID - 19$ in Brazil, evaluating the effects of school closures, social distancing, and home quarantine on transmission. Likewise, Musa et al. (2021) modeled $COVID - 19$ dynamics in Nigeria, incorporating awareness and hospitalization strategies. Their findings underscored that inadequate awareness exacerbates outbreaks, whereas timely public information and hospital interventions are essential for epidemic control.

Collectively, these studies highlight the pivotal role of behavioral and awareness factors in shaping epidemic trajectories. Integrating such socio-behavioral mechanisms and potentially holistic practices like yoga

into mathematical frameworks offers promising avenues for enhancing disease prevention and resilience in future epidemics.

Modeling Communicable Disease Dynamics with Yoga Pranayama

Prana, often described as the vital life energy, plays a central role in maintaining both physical health and mental equilibrium. Developing a balanced Prana at a deep level of awareness is essential for sustaining immunity and overall well-being. This concept extends beyond the breath itself to the subtle energy that underlies the body and mind, supporting the *chakras* and providing vitality to the physical form while extending into the energetic body (Maehle, 2007, 2012; Miller et al., 1996; Vivekananda, 2019). Particularly in the context of modern pandemics, where protective measures such as mask-wearing are employed, regular practice of Pranayama can enhance circulation and maintain the flow of both outer and inner Prana. Alternate nostril breathing (*nadi shodhan*), which balances airflow between the right and left nostrils (*pingala* and *ida*), has been associated with energetic balance, strengthened immunity, and mental calmness. Daily practice, especially in the morning and evening, is recommended to restore disrupted energies and support meditative states.

From a physiological perspective, the onset of many infectious diseases, ranging from the common cold to influenza, has been linked to a disruption in peripheral Pranic circulation. Reduced flow along the body's surface may allow pathogenic agents to penetrate, while environ-

mental factors such as heat, dampness, wind, or polluted air can further compromise Prana and reduce immune resilience (Eggleston, 2015; Haridwar, n.d.; Sivananda, 2017). Weak Prana can also affect digestive and metabolic functions, sleep quality, and higher cognitive and emotional regulation. Pranayama practices strengthen peripheral and central Pranic circulation, reinforcing the aura and the body's energetic field. Continuous, deep Pranic flow prevents the ingress of external pathogens, but its effectiveness depends on unobstructed vertical Prana along the spine, from the root to the crown chakra. Blockages or imbalances in spinal Prana can weaken peripheral circulation, while insufficient upward movement of consciousness may lead to excessive downward or outward Prana flow (*apana vayu*), resulting in both physical and mental disharmony. These insights highlight the potential integration of pranayama into communicable disease prevention frameworks, particularly as a complementary approach that supports immune function, mental resilience, and overall physiological equilibrium.

Raimundo, Yang, and Engel (2007) distinguished between infections in immunologically naive versus vaccinated individuals, highlighting that pandemic countermeasures include vaccination, antiviral therapy, and immune modulation. Vaccines are often strain-specific and antiviral therapies are costly, prompting interest in low-risk, cost-effective complementary interventions such as yoga, pranayama, and Ayurveda (Malik & Sharma, 2020).

Mathematical modeling of breathing patterns using unsteady-state

differential equations demonstrated that oxygen transport to the heart, lungs, and tissues is influenced by pranayama, reflecting the functional significance of the pranic body (Haridwar, n.d.). Empirical studies support these findings: Manaspure, Fadia, and Damodara Gowda (2011) reported improvements in lung capacity and ventilatory function, while Ross, Friedmann, Bevans, Thomas, et al. (2012) observed enhanced overall health through yoga practices, including asanas and pranayama. Sivaramakrishnan et al. (2019) synthesized evidence showing significant improvements in physical function and quality of life among older adults.

Community-focused interventions have also been effective. Nagarathna, Nagendra, and Majumdar (2020) developed integrated Yoga modules for children, adults, and the elderly, demonstrating preventive benefits. Continuous Yoga practice reduces BMI and body fat while increasing muscle mass in overweight participants (Na Nongkhai, Yamprasert, & Punsawad, 2021). Clinical studies indicate that Yoga enhances immunity and psychological health in patients with HIV and supports recovery during anti-tuberculosis treatment by improving lung capacity, reducing symptoms, and enhancing antioxidant status (Umesh, Ramakrishna, Jasti, Bhargav, & Varambally, 2021). Yoga is increasingly recommended in national and global guidelines for health promotion, though systematic evaluation in older adults without specific conditions is ongoing (Bhardwaj, Pathania, Pathania, Rathaur, et al., 2021; Maehle, 2012; Omkar, Mour, & Das, 2011).

Pranayama, a yogic practice of controlled breathing, has been shown

to improve overall health by enhancing respiratory function, boosting immune response, and reducing stress. It increases lung capacity, optimizes oxygenation, and strengthens respiratory muscles, which are critical in combating respiratory infections. By stimulating the parasympathetic nervous system, pranayama helps lower stress hormones and inflammation, supporting immune resilience. It promotes mental well-being, improves sleep quality, and enhances energy levels. These physiological and psychological benefits make pranayama a promising non-pharmaceutical intervention for epidemic prevention and health promotion, providing a potential strategy to reduce susceptibility and severity.

Modeling Communicable Disease Dynamics with Yoga Awareness

Yoga awareness encompasses food (ahara), physical activity (vihar), good conduct (achar), and thoughts or behavior (vichar)—collectively known as AVAV—which form the pillars of a healthy lifestyle. These practices can reduce disease transmission by promoting behavioral changes, including the use of face masks, hand hygiene, sanitizers, isolation, and quarantine (Maehle, 2012; Sivananda, 2017). Pranayama, in particular, enhances awareness of disease risk, contributing to containment efforts (Maehle, 2007)

Mathematical and computational models have increasingly incorporated awareness as a dynamic factor in disease transmission. Funk, Gilad, and Jansen (2010) showed that awareness arising in social networks can reduce susceptibility, infectivity, and infectious period through

self-imposed behavioral changes. Similarly, Kiss, Cassell, Recker, and Simon (2010) introduced media-influenced compartments, demonstrating that simultaneous spread of disease and awareness can create multiple equilibria and support optimal control strategies (Q. Wang, Zhao, Huang, Yang, & Wu, 2015). Network-based and mean-field models indicate that firsthand awareness, social distancing, and hygiene measures can lower incidence and prevent epidemics (Maharaj & Kleczkowski, 2012; Sun, Yang, Arino, & Khan, 2011). Media-driven interventions have been shown to influence epidemic dynamics significantly, with the timing and intensity of awareness programs affecting system stability and oscillations (G. Agaba, Kyrychko, & Blyuss, 2017a; M. Liu, Chang, & Zuo, 2016; Samanta et al., 2013).

Empirical evidence supports the physiological and preventive benefits of Yoga. Sivaramakrishnan et al. (2019) found improvements in physical function and health-related quality of life among older adults. During the COVID-19 pandemic, Yoga, meditation, and Yogic bio-cleansing practices enhanced immunity, mental health, and management of respiratory and non-communicable diseases (Sharma, 2020). Media-based interventions and awareness campaigns also effectively reduced infection peaks and final epidemic size (Imran, Wu, Zhao, Beşe, & Khan, 2021; Kim, Barber, & Lee, 2020). Deterministic and stochastic models provide complementary insights into transmission dynamics and intervention evaluation (Costa, Pires, Resque, & Almeida, 2021).

Kim et al. (2020) examined mass media's role in communicable

disease control. They developed a model with two media effect terms: a theory-based term dependent on infected numbers and a data-based term using real-world coverage from the 2009 H1N1 influenza outbreak. Their simulations indicated that increased media coverage reduces peak infection and final epidemic size. Similarly, Imran et al. (2021) used an SIR model with contact ratio considerations during COVID-19 and found that awareness measures, such as handwashing and physical distancing, effectively reduced infection risk.

Clinical studies indicate that continuous Yoga practice improves body composition, muscle mass, metabolism, and immune function (Na Nongkhai et al., 2021; Yeun & Kim, 2021). Holistic approaches, including Ayurvedic interventions, have shown potential in managing infectious diseases, such as COVID-19 in pregnant women, highlighting the value of integrated preventive strategies (Pandey, Kajaria, Sharma, & Kadam, 2022). Collectively, these findings underscore the role of Yoga awareness in modifying behavior, enhancing immunity, and reducing the spread of communicable diseases.

Mathematical Modeling with Quarantine and Yogachara at Saturated Incidence Rate

In the $SIQS$ model, infection does not confer immunity. Some susceptible individuals become infected, and while some remain in the infectious class (I) for their entire infectious period before returning to the susceptible class (S), others move into a quarantine class (Q). H. Het-

hcote, Zhien, and Shengbing (2002) analyzed the effects of quarantine in epidemic models using simple mass-action, standard, and quarantine-adjusted incidence rates. Motivated by the work of H. Hethcote et al. (2002); Joshi (2020); Q. Zhang and Zhou (2019), an $SIQS$ model with quarantine and a saturated incidence rate given by

$$g(S, I) = \frac{\beta SI}{1 + \alpha S}$$

is considered. This incidence rate provides a more realistic representation of disease transmission when the infection level is high and has been widely applied in epidemiological modeling. Several authors have analyzed epidemic models with saturated incidence and treatment, examining endemic and disease-free equilibria through differential stability and qualitative theory (Ajbar, Alqahtani, & Boumaza, 2021; Algehyne & ud Din, 2021; X. Liu & Yang, 2012; Razzaq, Khan, Faizan, Ara, & Ullah, 2021; Shah, Abdeljawad, & Din, 2022; Shah, Din, Deebani, Kumam, & Shah, 2021).

Various epidemic models such as SIS, SIR, $SIQS$, and $SIQRS$ incorporate saturated incidence rates and treatment terms, but only the $SIQS$ model is discussed in Chapter Five. Using differential stability and qualitative theory, these studies identified complete configurations of endemic and disease-free equilibria (R. Anderson & May, 1978; J. Wang & Jiang, 2014; W. Wang, Liu, & Zhao, 2013).

Several quarantine models with different incidence structures were

developed to study the effect of quarantine on disease transmission (Alexander & Moghadas, 2004; Nuno, Castillo-Chavez, Feng, & Martcheva, 2008; L.-I. Wu & Feng, 2000). These studies examined bilinear, nonlinear, standard, and non-monotone incidence forms to provide qualitative analyses of disease dynamics.

J. Zhang, Jia, Song, et al. (2014) examined the dynamics of an $SEIR$ model with a saturated incidence rate and treatment. They derived the basic reproduction number, determined threshold conditions for equilibria, and established sufficient conditions for backward bifurcation. Local stability was analyzed via the Routh–Hurwitz criterion, and global stability through the autonomous convergence theorem, supported by numerical simulations.

Q. Zhang and Zhou (2019) investigated a stochastic $SIQR$ model with saturated incidence, proving the existence and uniqueness of positive solutions using a Lyapunov function and verifying model dynamics with numerical simulations. Similarly, Ali et al. (2020) proposed a deterministic COVID-19 model incorporating asymptomatic, quarantine, and isolation compartments. Their analysis showed that the reproduction number is most sensitive to quarantine and isolation rates, highlighting the importance of maintaining these measures, especially during early outbreak stages.

Reno et al. (2020) developed an SIV model with susceptible, infected, and vaccinated classes using a generalized nonlinear incidence rate. Their Poincaré index analysis showed that nonlinear incidence can

generate bistability and periodicity without seasonal forcing, and that the basic reproduction number is independent of the incidence form.

Flaxman et al. (2020) studied the effects of major interventions in eleven European countries from February to May 2020. Their model estimated transmission backward from observed deaths, accounting for delays between infection and death, and used partial pooling of country data to enhance estimation accuracy. Assuming fixed epidemiological parameters and immediate behavioral response to interventions, they found $R_t < 1$ for all countries, indicating control of the epidemic. Their findings demonstrated that major non-pharmaceutical interventions, particularly lockdowns, effectively reduced transmission and should remain central to epidemic management.

X.-B. Zhang and Zhang (2021) proposed stochastic and deterministic SIS models with isolation and varying population size. In the deterministic case, $R_0 < 1$ implies a globally stable disease-free equilibrium, whereas $R_0 > 1$ leads to an endemic equilibrium. They identified a critical isolation rate δ^* above which the disease is eliminated, and showed that stochastic perturbations of the transmission rate can suppress spread, with the disease disappearing when the perturbation intensity σ^* exceeds a critical threshold.

Razzaq et al. (2021) presented a fractional-order compartmental model with susceptible, social distancing, exposed, quarantined, infected, isolated, and recovered groups. Using proportional fractional derivatives, they analyzed equilibrium states and reproduction numbers with memory

effects. The saturated incidence rate explained differences in transmissibility due to behavioral variation, and results were validated through fractional fourth-order Runge–Kutta simulations.

Integrating quarantine measures with healthy lifestyle practices offers a comprehensive, non-pharmaceutical approach to disease prevention. While quarantine effectively reduces disease transmission by limiting exposure, it can inadvertently disrupt daily routines, potentially leading to negative health outcomes such as increased sedentary behavior and poor dietary habits. Combining quarantine with the promotion of healthy lifestyles—such as regular physical activity, balanced nutrition, adequate sleep, and stress management—can mitigate these adverse effects and enhance overall well-being. Evidence supports the benefits of this integrated approach. For instance, a study by Mattioli, Sciomer, Cocchi, Maffei, and Gallina (2020) emphasized the importance of encouraging healthy diets and physical activity during quarantine to counteract potential negative impacts on cardiovascular health. Similarly, research by Doraiswamy, Cheema, Campagna, Mamtani, and Cheema (2024) highlighted that while COVID-19 lockdowns led to negative lifestyle changes for many, some individuals adopted healthier behaviors, suggesting that promoting healthy lifestyles during quarantine can lead to positive health outcomes. In conclusion, combining quarantine measures with the promotion of healthy lifestyles not only helps in controlling disease spread but also supports individual health, making it a valuable strategy in non-pharmaceutical disease prevention.

Dynamics of Susceptible and Hyper-Susceptible Populations in an Epidemic Model

People in a community do not possess the same immune system nor share identical traits that determine their likelihood of contracting an infection. The incidence of infection may vary according to age, sex, race, social practices, hygiene, vaccination status, yoga practice, exercise, and individual immune status. Unless otherwise specified, all individuals in a community are considered susceptibles non-infected persons assumed to have normal defense mechanisms (immune response) capable of combating infectious microorganisms. However, in many cases, individuals differ in their level of immunity, which may be suppressed to lower levels, rendering them more prone to infection. Such individuals can be categorized into a separate class termed hyper-susceptibles. Hyper-susceptibles are individuals who, for various reasons, have lower immunity levels compared with the majority of the population. This group includes very old or debilitated persons, diabetics, cancer patients, organ transplant recipients, hospitalized patients, and those with immune deficiency disorders (e.g., AIDS). The primary cause of hyper-susceptibility in this group is a reduced immune capacity to combat invading microorganisms; such individuals are often described as immunosuppressed or immunocompromised. Once infected, hyper-susceptible individuals exhibit diminished responses to vaccination or treatment and tend to remain infected for longer periods than those with normal immunity. The im-

mune system continuously works to maintain health and protect against bacteria, viruses, and other pathogens; however, it may sometimes overreact, causing hypersensitivity reactions that can be harmful or even fatal. These reactions result from exposure to foreign antigens on or in the body (Busenberg & Van den Driessche, 1990).

Hyman and Li (2005) formulated compartmental differential susceptibility (DS) susceptible-infective-removed (SIR) models by dividing the susceptible population into multiple subgroups according to individual susceptibility. They analyzed the impact of disease-induced mortality under scenarios where the number of contacts per individual was either constant or proportional to the total population. The researchers derived explicit formulas for the reproductive number of infections for each model and examined the local stability of the infection-free equilibrium. Using qualitative analysis and appropriately chosen Lyapunov functions, they demonstrated that the infection-free equilibrium is globally asymptotically stable. They further showed that if the reproductive number exceeds one, a unique endemic equilibrium exists, which is locally asymptotically stable for models without disease-induced mortality and those with contact numbers proportional to the population. Conditions for stability of the endemic equilibrium were also provided for other cases.

Hyman and Li (2009) extended this work by developing epidemic models with differential susceptibilities and staged disease progression, based on systems of ordinary differential equations. In these models,

the susceptibility of susceptible individuals varies, and infective individuals progress through stages of differing infectiousness. Contact rates were considered either proportional to the total population or constant, yielding bilinear or standard incidence forms. Explicit formulas for the reproductive number were derived, and it was shown that the infection-free equilibrium is globally asymptotically stable if $R_0 < 1$ for bilinear incidence. They also analyzed endemic equilibria, demonstrating the existence of a unique endemic equilibrium for bilinear incidence and at least one endemic equilibrium for standard incidence when $R_0 > 1$.

Kuniya (2011) formulated an age-structured SIR model described by a system of partial differential equations (PDEs) and analyzed the global asymptotic stability of endemic equilibria when the basic reproduction number exceeded unity. A multi-group epidemic model was constructed and studied, with equilibria analyzed by discretizing the age variable and converting the PDE system to an ordinary differential equation (ODE) system. Using classical Lyapunov function methods and a graph-theoretic approach with maximum value functions, the study established that the global stability of each equilibrium is fully determined by R_0. The disease-free equilibrium is globally asymptotically stable if $R_0 < 1$, whereas a unique endemic equilibrium exists and is globally asymptotically stable if $R_0 > 1$.

Muroya, Kuniya, and Wang (2015) investigated a delayed multi-group SIS epidemic model with nonlinear incidence rates and patch structures, accounting for time delay and population exchange between groups.

Employing a Lyapunov functional approach, they demonstrated that the global stability of the model is determined by a threshold parameter R_0. The disease-free equilibrium is globally asymptotically stable if $R_0 < 1$, while an endemic equilibrium exists if $R_0 > 1$.

Greenhalgh, Galvani, and Medlock (2015) analyzed an SIR model with discrete age groups to understand disease transmission in a structured host population. The basic reproduction number was derived as a sharp threshold: if $R_0 < 1$, the disease-free equilibrium is globally stable; if $R_0 > 1$, the disease-free equilibrium is unstable, the model is uniformly persistent, and an endemic equilibrium exists. The global stability of the endemic equilibrium under $R_0 > 1$ was established under sufficient conditions. Their model was applied to measles data to evaluate vaccination strategies and demonstrated that traditional differential equation models, while useful for predicting disease trajectories, may fail to capture probabilistic events dominating low-prevalence dynamics.

Seidu, Bornaa, and Makinde (2020) classified hyper-susceptible individuals and developed a mathematical model to examine the impact of hyper-susceptibility on Ebola virus disease dynamics. The model analysis revealed that the disease-free equilibrium is globally stable whenever the basic reproduction number is less than unity. Forward bifurcation was observed, suggesting that eradication is possible by maintaining $R_0 < 1$. Disease spread was highly sensitive to contact rates, transmission probability, death rate, and hyper-susceptibility. Numerical simulations confirmed the analytical results.

Implications of the Review of Literature

A review of the literature provides a method for searching and synthesizing relevant problems, constructing a successful research path, and shaping and refining the research problem.

In this dissertation, systems of simultaneous ordinary differential equations are employed to model infectious diseases with specified initial conditions. The model incorporates the dynamics of communicable diseases under the influence of pranayama and yoga awareness. An $SIQS$ epidemic model with a saturated incidence rate and Yogachara has also been utilized. Additionally, a simple SIR dynamic model and its extended forms were applied to determine key parameters and the basic reproduction number. The effects of pranayama, yoga awareness, and quarantine, in conjunction with Yogachara, on disease dynamics have been studied, and various parameters were established. Furthermore, the disease dynamics of hyper-susceptible populations relative to the susceptible population were analyzed under the influence of yoga awareness. From the review of relevant literature, research gaps were identified, leading to the development of the conceptual framework presented below.

Research Gap

In recent years, much research on communicable diseases has focused on different modeling patterns, with combined approaches used to en-

hance the accuracy of predictions. Research attention in communicable diseases should first emphasize the application of existing knowledge. At the same time, the development of new tools for disease control must also be explored. Previous studies have primarily focused on the mathematical theory, which needs to be integrated with practical applications to advance the field of epidemiology. To improve the efficacy and effectiveness of disease control measures, social and economic health system research is essential. This research aims to strengthen health systems through empowerment and capacity building at national, institutional, and individual levels. The gaps identified in prior research during the modeling of communicable diseases are addressed in this study.

Mathematical modeling of transmission dynamics of communicable diseases incorporating vaccination, treatment, and media awareness as control strategies has been extensively studied. However, these models do not account for disease prevention or control strategies based on yoga, pranayama, and yoga awareness (Yogachara). The effect of pranayama and yoga awareness on disease transmission dynamics has not been explored previously. In this research, improved epidemic models incorporating the efficacy of yoga are introduced. By adding a *yoga sadhaka* compartment to the existing SIR model, a new $SYIRS$ model is formulated. A new yoga-awareness effect term, $\beta_1 = \beta e^{-cM(t)}$, has been incorporated into the transmission rate. Additionally, a nonlinear saturated incidence rate, $\frac{\beta SI}{1+\alpha S}$, with a psychological (yoga awareness) prevention rate α, is included.

Not all individuals in a population possess the same immunity, and preventive measures do not have uniform effectiveness. Therefore, the concept of hyper-susceptibility—individuals being more susceptible than the general population despite the same protective measures—has also been incorporated.

Conceptual Frame Work

After an extensive review of the literature, four models based on yoga strategies were developed. Details of these models, including the governing ordinary differential equations, are presented in Chapters 3 through 6. The expected relationships between the proposed variables and parameters are established. The conceptual framework illustrating the structure of the research is shown in Figure 1.3.

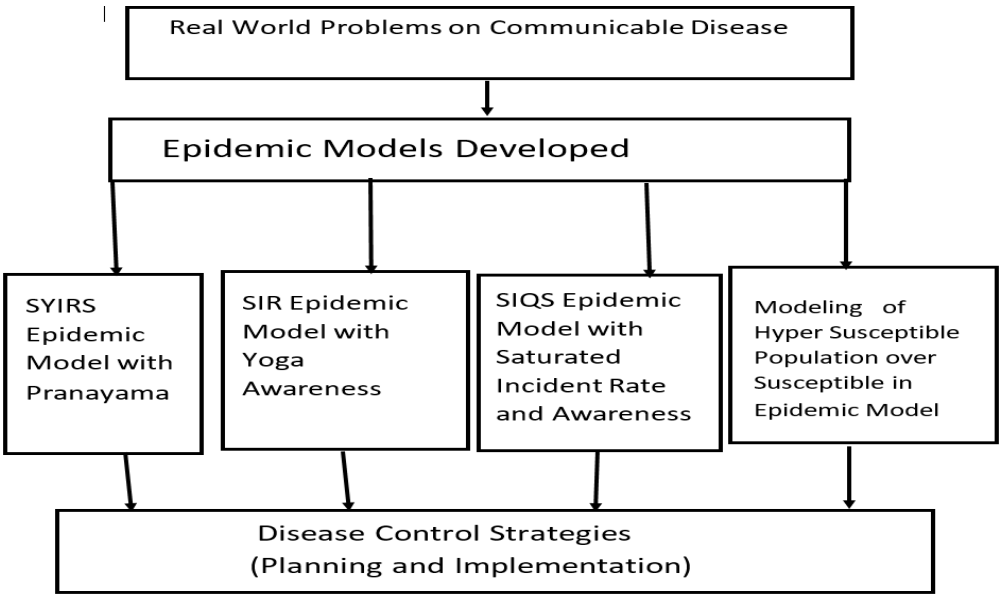


Figure 1.3: Developed conceptual framework.

Statement of the Problem

Communicable diseases like influenza and COVID-19 are serious global health problems caused by viruses and transmitted among humans mainly through direct contact with infected individuals, contact with contaminated objects, and inhalation of aerosols containing viral particles. They have been a major cause of morbidity and mortality throughout human history, with millions of people suffering or dying each year. During the twentieth century, influenza pandemics occurred in 1918, 1957, and 1968, with approximately one-third of the world population infected during the 1918–1919 pandemic. Despite significant advances in medical science, communicable diseases continue to affect large populations worldwide (e.g., $SARS$ in 2003, influenza in 2005, swine flu during 2009–2015, COVID-19 during 2019–2022). The rapid spread of diseases is facilitated by global migration and modern transportation. In this century, humanity faces even more complex environmental and health-related challenges than ever before.

Communicable diseases are major health concerns that affect the economy of nations. Their complexity and the limited understanding of their dynamics make them a serious public concern. Therefore, it should be a prime agenda for both developed and developing countries to educate people about transmission dynamics, prevention, and control strategies. Comparative knowledge of different preventive measures, such as public awareness, pranayama, yoga awareness, quarantine, and vaccina-

tion, is essential. Epidemiological modelers need to formulate models that clearly and precisely incorporate these measures.

The effect of yoga in controlling or preventing the spread of communicable diseases has not yet been mathematically modeled. Likewise, its efficacy on disease transmission dynamics and its role in improving health have not been systematically studied. Therefore, mathematical models that describe disease transmission dynamics in conjunction with yoga efficacy are needed. The formulation of the problem in this dissertation is inspired by ethnography from different social settings, the Eastern philosophy of yoga, and the recent COVID-19 pandemic.

Different theoretical works focusing on human health-related problems have been studied, and it has been determined that this research has broader scope than previous studies. People are particularly concerned about communicable diseases like COVID-19 due to their complexity and the lack of comprehensive knowledge. At the time of this study, no vaccine was universally available for COVID-19, and no definite control strategy had been established. Formulating an appropriate model that addresses the real situation of communicable disease with yoga efficacy can make the field of bio-mathematical modeling both exciting and highly relevant to medical research.

Many researchers have worked on biological phenomena using various mathematical approaches, including algebraic, transcendental, differential, and difference equations. In this dissertation, extended forms of the SIR and SIS epidemic models have been developed. These mod-

els are governed by ordinary differential equations (ODEs), which are solved analytically using existing mathematical theories and $Mathematica$ software. These model equations facilitate the following:

a. Formulate models in terms of variables and parameters.

b. Determine relationships between variables and parameters.

c. Analyze these models.

d. Interpret research outcomes in the context of real-world scenarios.

The relationships between variables and parameters are studied using mathematical theories and complemented by direct numerical simulations. It is assumed that disease propagation can be minimized or fully stopped by increasing control measures such as pranayama, yoga awareness, and quarantine with yoga awareness.

Research Questions

The following research questions were formulated:

1. What are the extended or improved forms of epidemic models that incorporate the efficacy of yoga?
2. How can epidemic models of communicable disease transmission dynamics be formulated to include yoga?
3. How can these models be analyzed?
4. Why are these models significant?

Objectives

The general objective of this research is to investigate the yoga efficacy in real-world communicable disease problems and to quantify their dynamic behaviors. The specific objectives are as follows:

1. To formulate and analyze an improved SIR epidemic model with a yoga–pranayama compartment
2. To develop and analyze an SIR epidemic model incorporating yoga awareness.
3. To formulate and analyze the SIS epidemic model with quarantine and yoga awareness at a saturated incident rate.
4. To develop a compartmental epidemic model incorporating hypersusceptibility and to analyze its dynamics.

Research Methodology

This research focuses on investigating and addressing the formulated research questions. The methodology emphasizes the formulation of realistic mathematical models that accurately capture disease dynamics in society. Developing a mathematical model for communicable disease transmission is a complex, but essential component of this study (Heathcote, 2014; J. D. Murray, 2001).

Research Philosophy. This research seeks to identify the objective truth regarding the efficacy of yoga on the transmission dynamics of communicable diseases through quantitative analysis and generalization of findings. The philosophical foundation guiding this work is rooted in objectivism (ontology) and positivism (epistemology), establishing an external and independent reality (Creswell, 2014).

The first step in modeling involves constructing a mathematical representation of a specific disease using appropriate variables and parameters. Based on underlying assumptions, the model is solved using mathematical techniques for non-linear ordinary differential equations and related methods. Mathematical modeling translates real-world phenomena into mathematical form, from which results are interpreted back into real-world implications (Brauer et al., 2019).

Ontology. Communicable diseases account for a significant proportion of global morbidity and mortality, especially in developing countries. They spread through multiple transmission routes, including air,

water, body secretions, and insect vectors. These diseases, however, can be prevented, controlled, and treated through interventions such as vaccination, quarantine, pharmacological treatment, and public awareness programs. While previous studies have investigated the positive effects of such interventions, the influence of yoga on disease prevention and control has not been explored through mathematical modeling using ordinary differential equations.

These mathematical models represent an objective reality independent of individual perspectives. From an ontological standpoint, this study assumes that mathematical modeling incorporating yoga as a preventive measure reflects an objective, external reality (objectivism). Such models are generalizable to similar contexts, demonstrating a universal perspective.

Epistemology. Epistemology concerns the generation and validation of reliable knowledge using scientific methods. In this study, observable and measurable scientific evidence is used to establish relationships between variables and parameters within mathematical models. Analytical and numerical methods guide both model formulation and validation, ensuring rigorous knowledge generation.

Positivism underpins the epistemological approach, emphasizing the use of scientific methods to examine these models. Mathematical modeling translates real-world problems into mathematical structures, which are then solved and reinterpreted in the real-world context. If the

outcomes are unsatisfactory, the model is refined and reanalyzed. Iterative application of this process leads to results that increasingly approximate the actual phenomenon.

Axiology. Axiology, the study of values, addresses the influence of researchers' perspectives on the research process. This study adopts a value-free approach, maintaining researcher neutrality and independence throughout. All analyses and interpretations are based on objective evidence and logical reasoning rather than personal belief or bias.

By focusing on the mathematical modeling of communicable disease dynamics with yoga and pranayama interventions, this research ensures unbiased, generalizable findings. Scientifically, it contributes to mathematical epidemiology by introducing novel model components, such as yoga awareness and hyper-susceptible populations, thereby enhancing understanding of non-pharmaceutical interventions.Socially and ethically, it highlights accessible, low-cost strategies for disease prevention—particularly valuable for resource-limited communities—supporting equitable public health outcomes.

Research Method. This study adopts a quantitative, model-based methodology to examine the transmission dynamics of communicable diseases and to evaluate the potential effects of yoga interventions, including pranayama and yoga awareness, on disease prevention and control. The methodology is systematically structured around the formulation, analysis, and validation of compartmental epidemic models, sup-

plemented by numerical simulations and conceptual interpretation. This approach is objective, reproducible, and grounded in a positivist epistemological framework.

Mathematical modeling serves as the principal research approach. Communicable diseases are represented by systems of ordinary differential equations (ODEs) that describe interactions among susceptible, infected, recovered, quarantined, and hyper-susceptible populations. These models are further extended to incorporate preventive interventions such as yoga practices and pranayama, which are hypothesized to reduce disease transmission and enhance immunity. The key methodological components are as follows:

Population $\Rightarrow$ Compartments $\Rightarrow$ ODE Model $\Rightarrow$ Analysis $\Rightarrow$ Simulation $\Rightarrow$ Interpretation

The results of the mathematical analyses and simulations are interpreted to derive meaningful insights for real-world applications. A conceptual framework is developed to map the relationships among population compartments, transmission parameters, and intervention strategies. This framework facilitates the translation of theoretical model outcomes into practical guidance for public health decision-making.

Overall, this methodology ensures that the research remains scientifically rigorous, objective, and generalizable. It provides evidence-based insights into the role of yoga interventions in disease control and supports the formulation of effective, non-pharmaceutical public health strategies.

Significance of the Research

Modeling the transmission dynamics of infectious diseases provides valuable insights for the Ministry of Health, hospitals, provincial governments, and researchers. This study enhances understanding of communicable diseases that confer immunity after recovery and has the following significance:

1. Identifying alternative approaches, such as yoga and yoga awareness, to control disease transmission.
2. Supporting public health officials in developing effective disease control strategies.
3. Examining the relationship between disease transmission and environmental factors globally.
4. Highlighting the benefits of yoga in the progression and natural history of diseases.
5. Developing models that realistically capture disease transmission scenarios.
6. Predicting the effectiveness of control strategies, including those based on Eastern philosophical practices.

Delimitations of the Research

The delimitations of this research are as follows:

1. The study primarily relies on secondary data, with primary data collected from 20 yoga centers in Sudurpashchim Province between March 2021 and August 2022. The study period is limited to March 2021–August 2022.

2. Only the SIS and SIR models, along with their extended forms, are utilized.

3. Only communicable diseases transmitted through direct contact and affecting the respiratory system (e.g., influenza and COVID-19) are considered, while vector-borne diseases are excluded.

4. Yoga in this dissertation refers specifically to the fourth limb pranayama of *Ashtanga yoga*. pranayama comprises eight practices: *Vasti*, *Kapalbhatika*, *Vahya-Pranayama*, *Ujjayi*, *Anulom-Vilom*, *Bhramari*, *Udgitha*, and *Pranawa-Dhyan*.

5. Yoga awareness encompasses four pillars of yoga: Aahar, Vihar, Aachar, and Vichar. Aachar includes practices such as handwashing, social distancing, mask-wearing, and sanitizing.

6. The study focuses solely on the impact of yoga on disease transmission rates, not on recovery rates; the average recovery rate is used instead.

Organization of the Dissertation

This dissertation is organized into seven chapters, each addressing a specific aspect of the study. The structure has been designed to systematically address the research questions, achieve the objectives, and provide a coherent flow from theory to application.

Chapter 1: General Introduction The present chapter provides an overview of the prerequisites relevant to this research. It includes key definitions, fundamental concepts, and a concise discussion of the philosophical foundations underlying the study. A brief review of the literature is presented, highlighting its implications for the dissertation. Research questions, objectives, significance, and delimitations of the study are also specified.

Chapter 2: Mathematical Preliminaries This chapter introduces the mathematical preliminaries and fundamental concepts of mathematical epidemiology that are applied throughout the dissertation. Relevant lemmas, theorems, and concepts used in subsequent chapters are also presented.

Chapter 3: Analysis of an $SYIRS$ Epidemic Model with Yoga A novel $SYIRS$ model incorporating a Yoga Sadhaka compartment is proposed in this chapter. The effective reproduction number is calculated, and stability conditions are analyzed. Since mathematical models cannot fully capture real-world transmission dynamics, numerical simulations using available data are conducted to illustrate the model's behav-

ior.

Chapter 4: Analysis of an $SIRS$ Epidemic Model Incorporating Yoga Awareness This chapter examines the impact of awareness dissemination—through yoga classes or awareness campaigns—on disease transmission. An SIR model is employed, and stability analysis is performed using the Routh-Hurwitz criteria, Lyapunov functions, and Dulac criteria. Numerical simulations are provided to validate the theoretical findings.

Chapter 5: Analysis of an $SIQS$ Epidemic Model with a Saturated Incidence Rate Incorporating Yogachara An $SIQS$ epidemic model with a saturated incidence rate is developed to study the effect of quarantine and Yogachara. Both local and global stability analyses are carried out using Routh-Hurwitz criteria, Lyapunov functions, and Dulac criteria. Numerical simulations are presented to support theoretical results.

Chapter 6: Dynamics of Susceptible and Hyper-Susceptible Populations in an Epidemic Model This chapter investigates the dynamics of hyper-susceptible populations compared to general susceptible populations. The basic reproduction number is determined, providing insights into disease persistence or elimination. Local and global stability analyses are conducted, followed by numerical simulations consistent with theoretical findings, illustrating the behavior of susceptible and hyper-susceptible populations.

Chapter 7: Findings and Conclusions The final chapter summa-

rizes the key findings of the study and presents conclusions. Recommendations for future research and potential applications of the models are also discussed.

Linking Chapters to Research Objectives and Questions This dissertation addresses specific research questions through extended epidemic models incorporating yoga-based interventions. Chapters 3–6 analyze model stability, evaluate the effects of yoga interventions on disease prevention, and present insights from numerical simulations. Collectively, these chapters enhance understanding of yoga's potential role in controlling communicable diseases.

Chapter 2

Mathematical Preliminaries

Preamble

This chapter introduces the fundamental mathematical concepts used throughout the dissertation, with a particular focus on differential equations and mathematical epidemiology. Mathematical modeling generally involves constructing equations that provide insight into real-world problems. In the context of infectious diseases, such models are essential for understanding transmission dynamics and forecasting disease spread. Various tools, including ordinary differential equations (ODEs), partial differential equations (PDEs), and other analytical methods, are commonly employed in mathematical modeling. In the present work, ordinary differential equations are used to describe the dynamics of communicable diseases.

Ordinary Differential Equations

Linear Differential Equations

Definition 2.1 (Linear Differential Equations)**.** *A system of first-order ordinary differential equations (ODEs) has the general form:*

$$\begin{aligned}
\frac{dX_1}{dt} &= F_1(t, X_1, X_2, \ldots, X_n), \\
\frac{dX_2}{dt} &= F_2(t, X_1, X_2, \ldots, X_n), \\
&\vdots \\
\frac{dX_n}{dt} &= F_n(t, X_1, X_2, \ldots, X_n),
\end{aligned}$$

where $X_1(t), X_2(t), \ldots, X_n(t)$ *are scalar functions of time* t*.*

The system can be written in compact vector form:

$$X'(t) = \frac{dX(t)}{dt} = F(t, X), \tag{2.1}$$

where

$$X(t) = (X_1(t), X_2(t), \ldots, X_n(t))^T,$$

$$F(t, X) = (F_1(t, X), F_2(t, X), \ldots, F_n(t, X))^T.$$

An n-dimensional system is *linear* if there exists a matrix A such that

$$X'(t) = A(t)X(t) + g(t), \tag{2.2}$$

where $g(t)$ is a vector of continuous functions. Equation (2.2) is homogeneous if $g(t) = 0$. A vector $X(t) = \phi(t)$ is a solution of (2.2) if its components satisfy the system on its interval of definition. We assume that the functions $A(t)$ and $g(t)$ are continuous on the interval (a, b) (Allman et al., 2004; Boyce, DiPrima, & Meade, 2021; Dym, 2004).

The initial conditions are

$$X_1(t_0) = a_1, \quad X_2(t_0) = a_2, \ldots, X_n(t_0) = a_n,$$

or in vector form $X(t_0) = a = (a_1, a_2, \ldots, a_n)^T$.

Not every system can be written as $X' = AX$. A single unknown requires one equation; multiple unknowns require a system. For example, the Lotka–Volterra (predator–prey) model is:

$$\begin{aligned} \frac{dx}{dt} &= ax - \alpha xy, \\ \frac{dy}{dt} &= -cy + \gamma xy, \end{aligned} \tag{2.3}$$

where $x(t)$ and $y(t)$ represent prey and predator populations. The constants a, α, c, and γ are empirical parameters depending on the species studied. Equation (2.3) can also be written in compact form as:

$$\mathbf{X}'(t) = \mathbf{F}(\mathbf{X}(t)), \qquad \text{where} \quad \mathbf{X}(t) = \begin{bmatrix} x(t) \\ \\ y(t) \end{bmatrix}, \quad \mathbf{F}(\mathbf{X}) = \begin{bmatrix} ax - \alpha xy \\ \\ -cy + \gamma xy \end{bmatrix}.$$

This vector form illustrates the application of ODEs in ecological modeling.

Nonlinear Differential Equations

Definition 2.2 (Nonlinear Differential Equations)**.** *A nonlinear differential equation is an equation of the form*

$$X_{n+1} = f(X_n, X_{n-1}, \dots),$$

where the function f depends on nonlinear combinations of its arguments.

These may include quadratic terms, exponentials, reciprocals, powers of the variables, or other nonlinear operations. A solution is a general formula relating X to the generation n and to some initially specified values X_0, X_1, and so on.

Equivalently, a nonlinear system of differential equations is a system that cannot be expressed in the linear form $X' = AX$ for some matrix A. In such equations, the unknown function and its derivatives do not produce a straight line when plotted; for example, products of a dependent variable with itself or with its derivative may occur.

Nonlinear differential equations may or may not be solvable exactly. Determining the existence, uniqueness, and extendibility of solutions, as well as the well-posedness of initial and boundary value problems, is often challenging. When exact solutions exist, it is considered a significant advancement in mathematical theory. However, if a differential equation accurately describes a real-world physical process, a solution must exist. Linear differential equations are often used to ap-

proximate nonlinear equations (Banerjee, 2021; N. F. Britton & Britton, 2003). For example, the harmonic oscillator equation serves as a close approximation of the nonlinear pendulum equation for small-amplitude oscillations. While some nonlinear differential equations have known exact solutions, many important applications involve equations for which exact solutions are not available. In such cases, linearization procedures, such as series expansions, are used to eliminate nonlinear terms; however, these methods fail when nonlinear terms significantly influence the solution.

Definition 2.3 (Autonomous Differential Equations)**.** *An n-dimensional system of differential equations*

$$X' = F(t, X)$$

is called autonomous if F depends only on X. Thus, when discussing autonomous systems, we write

$$X' = F(X).$$

These equations are used in growth or decline of population.

Definition 2.4 (Equilibrium Point)**.** *An equilibrium point of a dynamical system (2.1) is a point $X^* \in \mathbf{R}^n$ such that*

$$\frac{dX^*}{dt} = 0.$$

An equilibrium solution is a constant solution $X(t) = X^*$ for all t, where the derivatives of the system vanish; that is,

$$F(X^*) = 0.$$

Theorem 2.1 (Existence and Uniqueness Theorem)**.** *Let the function f and its partial derivative $\frac{\partial f}{\partial X}$ be continuous in a rectangle $\alpha < t < \beta$, $\gamma < X < \delta$ containing the point (t_0, X_0). Then there exists an interval $t_0 - h < t < t_0 + h$ within $\alpha < t < \beta$ in which the initial value problem*

$$\frac{dX(t,X)}{dt} = g(t,X), \quad X(t_0) = X_0$$

has a unique solution $X = \phi(t)$.

Definition 2.5 (Well-Posedness)**.** *The system of equations (2.2) is called well-posed if a solution exists, is unique, and depends continuously on the initial data. In other words, small changes in the initial data result in small changes in the solution.*

Proposition 2.1. *Let $X(t)$ be a solution to the autonomous system $X' = F(X)$ with $F \in C^1(\mathbf{R}^n)$, such that*

$$\lim_{t\to\infty} X(t) = X^*.$$

Then X^* is an equilibrium solution; that is,

$$F(X^*) = 0.$$

Stability

Once a solution to a system of ordinary differential equations has settled, its limiting value is an equilibrium solution. In physical systems, the only directly observable steady-state solutions are the stable equilibria. Unstable equilibria are difficult to sustain and will disappear when subjected to even small perturbations. We focus on autonomous systems

$$u' = F(u), \tag{2.4}$$

whose right-hand sides are continuously differentiable to ensure the uniqueness of solutions to the initial value problem. If every solution that starts near a given equilibrium solution tends to it, the equilibrium is called *asymptotically stable*. If solutions that start nearby remain nearby, the equilibrium is *stable*. More formally, we define stability as follows (Olver, 2006; Saaty, 2012; THOMAS, 2017).

Definition 2.6. *An equilibrium solution u^* to an autonomous system of first-order ordinary differential equations is called:*

- ***Stable** if, for every $\epsilon > 0$, there exists $\delta > 0$ such that every solution $u(t)$ with initial condition satisfying $||u(t_0) - u^*|| < \delta$ remains within $||u(t) - u^*|| < \epsilon$ for all $t > t_0$.*
- ***Asymptotically stable** if it is stable and, in addition, there exists $\delta_0 > 0$ such that whenever $||u(t_0) - u^*|| < \delta_0$, then $u(t) \to u^*$ as $t \to \infty$.*

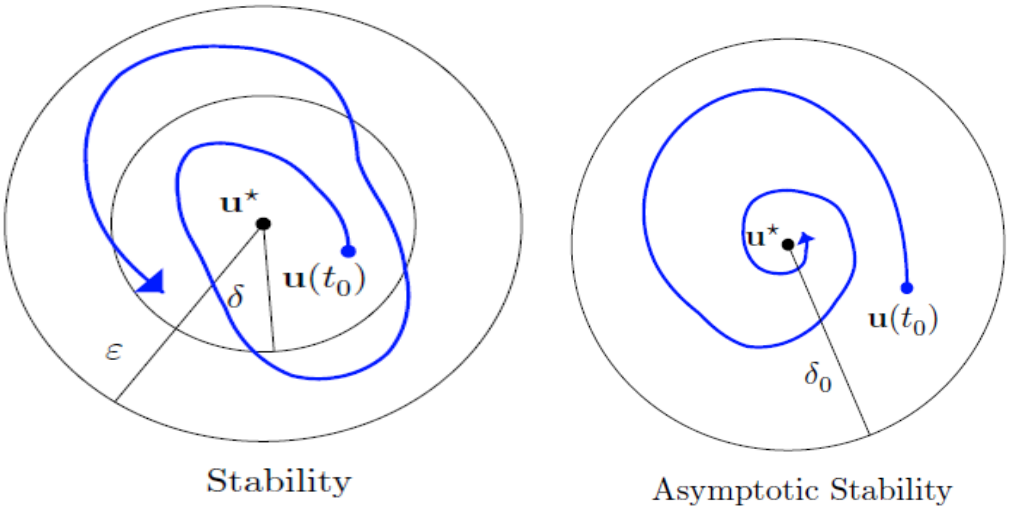


Figure 2.1: Stability of Equilibria.

Theorem 2.2 (Stability (G. O. Agaba, 2016))**.** *Let u^* be an equilibrium point of the system (2.4). Let λ_k for $k = 1, 2, \ldots, n$ be the eigenvalues of $J = DF(u^*)$. Then:*

- *u^* is asymptotically stable if all eigenvalues have negative real parts.*
- *u^* is stable if all eigenvalues are purely imaginary.*
- *u^* is unstable if at least one eigenvalue has a positive real part.*

Theorem 2.3. *An equilibrium point u^* of an autonomous scalar differential equation is asymptotically stable if and only if*

$$F(u) > 0 \text{ for } u^* - \delta < u < u^*, \quad F(u) < 0 \text{ for } u^* < u < u^* + \delta,$$

for some $\delta > 0$. This is illustrated in Figure 2.2.

Theorem 2.4. *Let u^* be an equilibrium point for a scalar ordinary dif-*

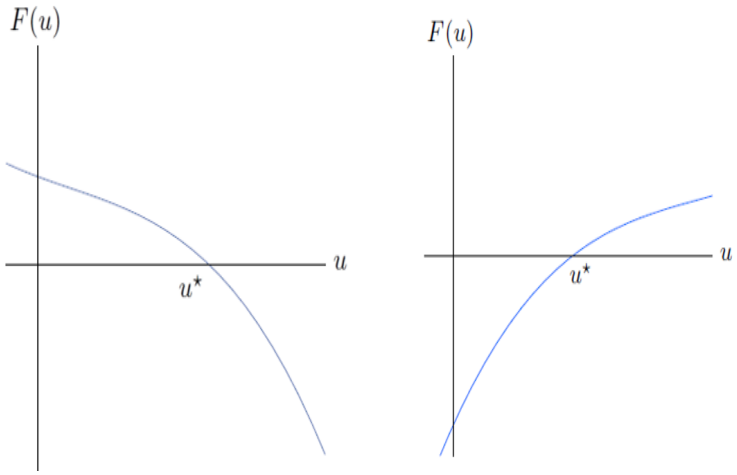


Figure 2.2: *Stable equilibria of ordinary differential equations.*

ferential equation $u' = F(u)$. *If* $F'(u^*) < 0$, *then* u^* *is asymptotically stable. If* $F'(u^*) > 0$, *then* u^* *is unstable. This is shown in Figure 2.3.*

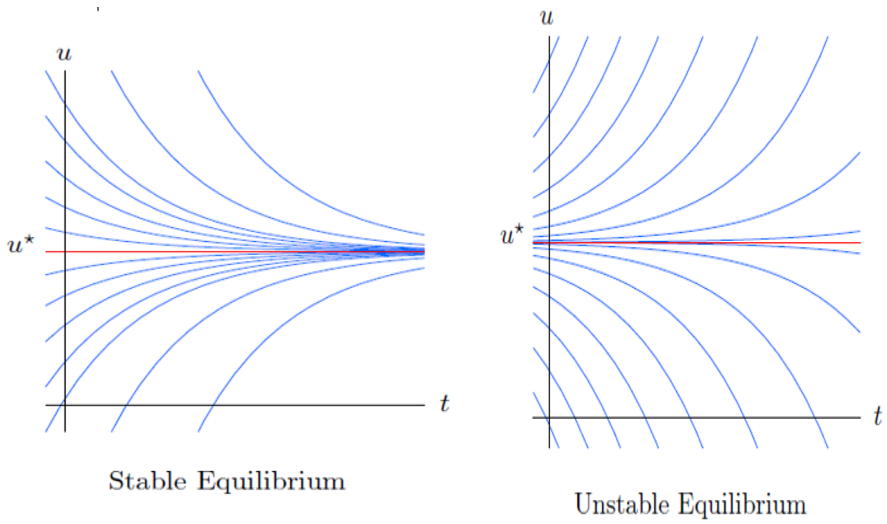


Figure 2.3: Stable Equilibria of Ordinary Differential Equations.

Proposition 2.2 (Linearization)**.** *For any* n*-dimensional system of differential equations of the form*

$$X' = AX + V$$

for some $V \in \mathbf{R}^n$, with a unique equilibrium point X_e, the change of coordinates $u = X - X_e$ yields a linear system

$$u' = Au$$

with a unique equilibrium point at 0.

Definition 2.7 (Jacobian Matrix)**.** *The Jacobian matrix of the system (2.1) is the matrix of all partial derivatives of the right-hand side of the system with respect to the state variables. That is,*

$$J = DF(X) = \begin{bmatrix} \frac{\partial F_1}{\partial x_1} & \frac{\partial F_1}{\partial x_2} & \cdots & \cdots & \frac{\partial F_1}{\partial x_n} \\ \frac{\partial F_2}{\partial x_1} & \frac{\partial F_2}{\partial x_2} & \cdots & \cdots & \frac{\partial F_2}{\partial x_n} \\ \vdots & \vdots & \ddots & & \vdots \\ \frac{\partial F_n}{\partial x_1} & \frac{\partial F_n}{\partial x_2} & \cdots & \cdots & \frac{\partial F_n}{\partial x_n} \end{bmatrix}. \tag{2.5}$$

Routh-Hurwitz Criteria

Consider an n-dimensional system of equations (2.1) in matrix form:

$$\frac{dX(t)}{dt} = AX,$$

where $X = (X_1, X_2, \ldots, X_n)^T$, $\frac{dX(t)}{dt} = \left(\frac{dX_1}{dt}, \frac{dX_2}{dt}, \ldots, \frac{dX_n}{dt}\right)^T$, and $A = (c_{ij})$ is an $n \times n$ matrix with $c_{ij} \in \mathbf{R}$ and $\det(A) \neq 0$.

The characteristic equation of A is

$$\det(A - \lambda I) = 0.$$

Expanding this determinant, we obtain the characteristic polynomial:

$$P(\lambda) = \lambda^n + c_1\lambda^{n-1} + c_2\lambda^{n-2} + \cdots + c_{n-1}\lambda + c_n, \tag{2.6}$$

where $P(\lambda)$ is a polynomial of degree n with real coefficients c_k, $k = 1, 2, \ldots, n$. The roots of (2.6) are the eigenvalues $\lambda_1, \lambda_2, \ldots, \lambda_n$ of the square matrix A.

In many cases, the characteristic equation $\det(A - \lambda I) = 0$ cannot be solved analytically for the n eigenvalues (Mahardika, Widowati, & Sumanto, 2019; Sungchasit, Tang, & Pongsumpun, 2022). The Routh-Hurwitz criteria provide a method to determine the asymptotic stability of the system based solely on the coefficients of the characteristic polynomial. The first few determinants in the Routh-Hurwitz array are:

$$H_1 = \begin{vmatrix} c_1 \end{vmatrix}, \quad H_2 = \begin{vmatrix} c_1 & 1 \\ c_3 & c_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} c_1 & 1 & 0 \\ c_3 & c_2 & c_1 \\ c_5 & c_4 & c_3 \end{vmatrix}, \cdots$$

In general, the ith determinant H_i is

$$H_i = \begin{vmatrix} c_1 & 1 & 0 & 0 & \cdots & 0 \\ c_3 & c_2 & c_1 & 1 & \cdots & 0 \\ c_5 & c_4 & c_3 & c_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ c_{2i-1} & c_{2i-2} & c_{2i-3} & c_{2i-4} & \cdots & c_i \end{vmatrix},$$

and the last determinant H_n is

$$H_n = \begin{vmatrix} c_1 & 1 & 0 & 0 & \cdots & 0 \\ c_3 & c_2 & c_1 & 1 & \cdots & 0 \\ c_5 & c_4 & c_3 & c_2 & \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & 0 & 0 & \cdots & c_n \end{vmatrix}.$$

The (x, y) entry of the matrix H_i is defined as:

$$(H_i)_{x,y} = \begin{cases} c_{2x-y}, & 0 < 2x - y \leq n, \\ 1, & 2x = y, \\ 0, & 2x < y \text{ or } 2x > y + n. \end{cases}$$

Theorem 2.5 (Routh-Hurwitz Criteria (Edelstein-Keshet, 2005))**.** *For a characteristic equation (2.6), all roots of the polynomial $P(\lambda)$ have negative real parts if and only if all determinants of the Hurwitz matrices are positive, that is,* $\det(H_k) > 0$ *for* $k = 1, 2, \ldots, n$.

Particular Cases.

- **Second-order system** ($n = 2$): The characteristic equation is

$$\lambda^2 + c_1\lambda + c_2 = 0.$$

The Hurwitz matrices are

$$H_1 = \begin{vmatrix} c_1 \end{vmatrix}, \quad H_2 = \begin{vmatrix} c_1 & 1 \\ 0 & c_2 \end{vmatrix}.$$

The Routh-Hurwitz criteria are:

$$\det(H_1) > 0 \Rightarrow c_1 > 0, \quad \det(H_2) > 0 \Rightarrow c_2 > 0.$$

- **Third-order system** ($n = 3$): The characteristic equation is

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0.$$

The Hurwitz matrices are

$$H_1 = \begin{vmatrix} c_1 \end{vmatrix}, \quad H_2 = \begin{vmatrix} c_1 & 1 \\ c_3 & c_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} c_1 & 1 & 0 \\ c_3 & c_2 & c_1 \\ 0 & 0 & c_3 \end{vmatrix}.$$

The Routh-Hurwitz criteria are:

$$c_1 > 0, \quad c_3 > 0, \quad c_1c_2 > c_3.$$

- **Fourth-order system** ($n = 4$): The characteristic equation is

$$\lambda^4 + c_1\lambda^3 + c_2\lambda^2 + c_3\lambda + c_4 = 0.$$

The Hurwitz matrices are

$$H_1 = \begin{vmatrix} c_1 \end{vmatrix}, \quad H_2 = \begin{vmatrix} c_1 & 1 \\ c_3 & c_2 \end{vmatrix}, \quad H_3 = \begin{vmatrix} c_1 & 1 & 0 \\ c_3 & c_2 & c_1 \\ 0 & c_4 & c_3 \end{vmatrix}, \quad H_4 = \begin{vmatrix} c_1 & 1 & 0 & 0 \\ c_3 & c_2 & c_1 & 1 \\ 0 & c_4 & c_3 & c_2 \\ 0 & 0 & 0 & c_4 \end{vmatrix}.$$

The Routh-Hurwitz criteria are:

$$c_1 > 0, \quad c_2 > 0, \quad c_3 > 0, \quad c_4 > 0, \quad c_1 c_2 c_3 > c_3^2 + c_1^2 c_4.$$

Mathematical Epidemiology

Overview of Communicable Diseases

Epidemiology deals with the distribution and determinants of disease prevalence in humans. Its primary function is to describe the distribution of disease (i.e., who is affected, by how much, where, and when). Another function is to identify the causes or risk factors for diseases, explaining why the disease does not affect everyone uniformly. A third function is to build and test theories, and a fourth is to plan, implement, and evaluate detection, control, and prevention programs. Epidemiological modeling plays an important role in the latter two functions, often allowing comparisons between different diseases in the same population, the same disease in different populations, or the same disease over time. Such models are also useful in evaluating the effects of prevention or control measures.

The goal of epidemiology in human health is not only to provide solutions or treatments for health problems but also to trace the causes of diseases so that policymakers can develop strategies to prevent future outbreaks. Communicable diseases are commonly described using the epidemiological triad, which links the host (who receives the infection), the agent (which causes or spreads the infection), and the environment (which supports the formation and transmission of the disease) along with their biological interactions. The relationship among these components is illustrated in Figure 2.4.

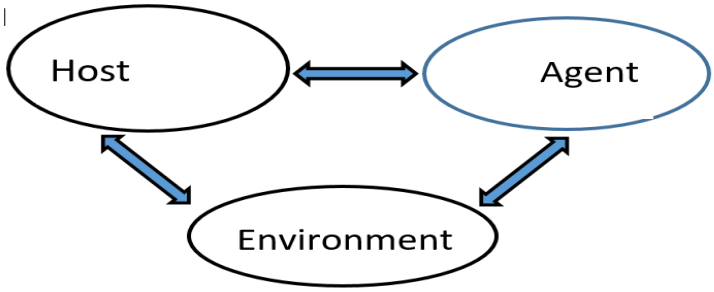


Figure 2.4: Relationship between the phases of virus spread in the epidemiological triad.

- **Agent:** A primary factor responsible for infection, which may include:

  a. Chemicals: Benzene, Oxygen, Asbestos.

  b. Microorganisms: Bacteria, Viruses, Protozoa.

  c. Physical energy sources: Radiation, Electricity.

  Often, multiple agents may contribute to a single disease.

- **Host:** In the epidemiological triad, the host and agent interact to cause disease. The infectious process involves transmission from one host to another via the agent.

- **Environment:** The external context influencing disease formation and transmission. Environmental factors include social, biological, and physical components. Physical environment factors, such as pollution, climate, and living conditions, can significantly affect disease spread. The host, agent, and environment are influenced by additional factors such as age, behavior, lifestyle, and genetic characteristics.

Basic Terminologies Used in Modeling (Martcheva, 2015)

Susceptible Individuals. Individuals who may be infected in the future by a disease that is present in other individuals are called susceptible individuals.

Exposed Individuals. A susceptible individual becomes exposed upon contact with a potentially infectious agent. Exposed individuals may or may not develop the disease and are typically not infectious. In mathematical models, it is often assumed that all exposed individuals eventually develop the disease.

Infected and Infectious Individuals. After a pathogen enters the body, it requires a period of development in the host. Individuals with an immature infection are termed infected, while those capable of transmit-

ting the disease are called infectious. Note that individuals may not be infectious during the entire infection period.

Latent Individuals. Latent individuals are infected but not yet infectious. The latent period is defined as the time from infection until the host becomes capable of transmitting the pathogen to others.

Incubation Period. The incubation period is the time between exposure to an infectious agent and the onset of the first signs or symptoms. In infectious diseases, this period corresponds to the time required for the pathogen to multiply to a threshold sufficient to produce detectable symptoms. The incubation period does not necessarily coincide with the latent period; for example, in influenza, individuals can become infectious approximately one day before exhibiting visible symptoms.

Incidence. Incidence refers to the number of individuals who become ill during a specified period, such as one year. Sometimes, it is expressed relative to the total population. Incidence is usually based on reported clinical cases, which may underestimate the true number of infections because subclinical cases are often unreported.

Prevalence. Prevalence is the number of individuals who have the disease at a specific time, sometimes expressed as a proportion of the total population.

Immunity. Immunity is the state of being protected against an infectious disease, either through previous infection or immunization.

Disease-Induced Mortality. Disease-induced mortality is the number of deaths caused by a disease during a given time period, often expressed relative to the total population.

Recovered Individuals. Individuals who have recovered from an infection are called recovered. Depending on whether immunity is lifelong, they may or may not be susceptible to reinfection.

Herd Immunity. Herd immunity occurs when a significant portion of a population is immune to an infectious disease, reducing its spread.

Epidemic. An epidemic occurs when the number of new cases significantly exceeds the expected baseline in a population over a defined period.

Endemic. An infection is endemic when it is consistently present at a baseline level within a geographic area without external input.

Pandemic. A pandemic occurs when a disease spreads across multiple countries in a short period and becomes difficult to control.

Mutation. Mutation refers to changes in the DNA or RNA sequence of a microorganism, often caused by errors during replication.

Vaccination. Vaccination is a safe and effective method to protect individuals against infectious diseases by stimulating an immune response before exposure.

Disease-Free Equilibrium. A disease-free equilibrium is a state where the infection is absent from the population, represented in mod-

els as $I = 0$.

Threshold Phenomenon. The threshold phenomenon refers to the critical level that must be reached for a particular effect to occur, such as the minimum number of susceptible individuals needed for disease spread.

Drugs. For infectious diseases lacking vaccines, drugs play a key role in treatment and reducing transmission. They can also be used prophylactically, either pre- or post-exposure, e.g., antimalarial prophylaxis for travelers to endemic regions (Joshi, 2020).

Quarantine. Quarantine is the separation and restriction of movement of individuals exposed to an infectious disease to monitor if they become sick. It has been used for diseases such as plague, cholera, influenza, SARS, and COVID-19. Quarantine strategies may include:

- Short-term voluntary isolation at home.
- Restriction of group gatherings (e.g., in schools or institutions).
- Suspension of public events and closure of theaters, schools, and colleges.
- Travel restrictions.

Immune Response. Immune response refers to the way the body's immune system detects and defends itself against harmful substances — such as bacteria, viruses, toxins, or other foreign invaders. The immune system protects the body from infection by recognizing and responding

to foreign antigens. It comprises lymphatic organs, blood cells, and their secreted products.

Mathematical Models

Linear and Nonlinear Models

A mathematical model is called *linear* if its governing equations are linear. For example, the following equations represent linear models:

$$\frac{dN}{dt} = \rho N, \tag{2.7}$$

where $\rho = \beta - \delta$ represents the net growth rate, given by the difference between the birth rate $\beta \geq 0$ and death rate $\delta \geq 0$, and

$$\frac{dN}{dt} = -KN, \quad K > 0. \tag{2.8}$$

Equations (2.7) and (2.8) describe population growth and radioactive decay, respectively. Conversely, a model is called *nonlinear* if its governing equations are nonlinear. For instance,

$$\frac{dN}{dt} = KN(C - N), \quad K > 0, \tag{2.9}$$

is a nonlinear model, known as the logistic differential equation. In these examples, N is the population, t is time, and K and C are constants.

Discrete and Continuous Models

A mathematical model is *discrete* if the dependent variable takes values only at discrete points of the independent variable. Such models are typically formulated using difference equations.

In contrast, a *continuous* model allows the independent variable to take any value within a given interval. Continuous models are usually expressed in the form of differential equations (ordinary or partial).

Static and Dynamic Models

A mathematical model is *static* if its equations are independent of time. An example of a static model is the flow of fluid through a rigid, diverging tube.

A model is *dynamic* if its dependent variables change with time. Most real-world phenomena, such as population growth, epidemic spread, and radioactive decay, are best represented by dynamic models.

Deterministic and Stochastic Models

If the changes in the variables of a model are predictable with certainty, the model is called *deterministic*. For example, the motion of a simple pendulum can be modeled deterministically.

In contrast, if the changes in variables are subject to chance and cannot be predicted with certainty, the model is *stochastic*. For example, dropping a rubber ball from a fixed height and measuring its bounce

repeatedly produces variable outcomes due to inherent randomness.

The differences between deterministic and stochastic models can be summarized as follows:

- A deterministic model produces the same output given the same initial conditions, whereas a stochastic model incorporates randomness and can yield different outcomes depending on the realized values of random variables.
- Deterministic models describe the mean trend of a process, without accounting for variability. Stochastic models capture both the mean trend and the variability around it.
- Deterministic models are appropriate for large populations, where individual-level random events have negligible effect. Stochastic models are more suitable for small populations, where random events can significantly impact dynamics.

Some studies have shown that stochastic models may predict disease extinction even when the basic reproduction number R_0 is greater than one. Differences between stochastic and deterministic models are particularly pronounced in small populations (e.g., 10 individuals), whereas in larger populations (e.g., 1000 individuals), the predictions of both models tend to converge. In stochastic models, some parameters may be uncertain and represented by probability distributions rather than fixed values.

The primary goal of constructing mathematical models is to explain observations, make predictions, and facilitate decision-making. Math-

ematical models are widely used in scientific research, across diverse fields, and are designed to provide a structured framework for understanding complex systems and guiding effective actions.

Basic Compartmental Epidemic Models

The concept behind compartmental models is to partition the entire population into distinct classes according to epidemiological status. The approach of dividing a population into compartments is widely used in epidemic modeling (G. Agaba et al., 2017a; Allman et al., 2004; Banerjee, 2021; N. Britton, 2005; Martcheva, 2015; Zill, 2012). In mathematical models, compartments are typically denoted as S, E, I, R, Q, and V, representing susceptible, exposed, infectious, recovered, quarantined, and vaccinated populations, respectively. Individuals who are at risk of infection belong to the S (susceptible) compartment. Persons who are infected but asymptomatic or unable to transmit the infection are placed in the E (exposed) compartment. Individuals who can transmit the infection to others belong to the I (infectious) compartment. Those who achieve permanent recovery are assigned to the R (recovered) compartment. Infected individuals who are isolated to reduce disease spread are assigned to the Q (quarantined) compartment, while individuals who have received vaccination are placed in the V (vaccinated) compartment.

In cases where immunity is temporary, recovered individuals eventually lose immunity and return to the susceptible compartment S. Different compartmental models can be formulated depending on disease

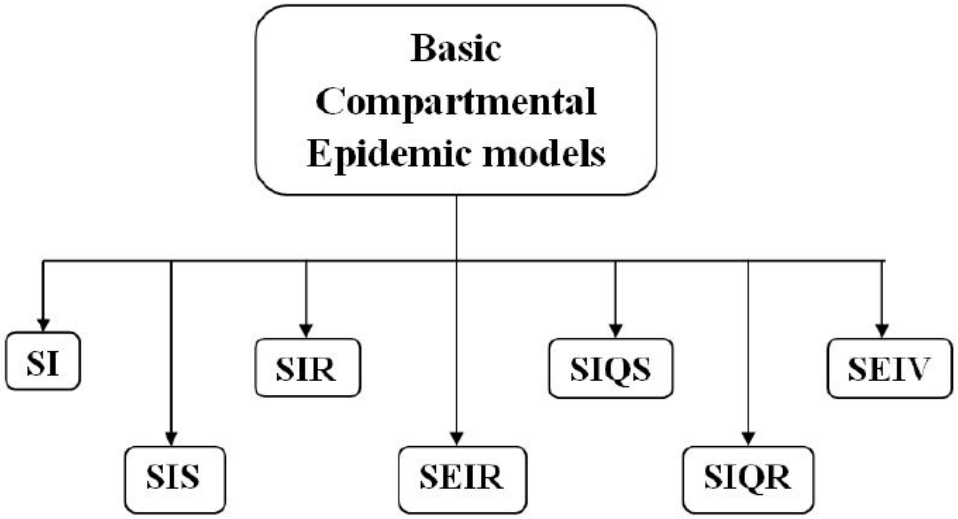


Figure 2.5: Some basic compartmental epidemic models.

characteristics and control measures, including SI, SIS, SIR, $SEIR$, $SIQS$, $SIQR$, $SEIV$, $SPIR$, and $SYIR$ models. SIS or $SEIS$ models are appropriate for diseases in which recovered individuals become susceptible again, as is the case with bacterial infections such as the common cold and influenza, where immunity is not permanent. SIR models are suitable for viral infections that confer permanent immunity, such as measles, mumps, and rubella. For diseases with an incubation period during which infected individuals are not yet infectious, $SEIR$ models are used to represent the exposed compartment E. Models such as $SIQS$, $SIQR$, or $SEIV$ are applied when quarantine or vaccination interventions are considered.

In all compartmental models, it is generally assumed that:

- All susceptible individuals have equal risk of infection.
- Populations are homogeneously mixed.

- Individuals acquire infection or recover at constant rates.

These simplifying assumptions allow for tractable mathematical analysis while capturing the essential dynamics of disease transmission.

Stability Analysis Criteria

Mathematical models often become highly complex when nonlinear equations of higher order are considered to represent real-world problems. In such cases, finding explicit solutions is very difficult or nearly impossible. While numerical simulations using fixed parameter values can provide good approximations of solutions, the general solution may remain undetermined. When a general solution cannot be obtained, stability analysis provides a useful tool to understand and predict the long-term behavior of solutions. To formalize the concept of stability, we define several key terms and state relevant theorems (Joshi, 2020; Khalil, 2002; Lamnabhi-Lagarrigue, 2005; Martcheva, 2015) that will be applied in later chapters.

Definition 2.8. *Consider an autonomous system*

$$X' = f(X), \tag{2.10}$$

where $X \subset \mathbf{R}^n$ is an open subset, and $f : X \to \mathbf{R}^n$ satisfies the standard conditions ensuring existence and uniqueness of solutions to the initial value problem associated with (2.10). Then X^* is called an equilibrium point of (2.10) if $f(X^*) = 0$.

Definition 2.9. *An equilibrium point X^* of (2.10) is stable if, for any given $\epsilon > 0$, there exists a $\delta > 0$ such that*

$$\|X(t_0) - X^*\| < \delta \implies \|X(t) - X^*\| < \epsilon, \quad \forall t > t_0. \tag{2.11}$$

An equilibrium point is unstable if it is not stable.

Definition 2.10. *An equilibrium point X^* is asymptotically stable if it is stable and, for initial values sufficiently close to X^*, the corresponding solution $X(t)$ satisfies*

$$\lim_{t\to\infty} X(t) = X^*. \tag{2.12}$$

If (2.12) holds for all solutions of (2.10), then X^* is said to be globally asymptotically stable.

Equivalently,

- A system is *locally asymptotically stable* (L.A.S.) at X^* if there exists $M > 0$ such that $\|X(0) - X^*\| \leq M \implies X(t) \to X^*$ as $t \to \infty$.

- It is *globally asymptotically stable* (G.A.S.) if, for every trajectory $X(t)$, $X(t) \to X^*$ as $t \to \infty$. This implies that X^* is a unique equilibrium point.

Global stability indicates that the equilibrium is stable for almost all initial conditions, not only for those in the vicinity of X^*. Establishing global stability is not always straightforward. If no other locally stable equilibria exist, a locally stable equilibrium may also be globally stable.

In this work, we use Lyapunov's stability theorem to establish the global stability of equilibrium points (Kuhl, 2021; Martcheva, 2015; Sigdel & McCluskey, 2014). For linear systems, local asymptotic stability implies global stability, and vice versa.

Definition 2.11. *(Khalil, 2002; Martcheva, 2015) Let* $F : \mathbf{R}^n \to \mathbf{R}$ *be a scalar function.* F *is called radially unbounded if* $F(X) \to \infty$ *as* $\|X\| \to \infty$.

Definition 2.12. *(Khalil, 2002; Martcheva, 2015) Let* $F : \mathbf{R}^n \to \mathbf{R}$ *be a scalar function.* F *is positive definite if*

- $F(X^*) = 0$,
- $F(X) > 0$ *for all* $X \in \mathbf{R}^n \setminus \{X^*\}$, *where* X^* *is an equilibrium point of system (2.10).*

Definition 2.13 ((Lamnabhi-Lagarrigue, 2005))**.** *A function* $F : \mathbf{R}^n \to \mathbf{R}$ *is positive definite if*

- $F(X) \geq 0$ *for all* X,
- $F(X) = 0$ *if and only if* $X = 0$,
- *All sublevel sets of* F *are bounded.*

Theorem 2.6. *(Joshi, 2020) Let* $X^* = 0$ *be an equilibrium point of (2.10) and let* V *be a positive definite function in a neighborhood of* X^*. *Then:*

- *If* $V' \leq 0$ *for all* $X \in \mathbf{R}^n \setminus \{0\}$, *then* 0 *is stable.*
- *If* $V' < 0$ *for all* $X \in \mathbf{R}^n \setminus \{0\}$, *then* 0 *is asymptotically stable.*

Lyapunov Theory

Lyapunov theory allows us to draw conclusions about the trajectories of a system

$$X' = F(X) \tag{2.13}$$

without explicitly solving the differential equation. A function V satisfying certain conditions can be used as a Lyapunov function, which can be interpreted as a generalized energy function (Lamnabhi-Lagarrigue, 2005; Zhu, Ni, et al., 2023).

Theorem 2.7. *A function $V : X \subset \mathbf{R}^n \to \mathbf{R}$ is called a Lyapunov function for (2.10) if:*

- *V is positive definite, and*
- *$V'(x) < 0$ for all $x \in X \setminus \{0\}$.*

Theorem 2.8. *Assume that $f(0) = 0$ and V is a Lyapunov function in X for the system (2.10). Let*

$$E = \{x \in X : V(x) = 0\},$$

and let M be the largest invariant subset in E with respect to (2.10). Then, every bounded solution of (2.10) in X tends to M as $t \to \infty$.

Theorem 2.9 (Lyapunov's Stability Theorem)**.** *If a function $f(x)$ is globally positive definite and radially unbounded, and its time derivative is*

globally negative, i.e.,

$$\frac{df(x)}{dt} < 0, \quad \forall x \neq x^*,$$

then the equilibrium point x^* is globally asymptotically stable.

Local and global stability analysis are commonly discussed in the literature. Local stability describes the behavior of the system near an equilibrium point, while global stability characterizes the behavior of the solution over the entire domain. Specifically, local stability of an equilibrium point implies that if the system starts near the equilibrium, it will return to it over time. In contrast, global stability means that the system returns to the equilibrium from any initial condition in the domain.

For a disease-free equilibrium, local stability indicates that introducing a small number of infected individuals (a small perturbation) will result in the system returning to the disease-free state. However, larger perturbations may drive the system toward an endemic equilibrium. On the other hand, global stability of the disease-free equilibrium ensures that the disease cannot persist in the population, regardless of the size of the perturbation.

Methods of Local Stability Analysis

To perform local stability analysis of equilibrium points, we first linearize the considered model system at the equilibrium point and then compute the characteristic values of the corresponding Jacobian (variation) ma-

trix. The signs of the real parts of these characteristic values determine the local stability of the equilibrium (Diekmann & Heesterbeek, 2000). Consider the system of ordinary differential equations

$$x' = f(x), \tag{2.14}$$

where $x = (x_1, x_2, \ldots, x_n)^T$ and $f = (f_1, f_2, \ldots, f_n)^T$. The local stability of an equilibrium x^* of system (2.14) is analyzed using the Jacobian matrix (Perko, 1996):

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial x_1} & \cdots & \frac{\partial f_n}{\partial x_1} \\ \vdots & \ddots & \vdots \\ \frac{\partial f_1}{\partial x_n} & \cdots & \frac{\partial f_n}{\partial x_n} \end{bmatrix}.$$

If all the eigenvalues of J at the equilibrium point x^* have negative real parts, then x^* is locally asymptotically stable. If one or more eigenvalues have positive real parts, then x^* is unstable. For $n = 3$, local stability can also be analyzed using the second additive compound matrix of the Jacobian. Consider a 3×3 matrix

$$A = \begin{bmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{bmatrix}. \tag{2.15}$$

Definition 2.14. *(M. Y. Li & Muldowney, 1995) The second additive compound matrix of (2.15), denoted $A^{[2]}$, is defined as*

$$A^{[2]} = \begin{bmatrix} a_{11}+a_{22} & a_{12} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ a_{31} & a_{21} & a_{22}+a_{33} \end{bmatrix}.$$

Theorem 2.10 ((McCluskey & Driessche, 2004))**.** *Let A be any 3×3 real matrix. All eigenvalues of A have negative real parts if and only if $\mathrm{tr}(A) > 0$, $\det(A) > 0$, and $\det(A^{[2]}) > 0$.*

Definition 2.15. *(Joshi, 2020) In many disease models, specific matrices with a definite sign pattern arise. One important form is the M-matrix, which has non-negative diagonal elements and non-positive off-diagonal elements.*

Methods of Global Stability Analysis

In epidemiological models, it is important to investigate the global stability of equilibrium states. Lyapunov's method (Guo, Li, & Shuai, 2012; Korobeinikov & Maini, 2004; Lyapunov, 1992) is widely used to study the global properties of epidemic models. This method requires constructing an auxiliary function (Lyapunov function) with precise properties, which can be challenging. Other approaches include Poincaré-Bendixson theory with Bendixson's negative criterion or its generalization by Dulac (Theodorakopoulos, Le Boudec, & Baras, 2012), geometric methods (M. Y. Li & Muldowney, 1996; Y. Li & Muldowney, 1993),

and the technique proposed by Busenberg and Van den Driessche (1990) based on nonexistence of periodic solutions in the feasible region.

Lyapunov Function. A Lyapunov function is a powerful tool to analyze the global stability of autonomous systems of ordinary differential equations. Although constructing an appropriate Lyapunov function can be complex, it is not unique. For epidemic models, Lyapunov functions can be constructed using a combination of the following forms (Vargas-De-León, 2009):

- Logarithmic form (commonly used in Lotka-Volterra systems):

$$L(x_1, x_2, \ldots, x_n) = \sum_{i=1}^{n} c_i \left(x_i - x_i^* - x_i^* \ln \frac{x_i}{x_i^*} \right)$$

- Quadratic form (used for linear and nonlinear systems):

$$V(x_1, x_2, \ldots, x_n) = \sum_{i=1}^{n} \frac{c_i}{2} (x_i - x_i^*)^2$$

- Composite quadratic form:

$$W(x_1, x_2, \ldots, x_n) = \frac{c}{2} \left[\sum_{i=1}^{n} (x_i - x_i^*) \right]^2$$

LaSalle's Invariance Principle. Let $V : \mathbf{R}^n \to \mathbf{R}$ satisfy:

- V is positive definite,

- $V'(x) < 0$ for all $x \neq 0$, with $V'(0) = 0$.

Then every trajectory of (2.14) converges to zero as $t \to \infty$, and the sys-

tem (2.14) is globally asymptotically stable (LaSalle & Lefschetz, 1976)

.

Bendixson-Dulac Criterion. (McCluskey & Muldowney, 1998)

Let $F_1(S, I)$, $F_2(S, I)$, and $H(S, I)$ be C^1 functions in a simply connected region $D \subset \mathbf{R}^2$ such that

$$\frac{\partial(F_1 H)}{\partial S} + \frac{\partial(F_2 H)}{\partial I}$$

does not change sign in D and vanishes at most on a set of measure zero. Then the system

$$\begin{cases} S'(t) = F_1(S, I) \\ I'(t) = F_2(S, I) \end{cases} \tag{2.16}$$

does not have any periodic solutions in D. The function $H(S, I)$ is called the Dulac function.

The application of all the above methods will be illustrated in subsequent chapters to establish the global properties of the proposed models.

Reproduction Number

Basic Reproduction Number

The concept of the basic reproduction number was introduced by Ross in 1909 and is defined in epidemiological modeling as the average number of secondary infections produced by a single infected individual introduced into a completely susceptible population. The basic reproduction

number, denoted by R_0, measures the transmission potential of a disease. For example, if R_0 for measles in a population is 12, we would expect each new case of measles to produce 12 new secondary cases, assuming all contacts are susceptible. Note that R_0 excludes new cases produced by secondary infections.

R_0 is one of the most important parameters in epidemiology, as it determines whether an infection will die out or persist in the population. This threshold parameter provides essential information about the spread of a disease and the effectiveness of control measures. In epidemic models, it is a key threshold that determines the disease outcome (Martcheva, 2015; J. Murray, 2004). The basic reproduction number can be expressed in several equivalent ways:

$$R_0 = \text{(Contact rate)} \times \text{(Number of susceptibles at DFE)} \times \text{(Average residence time in the infected class).}$$

or

$$R_0 = \text{(Rate of secondary infection)} \times \text{(Duration of infection)}$$

or, in a simple compartmental model,

$$R_0 = \frac{\text{Contact rate}}{\text{Recovery rate}} = \frac{\beta}{\gamma}.$$

If $R_0 < 1$, the infection cannot grow and will die out from the population, resulting in a disease-free equilibrium. Conversely, if $R_0 > 1$,

the disease-free equilibrium is unstable, and the infection can spread and persist in the population, leading to an endemic equilibrium.

An epidemic occurs when $R_0 > 1$. Therefore, to control or eliminate an epidemic, public health interventions aim to reduce R_0 below one.

Effective Reproduction Number

In reality, populations are rarely entirely susceptible to an infection. Some individuals may be immune due to prior infection conferring long-term immunity, or previous vaccination, or other protective measures. Consequently, the average number of secondary cases per infectious individual is lower than the basic reproduction number.

The effective reproduction number, denoted by R_e, represents the average number of secondary cases per infectious case in a population consisting of both susceptible and non-susceptible individuals. It reflects the number of people that an infected individual can infect at a given time. R_e changes over time as the population becomes increasingly immune, either through natural infection, vaccination, or other protective interventions such as Yoga or Yogachara practices.

The next-generation matrix method is commonly used to calculate the reproduction number.

Next Generation Matrix

In complex epidemic models, it is often difficult to compute the reproduction number directly from its definition. In such cases, the next generation matrix method, introduced by Van den Driessche and Watmough (2002), provides a systematic approach. Here, we present a general framework for computing the reproduction number.

Let $X = (X_1, X_2, \ldots, X_n)$ represents the state of individuals in all compartments, where $X_i \geq 0$ for each i. These compartments are divided into two categories: the first includes infected compartments ($i = 1, 2, \ldots, m$), and the second includes uninfected compartments ($i = m+1, m+2, \ldots, n$). Let X_s denote the set of all disease-free states, defined as

$$X_s = \{X : X_i = 0, \forall i = 1, 2, \ldots, m\}.$$

Define

$\mathcal{F}_i(X) =$ rate of new infections in the i-th compartment,

$\mathcal{V}_i^+(X) =$ rate of inflow into the i-th compartment by other means ,

$\mathcal{V}_i^-(X) =$ rate of outflow from the i-th compartment.

Then, the epidemic model can be expressed as the system of ordinary differential equations

$$\frac{dX_i}{dt} = \mathcal{F}_i(X) - \mathcal{V}_i(X), \quad i = 1, 2, \ldots, n, \tag{2.17}$$

where $\mathcal{V}_i(X) = \mathcal{V}_i^-(X) - \mathcal{V}_i^+(X)$.

At the disease-free equilibrium X_0, the Jacobian matrices are

$$D_X\mathcal{F}(X_0) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \quad D_X\mathcal{V}(X_0) = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix},$$

where F and V are $m \times m$ matrices defined by

$$F = \left[\frac{\partial \mathcal{F}_i(X_0)}{\partial X_j}\right], \quad V = \left[\frac{\partial \mathcal{V}_i(X_0)}{\partial X_j}\right], \quad 1 \leq i, j \leq m.$$

Here, F is a non-negative matrix, and V is non-singular. The matrix FV^{-1} is known as the *next generation matrix*, and the basic reproduction number R_0 is given by its spectral radius (i.e., the largest absolute value of its eigenvalues):

$$R_0 = \rho(FV^{-1}).$$

This method will be used to compute the reproduction number in subsequent chapters.

Incidence in Epidemic Models

The term *incidence rate* refers to the rate at which new cases of a disease occur over a specified period. It is defined as the ratio of the number of new cases during a given period to the population at risk. This measure is widely used in epidemiology to quantify the occurrence of disease, illness, or accidents. Importantly, incidence rate considers only new cases

rather than previously diagnosed or reported ones. It helps public health experts anticipate future events and plan interventions accordingly.

In epidemic models, the incidence rate represents the rate at which susceptible individuals become infected. Several forms of incidence rates have been studied, including mass action (bilinear), standard, saturated, specific nonlinear, and general incidence rates. In the following, we provide a brief discussion of some of the commonly used incidence rates.

Disease Transmission Rate

Infectious diseases are typically transmitted through direct contact. The number of individuals contacted by an infective per unit time is called the *contact rate*, denoted by $P(N)$, which depends on the total population N. If a susceptible individual is contacted by an infective, they may become infected. Let β_0 denote the probability of transmission per contact. Then, the product $\beta_0 P(N)$, called the *adequate contact rate*, describes the infection potential of infectives and typically depends on pathogen characteristics and environmental factors.

The effective contact rate between susceptible and infected individuals, denoted by β, is given by:

$$\begin{aligned}\beta &= (\text{average number of contacts per susceptible per unit time}) \\ &\quad \times (\text{probability of transmission per contact}) \\ &= P(N)\,\beta_0.\end{aligned}$$

Example 2.1. *If an individual has on average 10 contacts per day and*

the probability of disease transmission per contact is 0.2*, then*

$$\beta = 10 \times 0.2 = 2 \textit{ infections per day per susceptible individual.}$$

The unit of β is derived from the components: the average number of contacts per susceptible per unit time (e.g., contacts/day) and the dimensionless probability of transmission. Hence, the unit of β is contacts per unit time.

Example 2.2. *If the average number of contacts per susceptible per week is 50 and the probability of transmission per contact is 0.2, then*

$$\beta = 50 \times 0.2 = 10 \textit{ contacts/week.}$$

The unit of β *remains contacts/week.*

Key points regarding β:

- A higher β indicates a higher likelihood of disease transmission, assuming other factors remain constant. Factors influencing β include population density, individual behavior, interventions, and immunity levels.
- β may vary over time in response to interventions, awareness, or behavioral changes.
- In models such as SIR, β is used with other parameters like the infectious period and susceptible population to simulate disease spread.

The infection rate, representing the number of new infectives per unit time, is given by $\beta_0 P(N)\frac{S}{N}$, and the total number of new infectives in the infected compartment is

$$\text{Incidence} = \beta_0 P(N)\frac{SI}{N}.$$

Force of Infection

The *force of infection* quantifies the rate at which susceptible individuals become infected. It measures the risk of infection per susceptible per unit time based on current epidemiological conditions.

Example 2.3. *Consider a population of* $150,000$*, from which a sample of* $2,000$ *is surveyed. If* 100 *individuals are infected over one year:*

1. *Infection proportion in the sample:* $\frac{100}{2000} = 0.05$.
2. *Estimated infected individuals in population:* $0.05 \times 150,000 = 7,500$.
3. *Force of infection per day:* $\frac{7,500}{150,000\times 365} = 0.000137$ *infections/person/day.*
4. *Daily transmission rate assuming all individuals are initially susceptible ($S \approx N$):*

$$\beta = \frac{0.000137}{S/N} = 0.000137 \text{ infections/person/day.}$$

5. *Transmission rate per year:* $0.000137\times 365 = 0.05$ *infections/person/year.*

6. *If only 50,000 are susceptible,* $\beta = \frac{0.000137}{50{,}000/150{,}000} = 0.000411$ *infections/person/day = 0.15 infections/person/year.*

Types of Incidence

Bilinear Incidence. If the contact rate is proportional to population size ($P(N) = kN$), the incidence is given by βIS, where $\beta = \beta_0 k$ is the transmission coefficient. This is called bilinear incidence or simple mass action incidence. It assumes homogeneous mixing and is often suitable for small populations or airborne diseases. In large, heterogeneous populations, this assumption may not be realistic.

Standard Incidence. If the contact rate is constant ($P(N) = k$), the incidence is

$$\text{Incidence} = \frac{\beta SI}{N}, \quad \beta = \beta_0 k.$$

Here, S/N and I/N represent the fractions of susceptible and infectious individuals. Standard incidence normalizes the bilinear incidence by population size and is suitable for large or variable populations.

Comparative Analysis: Bilinear vs. Standard Incidence.

- **Population Size:** Bilinear incidence does not account for N, potentially overestimating spread in large populations. Standard incidence adjusts for N.
- **Contact Dynamics:** Standard incidence accounts for reduced effective contact in larger populations.

- **Applicability:** Bilinear is suited for small, isolated populations; standard incidence is better for large, heterogeneous populations.

- **Example:** With $\beta = 0.3$, $\gamma = 0.1$, $S = 999$, $I = 1$, $N = 1000$:

  Bilinear: $\frac{dI}{dt} = \beta SI - \gamma I = 299.6$, Standard: $\frac{dI}{dt} = \frac{\beta SI}{N} - \gamma I = 0.1996$.

Saturated Incidence. Saturated incidence accounts for limitations in effective contacts due to behavioral changes, healthcare capacity, or crowding. Common forms include:

$$\frac{\beta SI}{1 + \alpha S}, \quad \frac{\beta SI}{1 + \alpha I}.$$

Saturation limits the unbounded growth of infections, introducing nonlinear effects.

Nonlinear Incidence. Nonlinear incidence rates, such as $\beta I^p S^q$ (W.-m. Liu, Hethcote, & Levin, 1987; W.-m. Liu, Levin, & Iwasa, 1986), capture more complex interactions, allow multiple equilibria, and reflect realistic epidemic dynamics.

Non-monotonic Incidence. Non-monotonic incidence rates, e.g.,

$$g(I)S = \frac{\beta IS}{1 + \alpha I^2},$$

reflect scenarios where infection rates increase initially but decrease at high infection levels due to behavioral changes, protective measures, or resource constraints (Capasso & Serio, 1978). Other examples include

logistic, oscillatory, threshold-based, and Holling type III incidence rates.

Nonlinear and non-monotonic incidence rates provide richer frameworks for modeling disease dynamics, capturing realistic behaviors and intervention effects. These incidence rates are applied in subsequent chapters.

Chapter 3

Analysis of an SYIRS Epidemic Model with Yoga

Preamble

Mathematical modeling has been used to determine the transmission dynamics of epidemics and to evaluate the effectiveness of control strategies in preventing communicable diseases. The spread of a contagious disease is often accompanied by an increase in preventive measures such as yoga (pranayama) and heightened awareness within the social vicinity of infected individuals, leading to subsequent behavioral changes. Such reactions can manifest as reduced susceptibility, as people take precautions to avoid infection. They may also lower infectivity due to self-imposed behavioral changes and improved hygiene.

A compartmental model and its analysis have been employed, incorporating not only infectious agents but also environmental factors and human perceptions, culture, and behavior. Such models can support decision-making processes in epidemiology and public health. Epidemiological models governed by ordinary differential equations help provide insight into the dynamics of infectious diseases. Accordingly, we have formulated an SIR epidemic model to capture disease situations in soci-

ety, apply control strategies, and predict disease propagation.

Regular yoga practice and the implementation of integrated yoga programs can help boost immunity and improve the psychological health of individuals. Yoga has also been found beneficial in managing respiratory infectious diseases and enhancing lung capacity. There is substantial evidence suggesting the role of yoga in reducing the severity of infections by regulating immune responses. Today, yoga is recommended as an immunity-strengthening practice that helps to improve physical function and overall quality of life. The continuous practice of yoga (ranayama) contributes to better health conditions and may aid in the control of communicable diseases.

This chapter includes the formulation of the model, analysis of equilibrium points, derivation of the reproduction number, examination of the stability of disease-free and endemic equilibria, numerical simulation, and presentation of the results, followed by a concise discussion and conclusion.

Assumptions and Model Development

Data Source

Data for this research were collected from 20 yoga centres located in the Sudurpaschim Province of Nepal. A total of 1,982 Yoga Sādhaka (practitioners) participated in the survey. Participants responded to structured questions designed to determine whether they had been infected with

any communicable disease (e.g., influenza, COVID-19, or other febrile illnesses) between March 2021 and May 2022. Their responses were analyzed, and the efficacy of yoga practice was evaluated. Secondary data were also used from sources listed in Table 3.2.

In addition, data were obtained from articles published during the COVID-19 lockdown period. The Sudurpaschim Province implemented lockdown measures from April 29, 2020, to September 1, 2022. The number of Yoga Sādhaka (yoga teachers or practitioners) during this period was recorded, and the rate of increase in the number of practitioners was calculated.

Additional secondary data were obtained from the *Annual Health Report 2079/80* published by the Ministry of Health and Population (MoHP), Teku, Kathmandu, Nepal.

Parameter Estimation

This model utilized real-world yoga coverage data (primary data) collected from 20 yoga centres in the Sudurpaschim Province, along with secondary data as presented in Table 3.2.

The disease transmission rate among Yoga Sādhaka individuals, denoted by ρ_1, was estimated by fitting the model to the observed data. Its value was calculated directly by taking the percentage of infected Yoga Sādhaka individuals from real data and applying the relation

$$\rho_1 = \frac{\text{Force of infection}}{\frac{S}{N}},$$

where S represents the number of susceptible individuals and N denotes the total population size. The force of infection is defined as

$$\text{Force of infection} = \frac{\text{Total infected individuals}}{n \times T},$$

where n is the sample size and T is the time period (1 year = 365 days).

The yoga efficacy, σ, was calculated using the relation $\rho_1 = \beta(1 - \sigma)$, where β denotes the control group transmission rate, taken as 0.37 (the average of $\beta_1 = 0.125$ and $\beta_2 = 0.615$; (Chanda, Adhikar, Gautam, Pokharel, & Uprety, 2023).

Alternatively, yoga efficacy can be determined statistically using the formula

$$\text{Yoga efficacy} = \left(1 - \frac{\text{Yoga Sādhaka infection rate}}{\text{Non–Yoga Sādhaka infection rate}}\right) \times 100\%.$$

That is,

$$\sigma = \left(1 - \frac{r_1}{r_2}\right) = \left(1 - \frac{0.047}{0.37}\right) = 0.87.$$

Hence, the value of σ was taken to lie between 0.60 and 0.90. Other parameter values were used as provided in Table 3.2.

The number of Yoga Sādhaka individuals trained by the authority during the year 2020/2021 was recorded. These individuals conducted yoga classes, and the increase in the number of Yoga Sādhaka (yoga

practitioners) was also collected from each centre. The difference between the number of Yoga Sādhaka recorded at the end of March 2023 and the baseline number in 2020/2021 was calculated. From this difference, the Yoga Sādhaka increase rate, denoted by m, was derived from the real data.

Model Formulation

A deterministic compartmental model incorporating a Yoga Pranayama compartment is developed to describe the transmission dynamics of infection within a population. The total population, denoted by N, is divided into four mutually exclusive classes:

- S_1: Naïve susceptible individuals (non–Yoga Sādhaka susceptibles);
- Y_1: Yoga Sādhaka individuals who regularly practice Pranayama;
- I_1: Infected individuals, including both Yoga Sādhaka and non–Sādhaka infectives; and
- R_1: Recovered individuals.

The model assumes a constant total population size, maintained through equal natural birth and natural death rates. Hence, the overall population remains constant at size N. It is further assumed that interactions among the compartments follow the law of mass action.

Transitions Between Compartments. Individuals in the non–Yoga Sādhaka susceptible class (S_1) may shift to the Yoga Sādhaka class (Y_1)

at a rate m. In addition, naïve susceptible individuals (S_1) may acquire infection and move to the infected class (I_1). The incidence rate, representing new infections among susceptibles due to contact with infectives, is given by

$$\frac{\beta S_1 I_1}{N},$$

where β is the transmission rate of infection among non–Yoga Sādhaka individuals.

Effect of Yoga Pranayama on Immunity. It is assumed that Yoga Pranayama provides partial protection against natural infection, leading to the formation of a class of Yoga Sādhaka susceptibles (Y_1) who possess an enhanced immunological response. These individuals are capable of eliciting a quicker immune response upon exposure, thereby reducing their susceptibility to infection compared to the naïve susceptibles (S_1). Consequently, the number of new infections produced through adequate contact among Yoga Sādhaka individuals is expressed as

$$\frac{\rho_1 Y_1 I_1}{N},$$

where $\rho_1 = (1 - \sigma)\beta$ denotes the effective transmission rate within the Yoga Sādhaka class, and $0 \leq \sigma \leq 1$.

The parameter σ represents the efficacy of immunity induced by Yoga Pranayama. Specifically, $\sigma = 0$ implies that Yoga Pranayama has no protective effect, whereas $\sigma = 1$ indicates complete immunity and long-lasting protection against infection. In practical scenarios, Yoga

Pranayama is assumed to induce an immune response that enhances resistance to infection but does not confer permanent immunity, that is, $0 < \sigma < 1$. The induced immunity is assumed to wane over time at a per capita rate ρ_2, reflecting the gradual loss of immune protection if the practice of Pranayama is discontinued. Hence, recovered individuals may eventually return to the susceptible class. The average duration of immune protection, either from Yoga Pranayama or natural infection, is given by

$$d_1 = \frac{1}{\rho_2}.$$

Although theoretically the duration of immunity may tend toward infinity, in reality, it remains finite.

Recovery dynamics. Infectious individuals are assumed to recover at a constant rate $\gamma > 0$. The recovery period for Yoga Sādhaka infectives is typically shorter than that for non–Yoga Sādhaka individuals; however, an average recovery rate is used for simplicity. Recovered individuals gradually lose protection and return to the susceptible class (S_1). Finally, it is assumed that individuals may either enter the Yoga Sādhaka class (Y_1) at the same rate m or remain in the susceptible class (S_1). The notations used in the model are summarized in Table 3.1.

Variables/Parameter	Description
$S_1(t)$	Naïve susceptible individuals at time t
$Y_1(t)$	Yoga Sādhaka individuals (Pranayama class) at time t
$I_1(t)$	Infected (infectious) individuals at time t
$R_1(t)$	Recovered individuals at time t
Λ	Recruitment rate of individuals into the population
β	Disease transmission rate (contact rate)
σ	Yoga Pranayama efficacy ($\sigma \in [0, 1]$)
γ	Recovery rate
m	Rate at which susceptible individuals become Yoga Sādhaka
ρ_1	Disease transmission rate in Yoga Sādhaka individuals, (i.e., $\rho_1 = (1 - \sigma)\beta$)
ρ_2	Rate of waning immunity induced by natural infection
r_1	Infection rate among Yoga Sādhaka individuals
r_2	Infection rate among non–Yoga Sādhaka individuals
r_3	Infection rate among non–Yoga Sādhaka individuals for influenza-like illness (2022/2023)

Table 3.1: Description of variables and parameters used in the model.

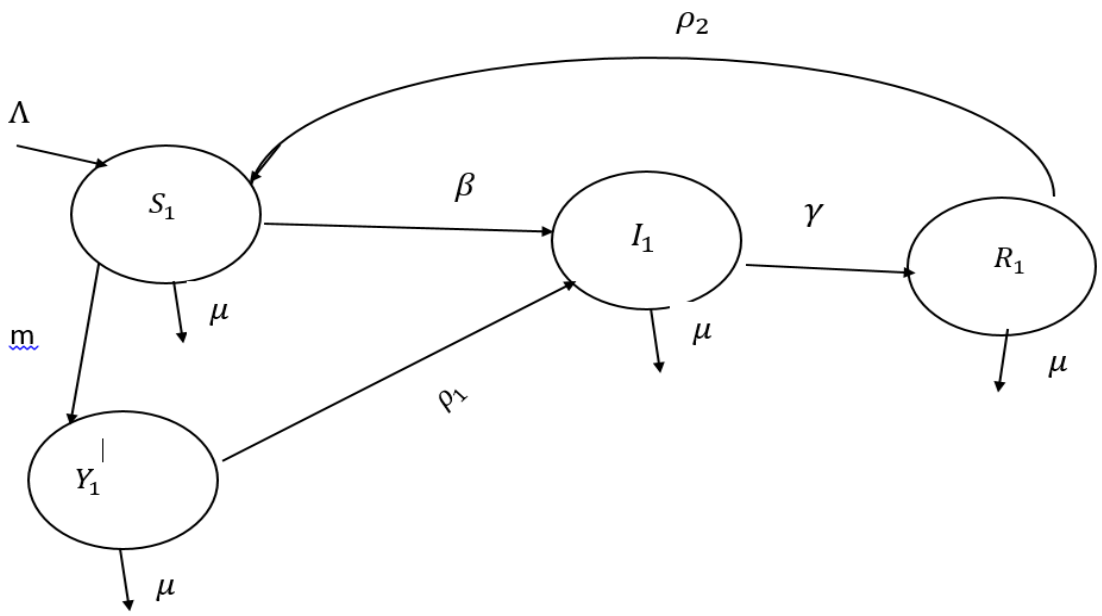


Figure 3.1: Transfer diagram for the system (3.1) representing the SYIRS model.

The transfer diagram of the model is shown in Figure 3.1.

The model is governed by following system of ordinary differential

equations:

$$\begin{aligned}
\frac{dS_1}{dt} &= \Lambda N - \frac{\beta S_1 I_1}{N} - (m+\mu)S_1 + \rho_2 R_1, \\
\frac{dY_1}{dt} &= mS_1 - \frac{\rho_1 Y_1 I_1}{N} - \mu Y_1, \\
\frac{dI_1}{dt} &= \frac{\beta S_1 I_1}{N} + \frac{\rho_1 Y_1 I_1}{N} - (\gamma+\mu)I_1, \\
\frac{dR_1}{dt} &= \gamma I_1 - (\rho_2+\mu)R_1,
\end{aligned} \tag{3.1}$$

with non-negative initial conditions $N(0) > 0$ and $S_1 + Y_1 + I_1 + R_1 = N$. The demographic equation assumes that the total population size remains constant because the birth rate is equal to the death rate. Since the model is homogeneous of degree one, the variables can be normalized by dividing by the total population size N, as follows:

$$S = \frac{S_1}{N}, \quad Y = \frac{Y_1}{N}, \quad I = \frac{I_1}{N}, \quad R = \frac{R_1}{N}.$$

This normalization leads to the following system of equations for the proportions of the population:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - (m+\mu)S + \rho_2 R, \\
\frac{dY}{dt} &= mS - \rho_1 YI - \mu Y, \\
\frac{dI}{dt} &= \beta SI + \rho_1 YI - (\gamma+\mu)I, \\
\frac{dR}{dt} &= \gamma I - (\rho_2+\mu)R.
\end{aligned} \tag{3.2}$$

where each variable denotes a fraction of the total individuals, so that $S + Y + I + R = 1$.

Results and Discussion

Positivity and Boundedness

To investigate the positivity and boundedness of the model, the following lemmas and theorems are considered (Rwezaura, Mtisi, & Tchuenche, 2010).

Lemma 3.1. *The feasible set of system (3.1) is given by*

$$\Omega = \{(S_1, Y_1, I_1, R_1) \in \mathbf{R}_+^4 : S_1 + Y_1 + I_1 + R_1 \leq \frac{\Lambda}{\mu}\}.$$

Proof. Adding the differential equations in system (3.1) and using $N = S_1 + Y_1 + I_1 + R_1$ gives

$$\frac{dN}{dt} \leq \Lambda - \mu N.$$

From this, we can write

$$\lim_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}.$$

Hence, the feasible region for system (3.1) is

$$\Omega = \{(S_1, Y_1, I_1, R_1) \in \mathbf{R}_+^4 : S_1 + Y_1 + I_1 + R_1 \leq \frac{\Lambda}{\mu}\}.$$

Thus, the dynamics of system (3.1) and (3.2) will be considered within Ω. □

Lemma 3.2. *The set Ω is positively invariant.*

Proof. Consider the following:

$$
\begin{aligned}
S_1 = 0 \Rightarrow \frac{dS_1}{dt} &= \Lambda + \rho_2 R_1, \\
Y_1 = 0 \Rightarrow \frac{dY_1}{dt} &= mS_1, \\
I_1 = 0 \Rightarrow \frac{dI_1}{dt} &= 0, \\
R_1 = 0 \Rightarrow \frac{dR_1}{dt} &= \gamma I_1.
\end{aligned}
$$

Since all parameters are positive, any vector field on the boundary of Ω is tangent to or directed inward. Therefore, the region Ω is positively invariant, and any solution of system (3.2) with an initial point on the boundary enters the interior of Ω and remains there. □

Lemma 3.3 (Existence)**.** *A solution of model system (3.2) is feasible in* Ω.

Proof. Since system (3.2) is dissipative, all feasible solutions are uniformly bounded in a proper subset $\Omega \subset \mathbf{R}^4_+$. Consequently, any solution with initial values in Ω attains its maximum and remains within the set. Hence, Ω is compact and positively invariant. □

Theorem 3.4. *Let the initial conditions satisfy* $S(0) > 0$, $Y(0) \geq 0$, $I(0) \geq 0$, *and* $R(0) \geq 0$, *where these variables are elements of* Ω. *Then, the solutions* $S(t)$, $Y(t)$, $I(t)$, *and* $R(t)$ *of system (3.2) are positive and bounded for all* $t > 0$.

Proof. System (3.2) describes the dynamics of disease propagation in the human population. Therefore, it is essential to demonstrate that sus-

ceptible, Yoga Sādhaka, infected, and recovered individuals remain non-negative for all time.

Let the initial conditions be $S(0) > 0$, $Y(0) \geq 0$, $I(0) \geq 0$, and $R(0) \geq 0 \in \Omega$. Assume that all parameters are positive.

From system (3.2), we have

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta SI - mS - \mu S + \rho_2 R, \\
\frac{dS}{dt} &\geq -S(\beta I + m + \mu), \\
\frac{dS}{S} &\geq -(\beta I + m + \mu)dt, \\
S &\geq S_0 \exp\left[-\int_0^t (\beta I + m + \mu)dt\right], \\
\Rightarrow S(t) &\geq 0.
\end{aligned}$$

Similarly,

$$\begin{aligned}
\frac{dY}{dt} &= mS - \rho_1 YI - \mu Y, \\
Y &\geq Y_0 \exp\left[-\int_0^t (\rho_1 I + \mu)dt\right] \geq 0, \\
\frac{dI}{dt} &= \beta SI + \rho_1 YI - (\gamma + \mu)I, \\
I &\geq I_0 \exp\left[-\int_0^t (\gamma + \mu)dt\right] \geq 0, \\
R &\geq R_0 \exp\left[-\int_0^t (\rho_2 + \mu)dt\right] \geq 0.
\end{aligned}$$

Adding all equations gives

$$\frac{dS}{dt} + \frac{dY}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = \Lambda - \mu.$$

On integrating and assuming $\mu = \Lambda$, we obtain $S+Y+I+R = 1$. Therefore, the solutions of the system are positive and bounded in the feasible region Ω. □

Equilibrium Points and Yoga Reproduction Number

For the equilibrium points of system (3.2), we have

$$\begin{aligned} \Lambda - \beta SI - (m+\mu)S + \rho_2 R &= 0, \\ mS - \rho_1 YI - \mu Y &= 0, \\ \beta SI + \rho_1 YI - (\gamma+\mu)I &= 0, \\ \gamma I - (\rho_2+\mu)R &= 0. \end{aligned} \tag{3.3}$$

System (3.2) always has a disease-free equilibrium (DFE) point given by

$$E_0 = \left(\frac{\Lambda}{m+\mu}, \frac{m\Lambda}{\mu(m+\mu)}, 0, 0 \right).$$

The Yoga reproduction number, denoted as R_e, is defined as the threshold quantity representing the average number of secondary infections produced by a single infectious individual introduced into a completely susceptible population. The term "Yoga reproduction number" is used because the model incorporates the Yoga Prāṇāyāma process as a control strategy. This parameter is particularly important, as it determines whether an infection will persist or die out in the population. We derive an expression for R_e using the next-generation matrix approach. (Capasso & Serio, 1978; Chukwu, Akinyemi, Adeniyi, & Salawu, 2020;

D. Xiao & Ruan, 2007).

Let $X = (S, Y, I, R)$, then from (2.17),

$$X' = \mathcal{F} - \mathcal{V},$$

where

$$\mathcal{F} = \begin{bmatrix} 0 \\ 0 \\ \beta SI + \rho_1 YI \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} -\Lambda + \beta SI + (m+\mu)S - \rho_2 R \\ -mS + \rho_1 YI + \mu Y \\ (\gamma + \mu)I \\ -\gamma I + (\rho_2 + \mu)R \end{bmatrix}.$$

At the disease-free equilibrium, the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ are given by

$$F_0 = \begin{bmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \beta S_0 + \rho_1 Y_0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V_0 = \begin{bmatrix} m+\mu & 0 & \beta S_0 & -\rho_2 \\ -m & \mu & \rho_1 Y_0 & 0 \\ 0 & 0 & \gamma + \mu & 0 \\ 0 & 0 & -\gamma & \rho_2 + \mu \end{bmatrix}.$$

The next-generation matrix for model (3.2) is given by $\mathbf{F_0 V_0^{-1}}$. The spectral radius (dominant eigenvalue) of $\mathbf{F_0 V_0^{-1}}$ is

$$\rho(\mathbf{F_0 V_0^{-1}}) = \frac{\beta S_0 + \rho_1 Y_0}{\gamma + \mu}.$$

Therefore, the Yoga reproduction number is

$$\begin{aligned} R_e &= \frac{\beta S_0 + \rho_1 Y_0}{\gamma + \mu}, \\ R_e &= \frac{\beta(1 - \sigma Y_0)}{\gamma + \mu}, \\ &= R_0(1 - \sigma Y_0) = \frac{\beta}{\gamma + \mu}\left[\frac{\mu(\mu + m) - m\sigma\Lambda}{\mu(m + \mu)}\right], \text{ where } R_0 = \frac{\beta}{\gamma + \mu}. \end{aligned} \tag{3.4}$$

Initially, $(S_0 + Y_0) = 1$. If there are no Yoga Sādhaka individuals (i.e., $Y_0 = 0$), then $R_e = R_0$.

Endemic Equilibrium. When the disease persists in the population ($I^* \neq 0$), there may exist one or more endemic equilibrium points (EEP), denoted by

$$P^* = (S^*, Y^*, I^*, R^*),$$

which satisfy the following system:

$$\begin{aligned} \Lambda - \beta S^* I^* - (m + \mu)S^* + \rho_2 R^* &= 0, \\ mS^* - (\rho_1 + \mu)Y^* &= 0, \\ \beta S^* I^* + \rho_1 Y^* I^* - (\gamma + \mu)I^* &= 0, \\ \gamma I^* - (\rho_2 + \mu)R^* &= 0. \end{aligned} \tag{3.5}$$

From the fourth and second equations of system (3.5), we obtain

$$R^* = \frac{\gamma I^*}{\rho_2 + \mu}, \quad \text{and} \quad Y^* = \frac{mS^*}{\rho_1 + \mu}.$$

From the third equation of system (3.5), we have

$$I^*[\beta S^* + \rho_1 Y^* - (\gamma + \mu)] = 0.$$

Since $I^* \neq 0$ at endemic equilibrium, using the second and third equations, we get

$$\begin{aligned} \beta S^* + \rho_1 Y^* &= \gamma + \mu, \\ \beta S^* + \rho_1 \frac{mS^*}{\rho_1 + \mu} &= \gamma + \mu. \end{aligned}$$

Solving for S^* gives

$$\begin{aligned} S^* &= \frac{(\mu + \gamma)(\rho_1 + \mu)}{\beta(\rho_1 + \mu) + m\beta(1 - \sigma)} \\ &= \frac{(\mu + \gamma)(\rho_1 + \mu)}{\beta[(1 - \sigma)(\beta + m) + \mu]} \\ &= \frac{(\rho_1 + \mu)}{R_0[(1 - \sigma)(\beta + m) + \mu]}. \end{aligned} \tag{3.6}$$

Also, from the second equation of system (3.5) and the condition $S + Y + I + R = 1$, we obtain

$$Y^* = \frac{m(\mu + \gamma)(\rho_1 + \mu)}{\beta(\rho_1 + \mu)[(1 - \sigma)(\beta + m) + \mu]}. \tag{3.7}$$

Finally, the equilibrium value of I^* is given by

$$I^* = \frac{(\rho_2 + \mu)\Big[\beta\big((1 - \sigma)(\beta + m) + \mu\big) - m(\mu + \gamma)(\rho_1 + m + \mu)\Big]}{\beta(\gamma + \rho_2 + \mu)\big((1 - \sigma)(\beta + m) + \mu\big)}. \tag{3.8}$$

Stability Analysis

In this section, the local and global stability analyses of the model are presented under both the disease-free equilibrium (DFE) and the endemic equilibrium conditions.

Local Stability Analysis. Local stability describes the behavior of the system near an equilibrium point. It determines whether small perturbations, such as a few additional infected individuals or slight changes in the number of Yoga Sādhaka practitioners, decay over time or amplify. If an equilibrium is locally asymptotically stable, small deviations diminish and the system returns to equilibrium. For the disease-free equilibrium

$$E_0 = (S_0, Y_0, 0, 0),$$

local stability implies that a small number of infections introduced into a population with otherwise constant susceptible and Yoga Sādhaka individuals will die out over time, provided the Yoga reproduction number satisfies $R_e < 1$. For the endemic equilibrium

$$E^* = (S^*, Y^*, I^*, R^*),$$

local stability means that if the system is slightly perturbed (e.g., small fluctuations in infected or Yoga Sādhaka individuals), it will return to the endemic state, as long as $R_e > 1$. The variational matrix method is employed to analyze the stability of both the disease-free and endemic

equilibrium points, as established in the following theorems.

Theorem 3.5. ***(Local Stability of the Disease-Free Equilibrium)*** *The disease-free equilibrium, denoted by E_0, is locally asymptotically stable if $R_e < 1$ and unstable otherwise.*

Proof. Consider the following system of functions:

$$\begin{aligned} f_1 &\equiv \Lambda - \beta SI - (m+\mu)S + \rho_2 R, \\ f_2 &\equiv mS - \rho_1 YI - \mu Y, \\ f_3 &\equiv \beta SI + \rho_1 YI - (\gamma+\mu)I, \\ f_4 &\equiv \gamma I - (\rho_2+\mu)R. \end{aligned}$$

The variational matrix $V(E)$ at an arbitrary point E of the system (3.2) is given by

$$V(E) = \begin{bmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial Y} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial Y} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial Y} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial Y} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial R} \end{bmatrix}.$$

Hence,

$$V(E) = \begin{bmatrix} -\beta I - m - \mu & 0 & -\beta S & \rho_2 \\ m & -\mu - \rho_1 I & -\rho_1 Y & 0 \\ \beta I & \rho_1 I & \beta S + \rho_1 Y - \gamma - \mu & 0 \\ 0 & 0 & \gamma & -\rho_2 - \mu \end{bmatrix}.$$

In the absence of infection ($I = 0$), the equilibrium condition represents the disease-free equilibrium. Thus, the variational matrix at the DFE, E_0, can be written as

$$V(E_0) = \begin{bmatrix} -\mu - m & 0 & -\beta S_0 & \rho_2 \\ m & -\mu & \rho_1 Y_0 & 0 \\ 0 & 0 & \beta S_0 + \rho_1 Y_0 - \gamma - \mu & 0 \\ 0 & 0 & \gamma & -\rho_2 - \mu \end{bmatrix}.$$

Here, S_0 and Y_0 denote the initial numbers of susceptible and Yoga Sadhaka individuals, respectively. At the DFE point,

$$E_0 = (S_0, Y_0, 0, 0) = \left(\frac{\Lambda}{m+\mu}, \frac{m\Lambda}{\mu(\mu+m)}, 0, 0 \right),$$

the characteristic polynomial of $V(E_0)$ is given by

$$\det(V(E_0) - KI) = 0.$$

This expands to

$$\begin{vmatrix} -\mu - m - K & 0 & -\beta S_0 & \rho_2 \\ m & -\mu - K & \rho_1 Y_0 & 0 \\ 0 & 0 & \beta S_0 + \rho_1 Y_0 - \gamma - \mu - K & 0 \\ 0 & 0 & \gamma & -\rho_2 - \mu - K \end{vmatrix} = 0.$$

Simplifying yields

$$(K+\mu)(K+\mu+m)(K+\rho_2+\mu)(K+\mu+\gamma-\beta S_0-\rho_1 Y_0)=0.$$

Therefore, the eigenvalues are

$$K_1=-\mu, \quad K_2=-\mu-m, \quad K_3=-\mu-\rho_2,$$
$$K_4=\beta S_0+\rho_1 Y_0-\mu-\gamma=\beta(1-\sigma Y_0)-\mu-\gamma.$$

Since K_1, K_2, and K_3 are negative, the system is stable if and only if $K_4<0$, that is,

$$\begin{aligned}\beta(1-\sigma Y_0)-\mu-\gamma &< 0,\\ \frac{\beta(1-\sigma Y_0)}{\mu+\gamma} &< 1,\\ R_e &< 1.\end{aligned}$$

Thus, all eigenvalues of $V(E_0)$ have negative real parts whenever $R_e<1$. Consequently, the DFE E_0 of system (3.2) is locally asymptotically stable if $R_e<1$ and unstable when $R_e>1$. □

Theorem 3.6. ***(Local Stability of the Endemic Equilibrium)***

The endemic equilibrium E^* is locally asymptotically stable in Ω if $R_e>1$.

Proof. The variational matrix at the endemic equilibrium E^* is given by

$$V(E^*) = \begin{bmatrix} -\beta I^* - m - \mu & 0 & -\beta S^* & \rho_2 \\ m & -\mu - \rho_1 I^* & -\rho_1 Y^* & 0 \\ \beta I^* & \rho_1 I^* & \beta S^* + \rho_1 Y^* - \gamma - \mu & 0 \\ 0 & 0 & \gamma & -\rho_2 - \mu \end{bmatrix}.$$

Let

$$V(E^*) = \begin{bmatrix} x_{11} & 0 & x_{13} & x_{14} \\ x_{21} & x_{22} & x_{23} & 0 \\ x_{31} & 0 & x_{33} & 0 \\ 0 & 0 & x_{43} & x_{44} \end{bmatrix},$$

where

$$x_{11} = -\beta I^* - \mu - m, \quad x_{13} = -\beta S^*, \quad x_{14} = \rho_2,$$

$$x_{21} = m, \quad x_{22} = -\mu - \rho_1 I^*, \quad x_{23} = \rho_1 Y^*,$$

$$x_{31} = \beta I^*, \quad x_{33} = \beta S^* + \rho_1 Y^* - \gamma - \mu, \quad x_{43} = \gamma,$$

$$x_{44} = -\rho_2 - \mu.$$

The characteristic equation of system (3.2) at E^* is

$$\det(V(E^*) - KI) = 0,$$

or equivalently,

$$\begin{vmatrix} x_{11}-K & 0 & x_{13} & x_{14} \\ x_{21} & x_{22}-K & x_{23} & 0 \\ x_{31} & 0 & x_{33}-K & 0 \\ 0 & 0 & x_{43} & x_{44}-K \end{vmatrix} = 0,$$

where I is the identity matrix of order 4, and K is the eigenvalue of $V(E^*)$.

By expansion, the characteristic equation is expressed as

$$K^4 + c_1K^3 + c_2K^2 + c_3K + c_4 = 0,$$

where

$$\begin{aligned} c_1 &= -\frac{1}{2}(x_{11}+x_{22}+x_{33}+x_{44}), \\ c_2 &= \frac{1}{4}\big[x_{11}(x_{22}+x_{33}+x_{44}) + x_{22}(x_{33}+x_{44}) + x_{33}x_{44} - x_{13}x_{31}\big], \\ c_3 &= \frac{1}{8}\big[x_{13}x_{22}x_{31} - x_{11}x_{22}x_{33} - x_{14}x_{31}x_{43} - x_{11}x_{22}x_{44} \\ &\quad + x_{13}x_{31}x_{44} - x_{11}x_{33}x_{44} - x_{22}x_{33}x_{44}\big], \\ c_4 &= x_{14}x_{22}x_{31}x_{43} - x_{13}x_{22}x_{31}x_{44} + x_{11}x_{22}x_{33}x_{44}. \end{aligned}$$

It is evident that $c_1, c_2, c_3 > 0$ and $c_1c_2 - c_3 > 0$. When $R_e > 1$, the endemic equilibrium point exists. According to the Routh–Hurwitz criterion, all eigenvalues of the characteristic equation possess negative real

parts if the following conditions are satisfied:

$$c_i > 0\ (i = 1, 2, 3, 4), \quad (c_1c_2 - c_3) > 0, \quad c_1c_2c_3 - c_3^2 - c_1^2c_4 > 0.$$

Therefore, the endemic equilibrium E^* is locally asymptotically stable if $R_e > 1$ and the above conditions hold. □

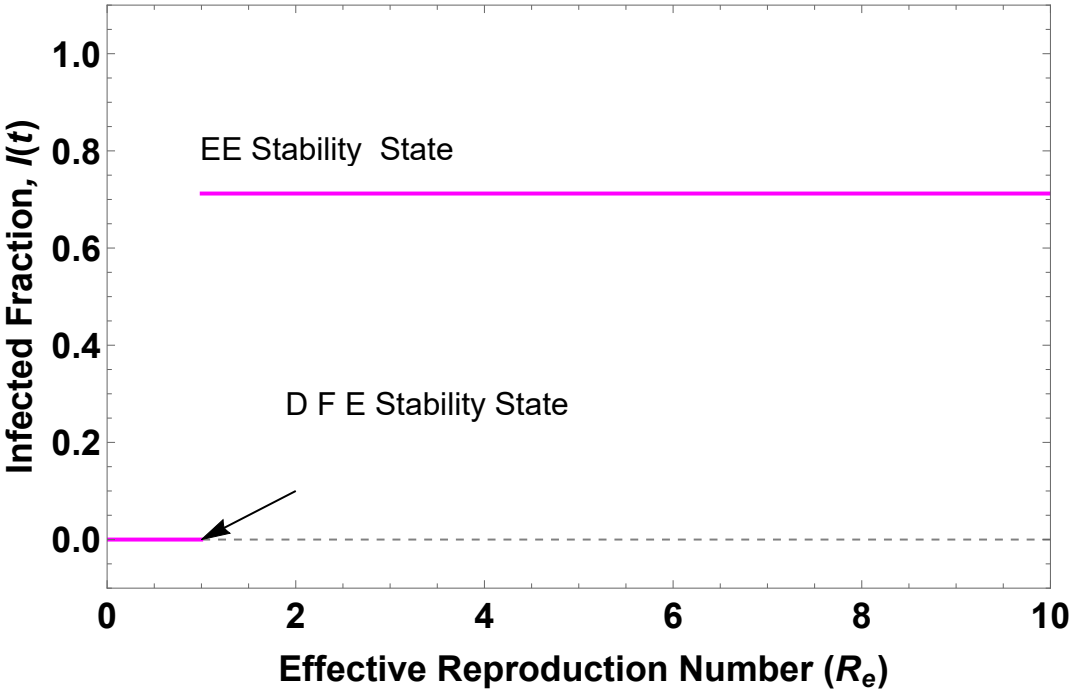


Figure 3.2: Disease-free and endemic equilibrium states.

Global Stability Analysis. In mathematical epidemiology, the concept of global stability describes the long-term behavior of a dynamical system, regardless of initial conditions, within a biologically feasible region. A disease-free equilibrium is considered globally asymptotically stable if, for all initial conditions within the model's domain, the system trajectories approach the equilibrium as time tends to infinity. This indicates that the disease will eventually die out, regardless of the initial

number of infected individuals. Conversely, an endemic equilibrium is globally asymptotically stable if, for any initial condition with a positive number of infected individuals, the trajectories of the system converge to a steady endemic state.

To establish global stability, Lyapunov functions and LaSalle's invariance principle are typically employed for the disease-free equilibrium, while the Dulac–Bendixson criterion and the Poincaré–Bendixson theorem are often used to exclude periodic solutions and demonstrate convergence toward the endemic equilibrium. These mathematical tools provide a rigorous theoretical foundation for determining whether an infection persists or dies out in the population.

Theorem 3.7. *If $R_e < 1$, then the disease-free equilibrium E_0 of system (3.2) is globally asymptotically stable in the region Ω. If $R_e > 1$, then the endemic equilibrium E^* is globally asymptotically stable in the region $\Omega - \{(S, Y, I, R) : I = 0\}$.*

Proof. First, we prove the global stability of system (3.2) at the disease-free equilibrium E_0 when $R_e < 1$. Consider the Lyapunov function $L = I$. Then,

$$\begin{aligned}
\frac{dL}{dt} &= \frac{dI}{dt} \\
&= [\beta S + \rho_1 Y - \mu - \gamma]I \\
&\leq [\beta S_0 + \rho_1 Y_0 - \mu - \gamma]I \\
&\leq 0.
\end{aligned}$$

Thus, if $R_e < 1$, then $\frac{dL}{dt} \leq 0$, and $\frac{dL}{dt} = 0$ if and only if $I = 0$. In this case, $S \to \frac{\Lambda}{\mu+m}$, $Y \to \frac{m\Lambda}{\mu(\mu+m)}$, and $R \to 0$. Therefore, the largest positively invariant set in $\{(S, Y, I, R) \in \Omega : \frac{dL}{dt} = 0\}$ is the singleton set $\{E_0\}$. By LaSalle's invariance principle, $E_0 = \left(\frac{\Lambda}{\mu+m}, \frac{m\Lambda}{\mu(\mu+m)}, 0, 0\right)$ is globally asymptotically stable in Ω.

Since the system is bounded in Ω, we now prove the global stability of E^* when $R_e > 1$, and the solutions of (3.2) lie in the region Ω.

The second and third equations of system (3.2) are independent of R. Therefore, in the positive quadrant of the YI-plane, we apply Dulac's criterion with the multiplier $D = \frac{1}{I}$.

Let

$$F_1 = mS - \rho_1 YI - \mu Y,$$
$$F_2 = [\beta S + \rho_1 Y - (\gamma + \mu)]I.$$

Then,

$$DF_1 = \frac{mS}{I} - \rho_1 Y - \mu\frac{Y}{I},$$
$$DF_2 = [\beta S + \rho_1 Y - (\gamma + \mu)].$$

Hence,

$$\frac{\partial(DF_1)}{\partial Y} + \frac{\partial(DF_2)}{\partial I} = -\rho_1 - \frac{\mu}{I} < 0.$$

Therefore, there is no periodic solution in the region Ω. By the Poincaré

Bendixson theorem, all solutions starting in the positive quadrant of the YI-plane with $I > 0$ and $Y + I \leq \frac{m\Lambda}{\mu(\mu+m)}$ approach (Y^*, I^*) as $t \to \infty$. In this case, the limiting form of the fourth equation of system (3.2),

$$\frac{dR}{dt} = \gamma I - (\rho_2 + \mu)R,$$

shows that $R \to R^*$. Similarly, the limiting form of the first equation of system (3.2),

$$\frac{dS}{dt} = \Lambda - \beta SI - (m + \mu)S + \rho_2 R,$$

shows that $S \to S^*$. Thus, the endemic equilibrium $E^* = (S^*, Y^*, I^*, R^*)$ is globally asymptotically stable in the region $\Omega - \{(S, Y, I, R) : I = 0\}$ for the original system (3.2). □

Sensitivity Analysis and Numerical Results

Sensitivity analysis tells us which parameters are most influential on the outcome. This helps prioritize interventions. If the model is highly sensitive to σ, improving yoga practices will significantly reduce disease spread.

Sensitivity Analysis. The model is numerically analyzed using the parameter values listed in Table 3.2. Sensitivity analysis evaluates the response of model outputs to changes in parameter values, which helps prioritize resources for follow-up experiments and field studies. It quantifies the effect of individual parameters on model outputs. The sensitivity

Parameter	Value (per unit time)	Reference
μ	$[0.1, 0.5]$	Chanda et al. (2023); Musa et al. (2021)
β	$[0.125, 0.615]$	Chanda et al. (2023); Musa et al. (2021)
σ	$0 < \sigma < 1$	Calculated
γ	$[0, 1]$	Chanda et al. (2023); Musa et al. (2021)
Λ	0.3	Chanda et al. (2023); Musa et al. (2021)
m	0.70	Calculated
ρ_1	$\rho_1 = \beta(1 - \sigma)$	Calculated
ρ_2	[0.08, 0.1]	Chanda et al. (2023); Musa et al. (2021)
r_1	0.047	Calculated
r_2	0.37	Chanda et al. (2023)
r_3	0.08	Calculated (Annual Health Report 2022/2023)

Table 3.2: Parameter values used in simulation

index of a parameter P with respect to the reproduction number R_e is defined as

$$S_P^{R_e} = \frac{\partial R_e}{\partial P}\frac{P}{R_e}.$$

(a) **Sensitivity of β on R_e:**

$$S_\beta^{R_e} = \frac{\partial R_e}{\partial \beta}\frac{\beta}{R_e} = 1$$

This indicates a direct proportionality between R_e and β.

(b) **Sensitivity of m on R_e:**

$$S_m^{R_e} = \frac{\partial R_e}{\partial m}\frac{m}{R_e} = -\frac{m\mu\sigma\Lambda}{\mu(m+\mu) - m\sigma\Lambda}$$

The negative sign indicates that R_e is inversely proportional to m.

(c) **Sensitivity of σ on R_e:**

$$S_\sigma^{R_e} = \frac{\partial R_e}{\partial \sigma}\frac{\sigma}{R_e} = -\frac{m\Lambda\sigma}{\mu(\mu+m) - m\sigma\Lambda}$$

This indicates that R_e is inversely related to σ.

(d) **Sensitivity of γ on R_e:**

$$S_{\gamma}^{R_e} = \frac{\partial R_e}{\partial \gamma}\frac{\gamma}{R_e} = -\frac{\gamma}{\gamma + \mu}$$

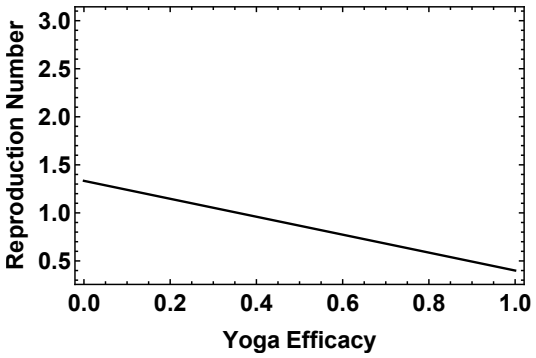
This shows an inverse relationship between γ and R_e. Similarly, μ has a negative effect on R_e.

The numerical sensitivity indices of R_e with respect to the above parameters are summarized in Table 3.3. From the table, we observe that β has a positive effect on R_e, whereas m, σ, and γ have negative effects. This table shows that a 10% increase (decrease) in β results in a 10% increase (decrease) in R_e, whereas a 10% increase in Yoga efficacy results in a 7.5% decrease in R_e, and a 12.3% increase in Yoga Sādhaka practitioners results in a 10% decrease in R_e. Thus, the sensitivity analysis indicates that the transmission rate directly influences R_e, while the increase in Yoga Sādhaka rate, Yoga efficacy, and recovery rate inversely affect R_e. Consequently, increased Yoga practice reduces the infection level. Sensitivity of parameters on reproduction number is shown in the

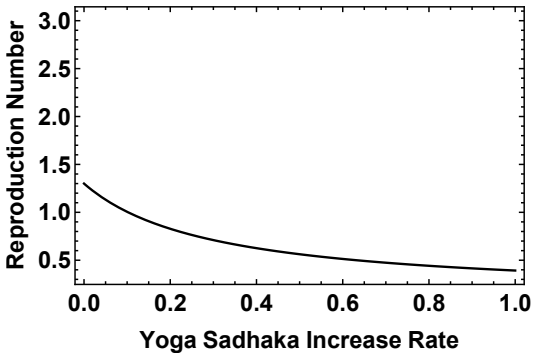
Parameter	Sensitivity Index of R_e
β	1
γ	- 0.625
m	- 1.235
σ	- 0.75

Table 3.3: Numerical values of sensitivity indices for R_e

Figures 3.3 and 3.4 which supports the numerical results.

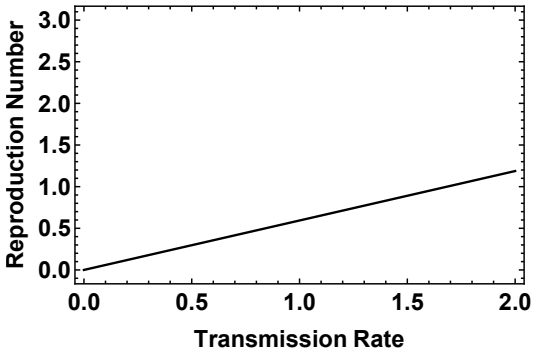


(a) Effect of Yoga efficacy (σ) on the reproduction number R_e.

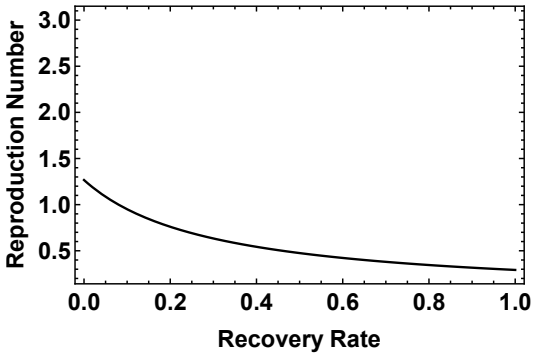


(b) Effect of Yoga Sādhaka increase rate (m) on the reproduction number R_e.

Figure 3.3: Sensitivity of the reproduction number R_e with respect to Yoga efficacy and the increase rate of Yoga Sādhakas.



(a) Effect of disease transmission rate (β) on the reproduction number R_e.



(b) Effect of recovery rate (γ) on the reproduction number R_e.

Figure 3.4: Sensitivity of the reproduction number R_e with respect to disease transmission and recovery rates.

Numerical Simulations. In this chapter, we conducted both local and global stability analyses of the $SYIRS$ model and observed that the yoga reproduction number, R_e, plays a critical role in controlling the disease. Our results indicate that if $R_e < 1$, the disease-free equilibrium is locally and globally stable, whereas if $R_e > 1$, an endemic equilibrium exists and is stable (both locally and globally). In addition to this analytical study, we present numerical simulations as follows.

At the disease-free equilibrium, using the parameters $\Lambda = 0.3$, $\mu = 0.3$, $m = 0.75$, $\sigma = 0.6$, $\gamma = 0.5$, $\beta = 1.1395$, and initial condi-

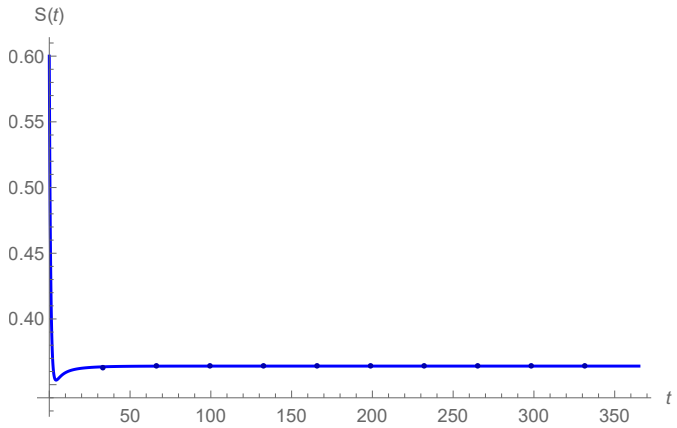
tions $(S(0), Y(0), I(0), R(0)) = (0.6, 0.3, 0.1, 0)$, we observe that $S(t)$ approaches its steady-state value, while $I(t)$ and $R(t)$ approach zero as time tends to infinity. This indicates that the disease eventually dies out as the number of Yoga Sādhaka individuals increases (see Figures 3.5 and 3.6).

Effect of Yoga Pranayama on Disease Dynamics

This section illustrates graphically how different parameters affect population classes. Pranayama reduces the level of epidemicity, leading all classes toward a positive steady state as the Yoga Sādhaka population increases. The effects are described below.

Reduced Susceptibility. Susceptible individuals at any time t are given by Equation (3.6). To understand the effect of Pranayama on susceptibility, we fixed parameters γ, μ, β, and σ and varied the Yoga Sādhaka increase rate m. Figure 3.7 shows that as m increases, S decreases and susceptibility stabilizes, indicating that Pranayama reduces susceptibility.

Reduced Infectivity. The infective population at time t is given by Equation (3.7). By varying m while keeping other parameters fixed, Figure 3.8 shows that infectivity decreases with an increase in Yoga Sādhaka individuals. A larger Yoga Sādhaka population can significantly control outbreaks. Continuous inflow of non-Yoga Sādhaka susceptible individuals, however, may shift the system from disease-free to endemic with



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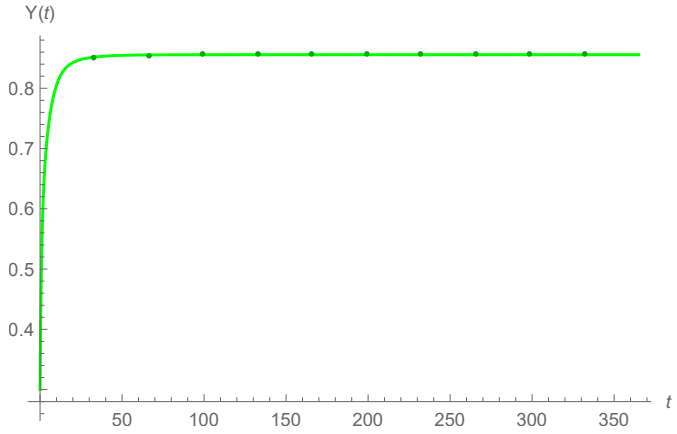

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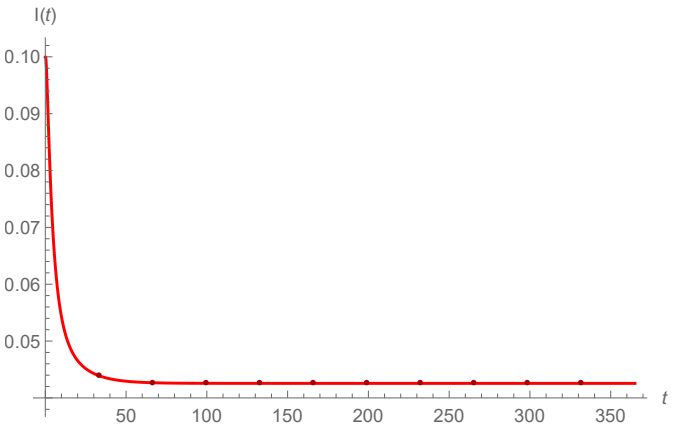
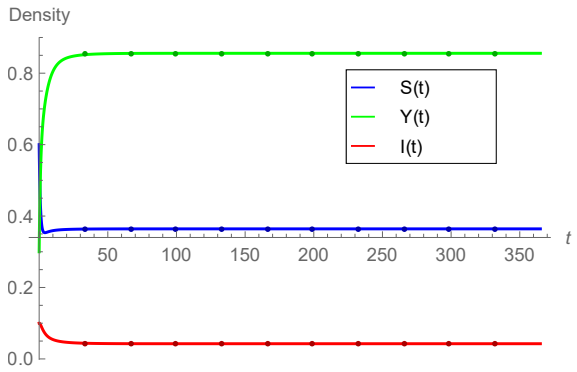


(c)

Figure 3.5: As the number of Yoga Sādhaka individuals increases, the number of susceptible and infectious individuals gradually decreases.

low infection.

Faster Recovery. Figure 3.10 demonstrates that recovery rate γ increases as Yoga Sādhaka individuals increase. Higher recovery rates



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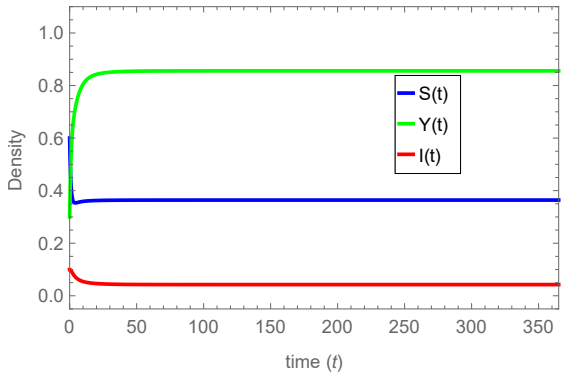


(b)

Figure 3.6: An increase in Yoga Sādhaka individuals reduces both susceptible and infective populations.

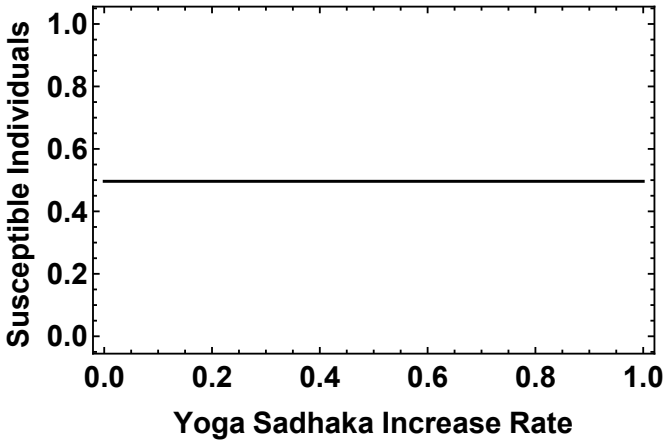


Figure 3.7: Susceptibility gradually decreases as Yoga Sādhaka individuals increase.

reduce the reproduction number R_e, consistent with the sensitivity analysis shown in Table 3.3.

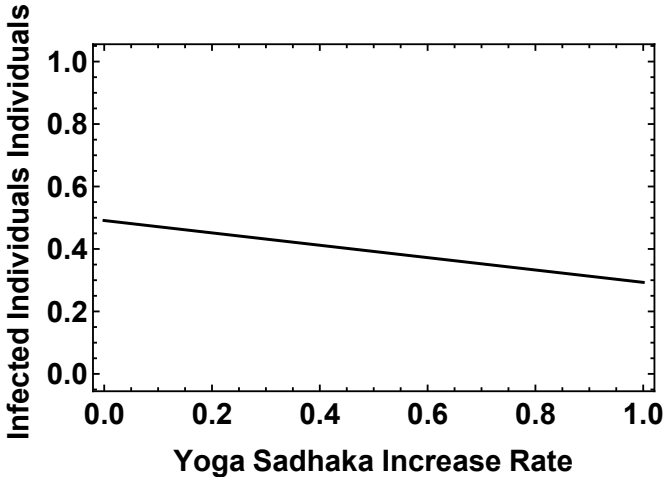


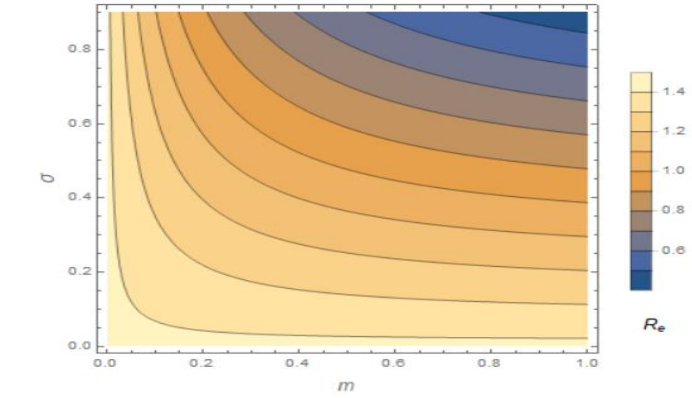
Figure 3.8: Pranayama reduces infectivity.

Longer Preservation of Immunity. Figures 3.7 and 3.8 illustrate that an increase in Yoga Sādhaka individuals reduces susceptibility and infectivity. Figure 3.10 shows that R_e decreases as γ increases, indicating faster recovery. This suggests that Yoga-induced immunity is preserved for a longer period.

Interpretation of Yoga Efficacy Results. Yoga efficacy represents the effectiveness of Pranayama in reducing susceptibility to infection among individuals practicing Yoga. The results of the model and sensitivity analysis indicate that higher values of σ lead to a significant reduction in the effective reproduction number R_e.

- Higher Yoga efficacy directly decreases the number of new infections, as indicated by the negative sensitivity index of R_e with respect to σ.

- Figures 3.3, 3.4, and 3.7 show that as σ increases, the susceptible population decreases more slowly while the infective population de-



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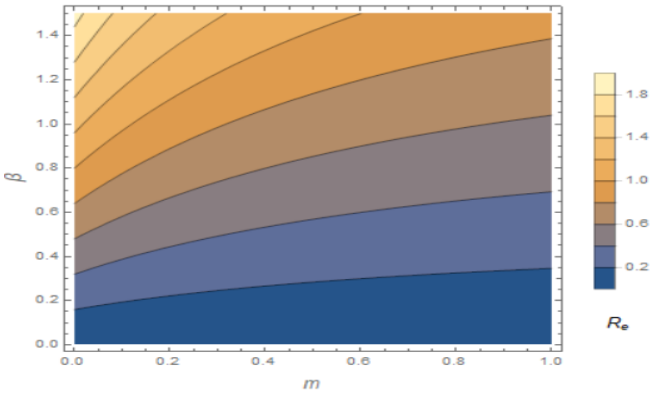


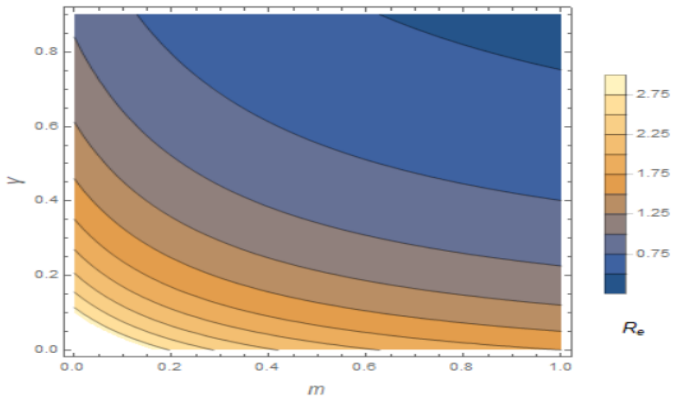
(b)

Figure 3.9: Relationship of reproduction number R_e with parameters m and σ in (a), and m and β in (b).

clines faster, demonstrating enhanced protection.

- The combined effect of increased Yoga efficacy and a higher Yoga Sādhaka increase rate (m) amplifies disease control, indicating that both individual adherence and the effectiveness of Prāṇāyāma are significant.

- Overall, improved Yoga efficacy contributes to lower susceptibility, reduced infectivity, faster recovery, and longer preservation of immunity, confirming that Prāṇāyāma is an effective preventive strat-



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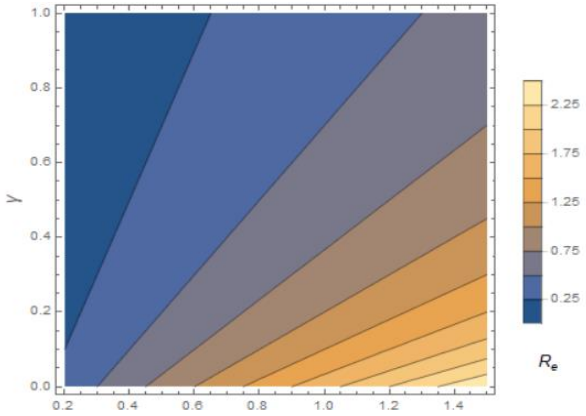


(b)

Figure 3.10: Relationship of reproduction number R_e with parameters m and γ in (a), and β and γ in (b).

egy.

- Figure 3.7 shows that as σ increases, the fraction of susceptible individuals stabilizes at a higher level, indicating reduced risk of infection due to enhanced protection from Prāṇāyāma.

- Figure 3.8 demonstrates that the infective population declines more rapidly with higher Yoga efficacy, reflecting reduced transmission in the community.

- Figures 3.9 and 3.10 illustrate the combined effects of Yoga efficacy

(σ) and the Yoga Sādhaka increase rate (m) on the reproduction number R_e. The contour plots show that higher σ and m jointly lead to a lower R_e, confirming that both the effectiveness of Prāṇāyāma and the number of practitioners are critical for disease control.

- Sensitivity analysis (Table 3.3) supports these observations by indicating that R_e is inversely related to σ. This means that as Yoga efficacy improves, the reproduction number decreases, leading to lower susceptibility and infectivity, faster recovery, and longer preservation of immunity.

These findings highlight that even moderate improvements in Yoga efficacy can substantially decrease the spread of infection, supporting the use of Pranayama as a practical and cost-effective public health intervention, especially in settings with limited access to medical facilities.

Discussion

This study develops a mathematical model to examine the transmission dynamics of a communicable disease with Yoga Pranayama as a preventive intervention. The model focuses on how Yoga efficacy (σ) and the rate of Yoga Sādhaka increase (m) influence disease spread and control.

Analytical results show that the disease-free equilibrium (DFE) is locally and globally stable when the Yoga reproduction number $R_e < 1$, while the endemic equilibrium (EE) exists and is stable for $R_e > 1$. These findings indicate that increasing Yoga practice can effectively re-

duce the risk of infection. Both the basic reproduction number R_0 and the effective reproduction number R_e decrease as the number of Yoga Sādhaka individuals increases, highlighting the potential of Pranayama as a non-pharmaceutical intervention.

Numerical simulations support these analytical results. Figures 3.5 and 3.6 show that higher numbers of Yoga Sādhaka reduce susceptibility (S) and infectivity (I) over time. Figure 3.7 demonstrates that increasing Yoga efficacy lowers susceptibility, while Figure 3.8 indicates that infectivity declines faster with higher efficacy. Contour plots (Figures 3.9 and 3.10) illustrate that both increased σ and m reduce R_e, emphasizing the combined importance of practitioner density and Yoga effectiveness.

Sensitivity analysis (Table 3.3) confirms that R_e is positively associated with disease transmission rate (β) and inversely related to Yoga efficacy, Yoga Sādhaka increase rate, and recovery rate (γ). This underscores that enhancing Yoga participation or efficacy can substantially lower disease transmission and accelerate recovery, aligning with earlier findings that preventive strategies reduce epidemic potential (Nadholta, Kumar, & Anand, 2023; Pandey et al., 2022; Sharma, 2020; Yeun & Kim, 2021).

Unlike previous theoretical studies (Kim et al., 2020; Y. Xiao, Zhao, Tang, et al., 2013; Yadav & Ganpat, 2021), this study formulates ODEs for disease dynamics and integrates numerical simulations, providing quantitative evidence that increasing Yoga Sādhaka density reduces infection rates and preserves immunity. The results also suggest

that Pranayama can serve as an effective preventive measure, especially in regions with limited access to conventional healthcare, where controlling disease spread through non-pharmaceutical interventions is critical.

In summary, this study demonstrates that structured Yoga programs, through higher efficacy and practitioner participation, lead to lower susceptibility, reduced infectivity, faster recovery, and prolonged immunity. These findings provide strong support for incorporating Yoga Pranayama into public health strategies as a practical and effective means of infectious disease prevention.

Conclusion

Mathematical models are widely recognized as powerful tools for designing and evaluating control strategies for infectious diseases. In this study, we developed an epidemiological model incorporating Yoga Prāṇāyāma as a non-pharmaceutical intervention. The model differentiates between Yoga Sādhaka and native susceptible populations, predicting trends in susceptibility and infectivity based on the Yoga Sadhaka increase rate and Yoga efficacy.

Both qualitative and quantitative analyses were conducted to assess the biological implications and stability of the system. Using realistic parameter values, the results showed that the disease-free equilibrium exists when the effective reproduction number, $R_e < 1$, whereas the endemic equilibrium persists when $R_e > 1$. Sensitivity analysis further demonstrated that practicing Yoga Prāṇāyāma reduces susceptibility

and infectivity, accelerates recovery, and prolongs immunity. It can be concluded that infections can be effectively controlled by increasing the rate of Yoga Sādhaka individuals and enhancing Yoga efficacy. Therefore, Pranayama is confirmed as a practical and effective strategy for the prevention and control of communicable diseases, particularly in populations with limited access to conventional healthcare facilities.

Chapter 4

Analysis of an SIRS Epidemic Model Incorporating Yoga Awareness

Preamble

In Chapter 3, we studied Yoga Pranayama as a strategy to control communicable diseases. Yoga Pranayama works best when people also adopt healthy behaviors, including a proper diet (Aahar), exercise or physical activity (Aashan or Vihar), ethical conduct (Aachar), and positive thinking (Vichar). This chapter extends that study by including Yoga awareness along with Pranayama and other lifestyle awareness programs. We propose a mathematical model that assumes Yoga awareness can reduce disease transmission. The model accounts for different sources of awareness: Yoga practitioners, organized awareness campaigns, direct interaction between aware and unaware individuals, and reported infection cases. Yoga classes themselves help spread awareness. A $SIRS$ epidemic model has been formulated that incorporates Yoga awareness.

Diseases such as COVID-19, influenza, Ebola, SARS, avian influenza, and swine influenza continue to spread and cause serious health

and economic problems. Educating people about disease dynamics and promoting new control strategies is essential. Disease outbreaks often increase awareness in society, especially among those who are close to infected individuals. Health-focused programs, including Yoga classes and wellness campaigns, also raise awareness. People then change their behavior, reducing their risk of infection. Awareness campaigns can therefore help control disease spread and prevent recurrence. Previous studies have shown that public awareness can significantly reduce disease transmission (Sivananda, 2019; Vidyapeeth, 2015). Zewdie and Gakkhar (2022) developed a $SWEIQR$ model in which W represents the aware population in transport-related infection dynamics with entry-departure screening. They calculated the reproduction number and studied its dependence on awareness and screening. They found that the disease-free state remains stable when the reproduction number is below one, and unstable otherwise. Simulations confirmed that raising awareness reduces disease spread. Screening also helps control transmission across locations. Similarly, interventions such as school closures, mask use, and isolation of infected individuals (social distancing) slow pandemics (Shariff et al., 2019). Rwezaura et al. (2010) built a deterministic model combining vaccination and treatment for influenza. They found that doing both together is more effective than focusing on either alone. They also studied solution properties, stability, and sensitivity of parameters. Raimundo et al. (2007) studied reinfection dynamics, showing that disease control depends on vaccination coverage and vaccine efficacy. Singh and Dhar

(2018) developed a $SIRS$ model that included media awareness, demonstrating that awareness reduces disease transmission. Our model builds on these studies but adds Yoga awareness as a control strategy.

We use knowledge from Eastern philosophy to reduce disease spread and disrupt the host-environment-agent chain. Ancient seers practiced Yoga to explore both the external and internal world. They focused on personal verification rather than belief (Bhavanani, 2012; Feuerstein et al., 1979; Sivananda, 2019; Swanson, 2019; White, 2019). Today, Yoga is practiced worldwide, including in Western societies (Garfinkel & Schumacher Jr, 2000). Yoga teaches living in harmony with life. Sage Patanjali recorded these teachings in the *Yoga Sutras*, a 2000-year-old text. Yoga restrains thought processes and calms the mind. It also promotes virtues like Ahimsa (non-injury) and Satya (truth), and qualities such as amity and compassion. In this chapter, we focus on the fourth limb of Yoga, Pranayama, which improves physical development and physiological functions (Feuerstein et al., 1979). Yoga consists of four pillars—Aahar, Vihar, Aachar, and Vichar (AVAV)—which promote self-awareness and help people understand their thoughts, feelings, values, beliefs, and actions. Practicing these principles develops conscious behavior.

This chapter presents the model formulation, analyzes the system's dynamic behavior, derives equilibrium points, and the basic reproduction number, examines the stability of disease-free and endemic states, and concludes with numerical results, discussion, and conclusion.

Assumptions and Model Development

Formulation of Model

A $SIRS$ epidemic model is proposed to investigate the effects of Yogachara on disease transmission dynamics. This mathematical model builds on the previous study by (Kim et al., 2020). A standard compartmental approach divides the population into Susceptible, Infectious, and Recovered classes. Since disease-induced deaths are negligible and the birth rate nearly equals the death rate, we assume that the total population size, $N(t)$, remains constant. In this chapter, the Susceptible and Yoga Sadhaka compartments are both considered as Yoga-aware susceptible (S), as they share the same risk of infection and mix homogeneously. Thus, the total population is represented as $N(t) = S(t) + I(t) + R(t)$.

We assume that the population is entirely susceptible at the start of the epidemic. The baseline transmission rate is denoted by β, w represents the Yoga-aware population, and c measures the efficacy of Yoga awareness, providing partial immunity such that aware individuals are less susceptible than unaware individuals. Infectious individuals recover at rate γ, and recovered individuals gain temporary immunity, returning to the susceptible class at rate λ. The following assumptions guide the model development:

1. The population mixes homogeneously.

2. Yoga awareness (Yogachara) includes Pranayama as well as Aahar,

Vihar, Aachar, and Vichar. This combination reduces disease transmission.

3. Recovered individuals acquire temporary immunity, which wanes at rate λ.

4. The disease transmission rate, β_1, varies over time and depends on the Yoga-aware infected mass. Its rate of change with respect to Yoga-aware infected mass M is proportional to the transmission rate:

$$\frac{d\beta_1}{dM} = -c\beta_1,$$

where $c \geq 0$ represents the level of awareness coverage. The negative sign indicates that transmission decreases as Yoga awareness increases. Therefore, the effective transmission rate is

$$\beta_1 = \beta e^{-cM(t)},$$

where β is the transmission rate in the absence of yoga awareness. This assumes that individuals adopt behaviors that reduce the probability of infection.

Yoga awareness is incorporated with $M(t) = p \times w(t)$, where $w(t)$ is the Yoga-aware population at time t. This includes Yoga Sadhakas, such as Yoga teachers and class participants who have adopted behavior changes. The scale constant p is estimated using the least squares method. This term assumes that individuals adopt

healthy habits along with Pranayama to reduce disease risk (Kim et al., 2020). The flow diagram of the model is shown in Figure 4.1.

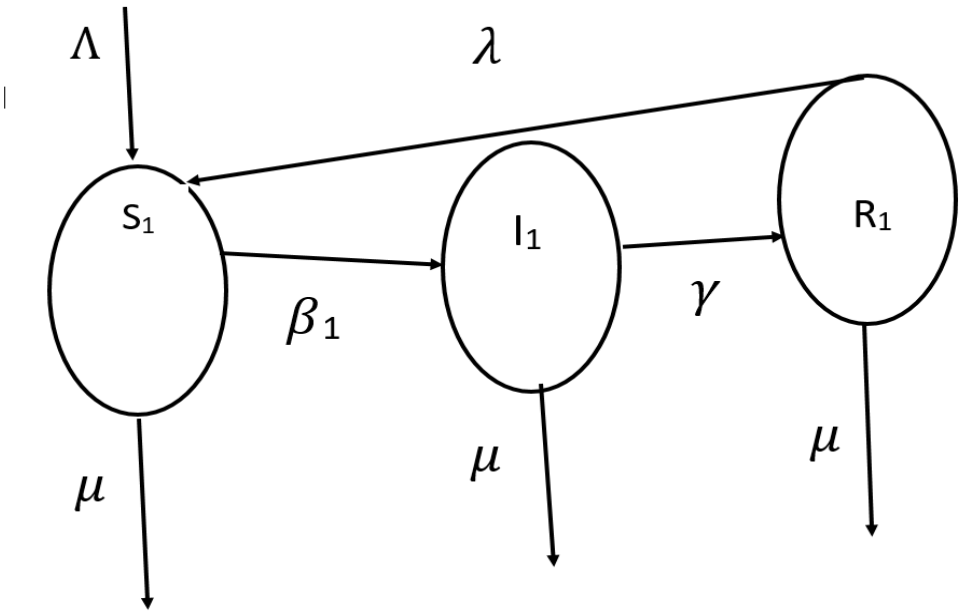


Figure 4.1: $SIRS$ model diagram with Yoga awareness transmission rate, $\beta_1 = \beta e^{-cM(t)}$.

Under these assumptions, the system of differential equations describing disease transmission dynamics is:

$$\begin{aligned}
\frac{dS_1}{dt} &= \wedge N - \beta e^{-cM(t)} \frac{S_1 I_1}{N} + \lambda R_1 - \mu S_1, \\
\frac{dI_1}{dt} &= \beta e^{-cM(t)} \frac{S_1 I_1}{N} - \mu I_1 - \gamma I_1, \\
\frac{dR_1}{dt} &= \gamma I_1 - \mu R_1 - \lambda R_1.
\end{aligned} \tag{4.1}$$

The initial conditions are:

$$S_1(0) = S_0 > 0, \quad I_1(0) = I_0 > 0, \quad R_1(0) = R_0 > 0. \tag{4.2}$$

The total population satisfies:

$$N(t) = S_1(t) + I_1(t) + R_1(t).$$

Consider the three-dimensional region:

$$\Omega = \{(S_1, I_1, R_1) : 0 \leq S_1, I_1, R_1 \leq \frac{\wedge}{\mu}\}.$$

Table 4.1 describes the parameters used in system (4.1).

Parameter	Description
$\wedge$	Recruitment rate
$\frac{1}{\mu}$	Average life span
β	Disease transmission rate without Yoga awareness
β_1	Disease transmission rate with Yoga awareness
c	Level of Yoga awareness coverage
γ	Recovery rate
λ	Rate at which disease-induced immunity wanes

Table 4.1: Definition of model parameters

Data Source and Parameter Estimation

In this section, parameters were estimated for the Yoga awareness effect term included in the model through $M(t)$. This term modifies the incidence rate as

$$\beta e^{-cM(t)} SI.$$

Real-world Yoga awareness coverage data were collected from 20 Yoga centers in Sudurpashchhim Province, Nepal (Appendix D). We defined $M(t) = p \times w(t)$, where $w(t)$ represents the Yoga awareness coverage at time t, $M(t)$ denotes the Yoga-aware infected mass, and p is a scale

constant.

The observed data, $w(t) = (x_1, x_2, \ldots, x_n)$ and $M(t) = (y_1, y_2, \ldots, y_n)$, represent the numbers of Yoga-aware and Yoga-aware infected individuals per center, respectively. The scale constant p was estimated using the least-squares method to fit $M(t)$ to $w(t)$, based on data from March 2021 to May 2022, processed and analyzed in *Mathematica* software.

The transmission rate β_1 in the Yoga Sādhaka population is given by

$$\beta_1 = \frac{\text{force of infection}}{S/N},$$

where the force of infection is defined as

$$\text{Force of infection} = \frac{\text{Total Infected Individuals}}{n \times T},$$

with T denoting the time period and n the sample size. The remaining parameter values were fixed as listed in Table 4.2.

Results and Discussion

Positivity and Boundedness

Since the model system with given initial conditions (4.2) represent the dynamics of population, it is essential to show that its solutions are positive and bounded.

Theorem 4.1. *(Singh, n.d.) The solution of system (4.1) with initial condition (4.2) are positive and bounded, i.e., all the trajectories of system (4.1) initiating inside Ω, will stay within the interior of Ω.*

Proof. Let $R_+^3 = \{(S_1, I_1, R_1) \in R_+^3 : S_1 \geq 0, I_1 \geq 0, R_1 \geq 0\}$ be the three dimensional space.

From (4.1), we observed that $\frac{dS_1}{dt} = \wedge N + \lambda R_1 > 0$ when $S_1 = 0$

$\frac{dI_1}{dt} = 0$ when $I_1 = 0$

$\frac{dR}{dt} = \gamma_1 I_1 \geq$ when $R_1 = 0$

and $S_1(t), I_1(t), R_1(t)$ are continuous function of t. Thus the vector field initiating in R_+^3 will remain inside R_+^3 for all the time. Also the total population, $N(t) = S_1(t) + I_1(t) + R_1(t)$ is constant satisfies $\frac{dN}{dt} = 0$. Therefore, system (4.1) is bounded and its any solution originates from Ω remains in Ω. □

Normalization of the Model

We can normalize the above system (4.1) with initial conditions (4.2) as follow

$$S = \frac{S_1}{N}, \qquad I = \frac{I_1}{N}, \qquad R = \frac{R_1}{N}.$$

The rescaled equations are

$$\begin{aligned} \frac{dS}{dt} &= \wedge - \beta e^{-cM(t)} SI + \lambda R - \mu S \\ \frac{dI}{dt} &= \beta e^{-cM(t)} SI - \mu I - \gamma I \\ \frac{dR}{dt} &= \gamma I - \mu R - \lambda R \end{aligned} \tag{4.3}$$

with initial conditions

$$S(0) = S_0 > 0, I(0) = I_0 > 0, R(0) = R_0 > 0.$$

Parameter	Value	Reference
R_a	[0.5 - 1.6]	Calculated
β_1	0.05	Calculated
β	[0.05 , 1.6]	Musa et al. (2021)
λ	0.02	Musa et al. (2021)
c	$c \in [0, 1]$	Assumed
γ	[0.05,0.99]	Musa et al. (2021)
p	0.0231	Estimmated
μ	0.05	Musa et al. (2021)

Table 4.2: Estimated parametric values

Dynamic Behaviour of the Model

In this section, we calculate the awareness reproduction number, feasible steady states and analyze the stability of equilibria for the proposed system. The biologically feasible region for the non-dimensional system is $\Omega = \{(S, I, R) : 0 \leq S, I, R \leq 1\}$.

Equilibrium States and Awareness Reproduction Number. The equilibrium states of the system correspond to conditions where the disease dynamics reach a steady state, i.e., the rates of change of all compartments become zero. For equilibrium point of the model (4.3), we

have

$$\begin{aligned} \wedge - \beta e^{-cM(t)} SI + \lambda R - \mu S = 0 \\ \beta e^{-cM(t)} SI - \mu I - \gamma I = 0 \\ \gamma I - \mu R - \lambda R = 0. \end{aligned} \tag{4.4}$$

The system (4.3) has two equilibrium (steady) states: the disease-free equilibrium (DFE) $E^0 = (1, 0, 0)$ and the endemic equilibrium (EE) $E^* = (S^*, I^*, R^*)$. We now calculate the reproduction number by defining the threshold quantity R_a as the average number of secondary infections produced when a single initially infected individual is introduced into a susceptible, yoga-aware population. The quantity R_a is referred to as the *awareness reproduction number* because, in this model, yoga-based awareness acts as a control mechanism to reduce disease transmission. This parameter plays a crucial role in determining whether the infection will die out or persist within the population. The expression for R_a will be derived using the method described in Chapter 2.

Let $X = (S, I, R)$. Therefore, $\frac{dX}{dt} = \mathcal{F} - \mathcal{V}$, where

$$\mathcal{F} = \begin{bmatrix} 0 \\ \beta_1 SI \\ 0 \end{bmatrix}$$

and

$$\mathcal{V} = \begin{bmatrix} -\Lambda + \beta_1 SI - \lambda R + \mu S \\ \mu I + \gamma I \\ -\gamma I + \mu R + \lambda R \end{bmatrix}$$

At disease-free equilibrium, variation matrices of $\mathcal{F}$ and $\mathcal{V}$ are given by

$$F_0 = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \beta_1 S_0 & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

$$V_0 = \begin{bmatrix} \mu & \beta_1 S_0 & -\lambda \\ 0 & \mu + \gamma & 0 \\ 0 & -\gamma & \lambda \end{bmatrix}$$

The next generation matrix for the model equation (4.3) is

$$F_0 V_0^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{\beta_1 S_0}{\mu+\gamma} & 0 \\ 0 & 0 & 0 \end{bmatrix}$$

The spectral radius of the matrix $F_0 V_0^{-1}$ gives expression R_a. Awareness reproduction number is $R_a = \frac{\beta_1}{(\gamma+\mu)} = \frac{\beta e^{-cM}}{(\gamma+\mu)} = \frac{R_0}{e^{cM}}$.

Now, solving (4.4), we obtain a unique positive equilibrium known as endemic equilibrium point $E_1 = (S^*, I^*, R^*)$. From last equation of system (4.4), we get, $R^* = \frac{\gamma I^*}{\lambda+\mu}$.

From second equation of system (4.4), we get,

$$S^* = \frac{(\gamma+\mu)e^{cM_0}}{\beta}$$

$$S^* = \frac{e^{cM}}{R_0}$$

$$M^* = \frac{ln(\frac{S^*\beta}{\gamma+\mu})}{c} = \frac{ln(S^*R_0)}{c}$$

Using, $S^* = 1 - I^* - R^*$, the value of I^* is given by

$$\begin{aligned}
\frac{e^{cM^*}}{R_0} &= 1 - (\frac{\lambda+\mu+\gamma}{\lambda+\mu})I^* \\
I^* &= \frac{\lambda+\mu}{\gamma+\lambda+\mu}(1 - \frac{\gamma+\mu}{\beta}e^{cM}) \\
&= \frac{\lambda+\mu}{\lambda+\mu+\gamma}(1 - \frac{e^{cM}}{R_0}) \\
&= (\frac{\lambda+\mu}{\lambda+\mu+\gamma})(1 - \frac{1}{R_a})
\end{aligned} \tag{4.5}$$

If there is no yoga awareness effect, i.e., $c = 0$, then

$I^* = \frac{\lambda+\mu}{\gamma+\lambda+\mu}(1 - \frac{\gamma+\mu}{\beta}) = (\frac{\lambda+\mu}{\lambda+\mu+\gamma})(1 - \frac{1}{R_0})$

Clearly , I^* exists if and only if $R_a > 1$.

The endemic equilibrium does not exist for $R_a \leq 1$ and exist for $R_a > 1$. Also, in presence of Yoga awareness, endemic equilibrium does not exist for $R_a < 1$ which is shown in Figure (4.2). But endemic equilibrium exist for $R_a > 1$ which is shown in Figure (4.3)

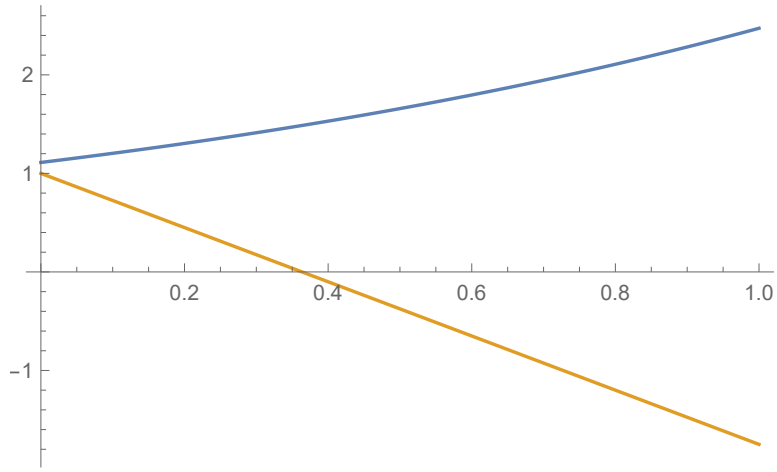


Figure 4.2: Non-existence of endemic equilibrium for parametric values $\beta = 0.08$, $\gamma = 0.2$, $\lambda = 0.02$, $\mu = 0.05$, $R_a < 1$, where blue color represent $\frac{e^{cM}}{R_a}$ and pink color represent the curve $1 - (1 + \frac{\gamma}{\lambda+\mu})I^*$.

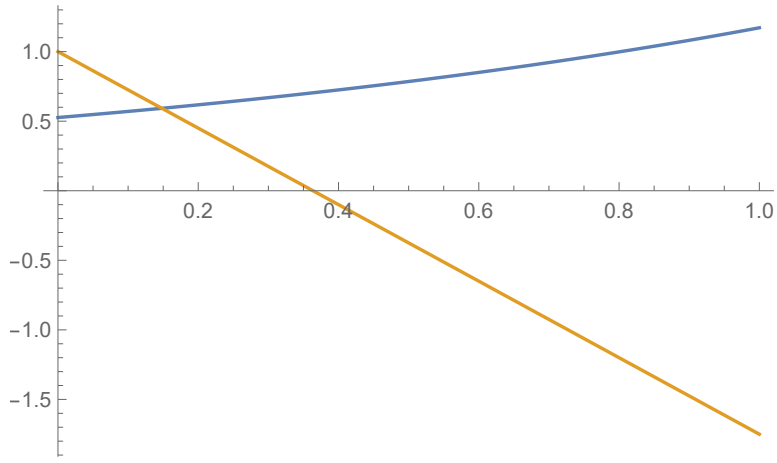


Figure 4.3: Existence of endemic equilibrium for parametric values $\beta = 0.8$, $\gamma = 0.3$, $\lambda = 0.02$, $\mu = 0.05$,$R_a > 1$, where blue color represent $\frac{e^{cM}}{R_0}$ and pink color represent the curve $1 - (1 + \frac{\gamma}{\lambda+\mu})I^*$.

Stability Analysis of the System

Stability analysis of equilibrium states reveals the long-term behavior of the system and the effectiveness of Yoga awareness interventions in controlling disease transmission. It determines whether small perturbations around an equilibrium decay over time (indicating stability) or amplify (indicating instability). This section presents the local and global stability analysis of the model.

Local Stability Analysis. Local stability describes a system's response to small disturbances from its equilibrium. In a population, minor perturbations, such as a few individuals becoming infected or recovering, may temporarily disrupt a steady state. If the system returns to equilibrium after such perturbations, it is locally stable, indicating resilience to small shocks; if it diverges further, it is unstable, implying amplification of disturbances. Conceptually, local stability reflects the system's tendency to revert to balance after minor fluctuations, analogous to a ball returning to the bottom of a valley when slightly displaced. In the Yoga awareness–based SIR model, the disease-free equilibrium is locally stable if small infections disappear over time, demonstrating the effectiveness of practices such as Aahar (balanced diet), Vihar (healthy lifestyle), Aachar (ethical conduct), and Vichar (positive thinking). Conversely, the equilibrium is unstable if infections increase. Endemic equilibrium represents a situation where disease persists at a roughly constant level; small changes in infection numbers naturally return to this level, illustrating local stability, where minor increases or decreases do not lead to major outbreaks or disease disappearance.

Theorem 4.2. *The system 4.3 has*

(i) the disease-free equilibrium (DFE) $E^* = (1, 0, 0)$ *for all parameter values,*

(ii) no endemic equilibrium (EE) if $R_a \leq 1$*, and*

(iii) a unique endemic equilibrium if $R_a > 1$.

Theorem 4.3. *The disease-free equilibrium (DFE)* E^0 *is*

(i) locally asymptotically stable if $R_a < 1$,

(ii) unstable if $R_a > 1$, *and*

(iii) disease-free when $R_a = 1$.

Proof. The variation matrix for the DFE is

$$V(E_0) = \begin{bmatrix} -\mu & -\beta & \lambda \\ 0 & \beta - \gamma - \mu & 0 \\ 0 & \gamma & -\lambda - \mu \end{bmatrix}. \tag{4.6}$$

If I is the identity matrix of order three and K is a scalar, the characteristic equation of $V(E_0)$ is

$$|V(E_0) - KI| = \begin{vmatrix} -\mu - K & -\beta & \lambda \\ 0 & \beta - \gamma - \mu - K & 0 \\ 0 & \gamma & -\lambda - \mu - K \end{vmatrix} = 0. \tag{4.7}$$

Expanding, we obtain

$$(-\mu - K)(\beta - \gamma - \mu - K)(-\lambda - \mu - K) = 0.$$

Hence,

$$K_1 = -\mu, \quad K_2 = -\lambda - \mu, \quad K_3 = \beta - \gamma - \mu.$$

Two eigenvalues are negative, and the third is negative if $(\beta - \gamma - \mu) < 0$,

i.e., if $\beta \leq \gamma + \mu$, which gives

$$R_a = \frac{\beta}{\gamma + \mu} < 1. \tag{4.8}$$

Therefore, all eigenvalues are negative if $R_a < 1$, making the DFE locally asymptotically stable. The system is unstable when $R_a > 1$. When $R_a = 1$, from expression (4.5), $I^* = 0$, so the population is disease-free. □

Theorem 4.4. *The endemic equilibrium (EE) is locally asymptotically stable for* $R_a > 1$.

Proof. From the endemic equilibrium (4.5),

$$I^* = \left(\frac{\lambda + \mu}{\lambda + \mu + \gamma}\right)\left(1 - \frac{1}{R_a}\right). \tag{4.9}$$

Since λ, μ, and γ are positive, if $R_a < 1$, then $I^* < 0$, which is a contradiction because $I^* > 0$ for an endemic equilibrium. Therefore, the endemic equilibrium exists and is locally asymptotically stable only if $R_a > 1$. □

Theorem 4.5. *The endemic equilibrium* E_1 *is locally asymptotically stable if the coefficients of the characteristic equation of system (4.3) at* E^* *satisfy the Routh–Hurwitz criterion.*

Proof. The variational matrix at the endemic equilibrium is

$$E^* = \begin{bmatrix} -\beta e^{-cM} I^* - \mu & \beta e^{-cM} S^* & \lambda \\ \beta e^{-cM} I^* & \beta e^{-cM} S^* - \gamma - \mu & 0 \\ 0 & \gamma & -\mu - \lambda \end{bmatrix} = \begin{bmatrix} x_{11} & x_{12} & x_{13} \\ x_{21} & x_{22} & 0 \\ 0 & x_{32} & x_{33} \end{bmatrix}, \tag{4.10}$$

where

$$\begin{aligned} &x_{11} = -\beta e^{-cM} I^* - \mu, \quad && x_{12} = -\beta e^{-cM} S^*, && x_{13} = \lambda, \\ &x_{21} = \beta e^{-cM} I^*, && x_{22} = \beta e^{-cM} S^* - \gamma - \mu, && x_{32} = \gamma, \\ &x_{33} = -\lambda - \mu. \end{aligned}$$

The characteristic equation is

$$|KI - E^*| = 0 \quad \Rightarrow \quad K^3 + AK^2 + BK + C = 0,$$

with

$$\begin{aligned} A &= -(x_{11} + x_{22} + x_{33}) = \beta_1(I^* - S^*) + 3\mu + \gamma + \lambda, \\ B &= x_{11}x_{22} + x_{11}x_{33} + x_{22}x_{33} - x_{12}x_{21} \\ &= \beta_1[I^*(\gamma + \lambda + 2\mu) - S^*(2\mu + \lambda)] + (2\mu\gamma + 2\lambda\mu + 3\mu^2 + \gamma\lambda), \\ C &= x_{12}x_{21}x_{33} - x_{13}x_{21}x_{32} - x_{11}x_{22}x_{33} \\ &= (\lambda + \mu)[\beta_1 I^*\gamma + \beta_1 I^*\mu - \beta_1 S^*\mu + \gamma\mu + \mu^2] - \beta_1 I^*\lambda\gamma, \end{aligned}$$

where $\beta_1 = \beta e^{-cM}$ and M is a constant.

According to the Routh–Hurwitz criterion (Mahardika et al., 2019),

all eigenvalues have negative real parts if

$$A > 0, \quad B > 0, \quad C > 0, \quad AB - C > 0, \quad ABC - C^2 > 0.$$

Hence, the endemic equilibrium E^* is locally asymptotically stable if $R_a > 1$ and the above conditions hold. □

Global Stability Analysis. Global stability describes a system that eventually settles into a steady state (equilibrium) regardless of initial conditions. In epidemiological models, if the disease-free equilibrium is globally stable, the disease will eventually die out, no matter how many individuals are initially infected. If the endemic equilibrium is globally stable, the disease stabilizes at a constant number of cases irrespective of initial infections. This concept can be illustrated by the analogy of a ball in a valley: under local stability, a slight nudge returns the ball to the bottom, while global stability ensures the ball reaches the bottom from any point on the hill. Formally, a system is globally stable if all trajectories converge to a single equilibrium point over time (Martcheva, 2015).

Theorem 4.6. *If $R_a < 1$, then the disease-free equilibrium E_0 of the system (4.3) is globally asymptotically stable in the region Ω. If $R_a > 1$, then the endemic equilibrium E^* is globally asymptotically stable in the region $\Omega = (S, I, R)$.*

Proof. First we prove the global stability at the disease-free equilibrium E_0 when $R_a < 1$. Consider a Lyapunov function $L = I$. Then the

Lyapunov derivative will be

$$\begin{aligned}\frac{dL}{dt} &= \frac{dI}{dt}\\ \frac{dL}{dt} &= [\beta e^{-cM(t)}S - \mu - \gamma]I\\ &= [\beta - \mu - \gamma]I\\ &\leq 0, \text{since } R_a < 1.\end{aligned}$$

Thus, if $R_a < 1$, then $\frac{dL}{dt} \leq 0$. Therefore the largest positive invariant set in $(S, I, R) \in \Omega$ is the singleton set E_0 , where E_0 is the disease-free equilibrium. Thus by Lasalle's invariant principle E_0 is globally asymptotically stable in Ω.

In order to prove the global stability of E^* when $R_a > 1$ the system (4.3) can be rewritten as

$$\begin{aligned}\frac{dI}{dt} &= [\beta e^{-cM(t)}(1 - I - R) - \mu - \gamma]I\\ \frac{dR}{dt} &= \gamma I - \mu R - \lambda R.\end{aligned}$$

Now, we discuss in the first quadrant of IR-Plane. Using Dulac's criteria with multipliers $D_1 = \frac{1}{I}$.

Let,

$$\begin{aligned}F_1 &= [\beta e^{-cM}(1 - I - R) - \mu - \gamma]I,\\ F_2 &= \gamma I - \mu R - \lambda R,\end{aligned}$$

$$D_1F_1 = [\beta e^{-cM}(1 - I - R) - \mu - \gamma]$$
$$D_1F_2 = \gamma - (\frac{\mu + \lambda}{I})R$$

We have,

$$\frac{\partial D_1F_1}{\partial I} + \frac{\partial D_1F_2}{\partial R} = -\beta e^{-cM} - (\frac{\mu + \lambda}{I}) < 0$$

Thus, there is no limit cycle, i.e., no periodic solutions exist in the region. Hence by Poincare-Bendixson theory, endemic equilibrium E^* is globally asymptotically stable in the region Ω for the system (4.3) and hence for the original system (4.1). □

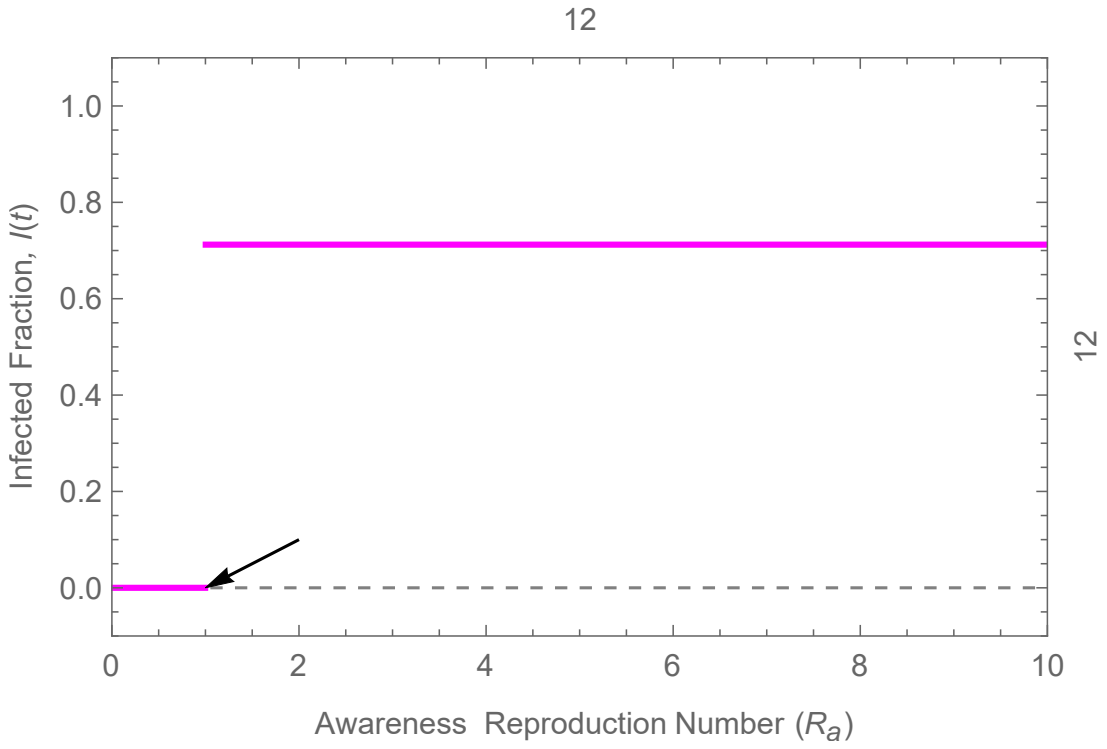


Figure 4.4: Disease-free equilibrium when $R_a < 1$ and endemic equilibrium when $R_a > 1$.

Sensitivity Analysis and Numerical Results

Sensitivity analysis was performed using the parameter values listed in Table 4.2 to evaluate the effects on the reproduction number R_a and the endemic equilibrium. The sensitivity index is defined as

$$S_y^p = \frac{\partial P}{\partial y}\frac{y}{P},$$

where y denotes the parameter considered. The sensitivity indices of R_a with respect to the model parameters are summarized in Table 4.3.

Parameter (y)	Sensitivity index of R_a w.r.t y	Numerical Value
β	1	1
λ	0	0
c	$-cM$	-0.0231
γ	$-\frac{\gamma}{\gamma+\mu}$	-0.945
M_0	$-cM$	-0.0231

Table 4.3: Sensitivity of R_a with respect to model parameters.

Sensitivity indices of the state variables $S(t)$, $I(t)$, and $R(t)$ with respect to parameters listed in Table 4.2 are given in Table 4.4.

Sensitivity indices of the state variables $S(t)$, $I(t)$, and $R(t)$ with respect to the parameters listed in Table 4.2 are given in Table 4.4.

Parameter (y)	$S_y^{S^*}$	$S_y^{I^*}$	$S_y^{R^*}$
β	-1	0.00417	0.02817
λ	0	0.2506	-1.5325
c	-0.01155	-0.03967	-0.01072
γ	0.909	-0.000172	-0.00642

Table 4.4: Sensitivity indices of state variables S^*, I^*, and R^* with respect to parameters β, λ, c, and γ.

The reproduction number R_a is most sensitive to β (positive) and γ (negative), with minor negative effects from Yogachara-related param-

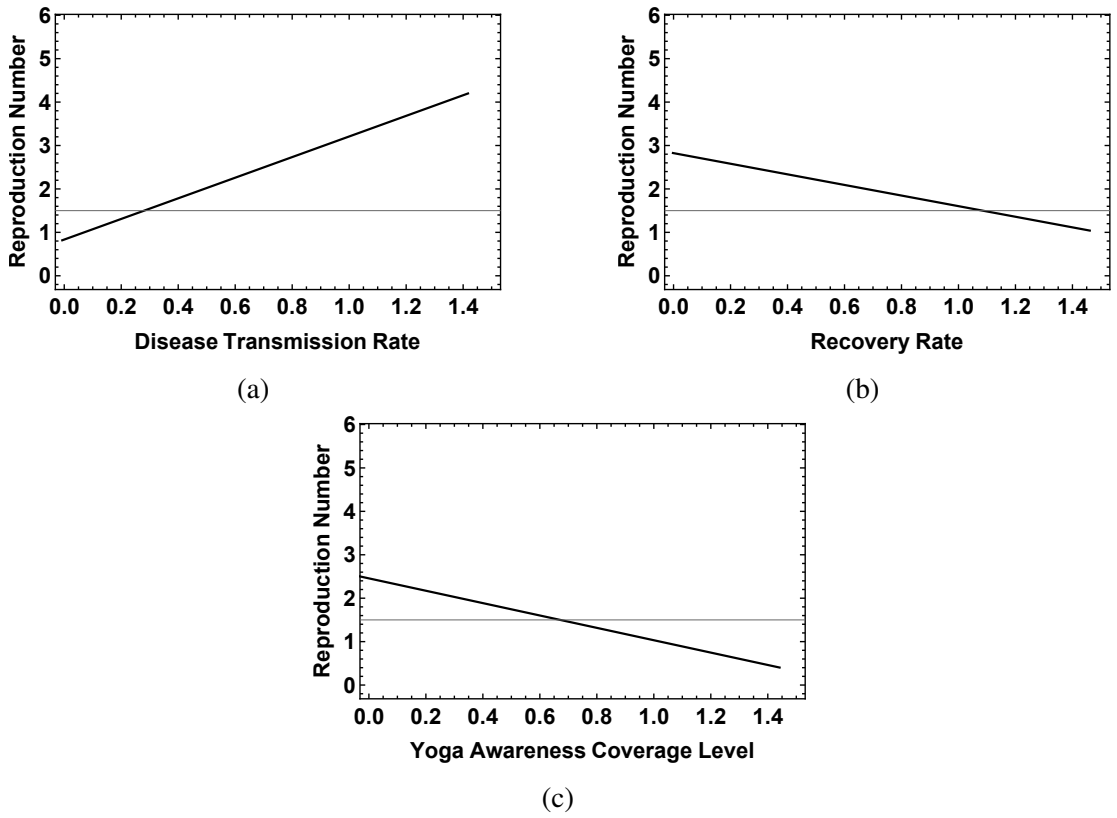


Figure 4.5: Relationship of reproduction number R_a with selected parameters.

eters c and M_0, while λ has negligible impact (Table 4.3; Figure 4.5). Susceptibles S^* decrease with β and increase with γ, infected I^* responds mainly to λ, and recovered R^* is negatively influenced by λ and slightly positively by c and β (Table 4.4; Figures 4.6–4.10). Overall, β and γ dominate system dynamics, whereas Yogachara-related interventions provide modest stabilizing effects.

Impact of Yoga Awareness on Disease Dynamics

In this section, we investigate the impact of Yoga awareness on disease transmission dynamics. Numerical simulations were performed using the estimated parameter p and the baseline parameter values listed in Table 4.2. Figures 4.5 and 4.6 illustrate the sensitivity of the reproduction

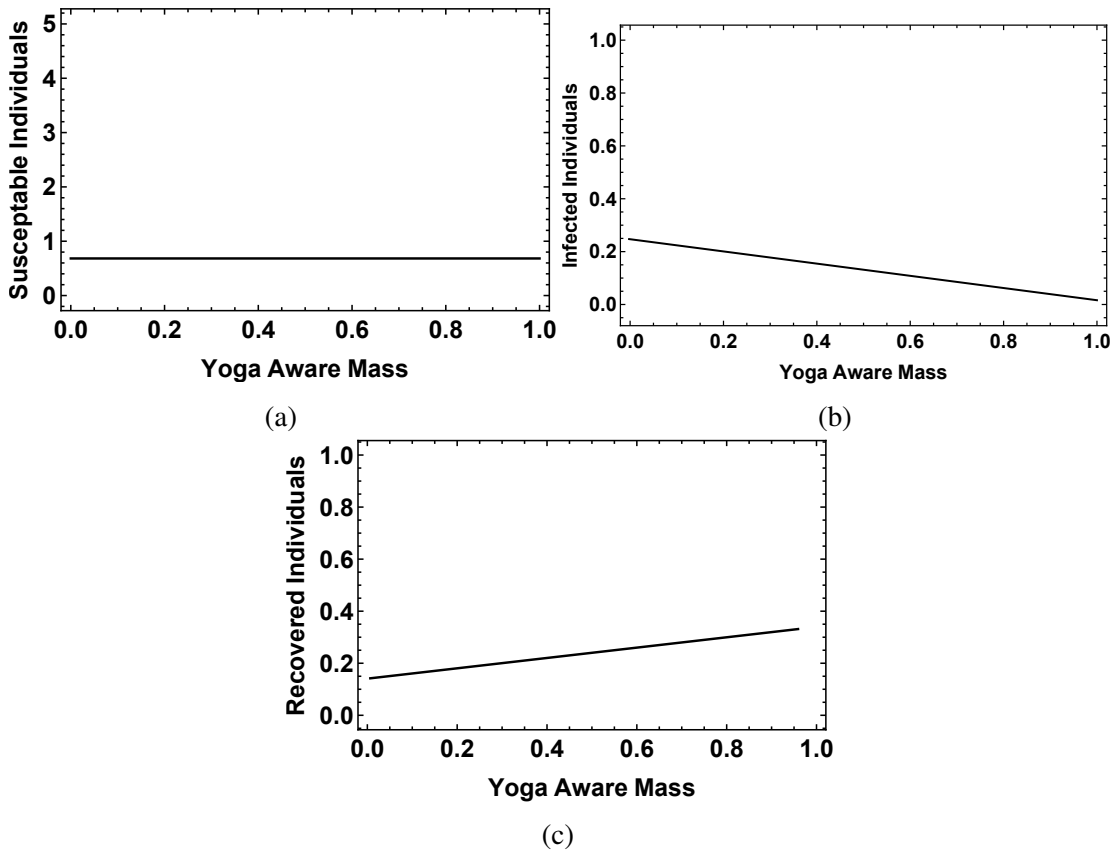
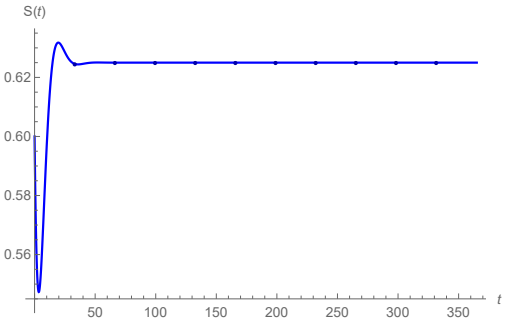
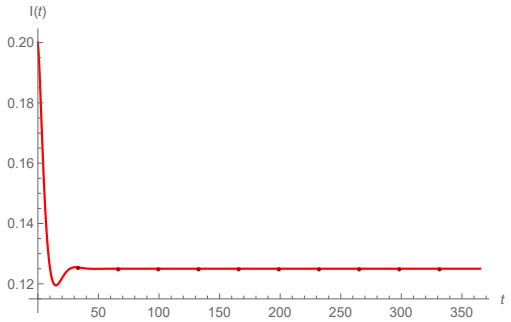


Figure 4.6: Relationship of state variables $S(t)$, $I(t)$, and $R(t)$ with parameters.

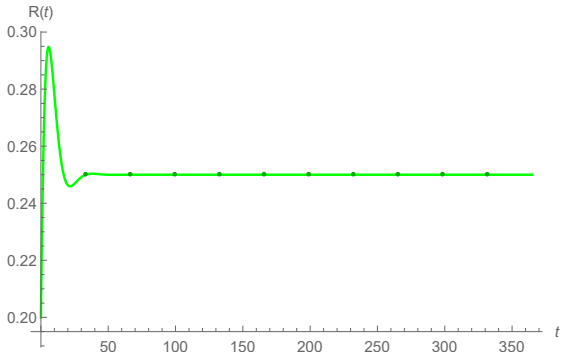
number to model parameters and show its relationship with Yoga-aware mass. These results support the assumptions made in our model. Figure 4.7 depicts the dynamics of susceptible, infected, and recovered individuals, while Figures 4.10(a),(b),and (c) present density plots indicating that Yoga awareness mass $w(t)$ reduces susceptibility and infectivity, gradually leading populations toward steady states. We used $c = 0.5$ and 1 to illustrate the incidence and cumulative incidence of disease dynamics. Higher Yoga awareness coverage corresponds to smaller slopes in the epidemic curves. Figure 4.9 shows the vector field of the disease dynamics, indicating that the system stabilizes near the $I = 0$ line, reflecting decreased infection levels as Yoga awareness increases.



(a) Dynamics of susceptible population over time.



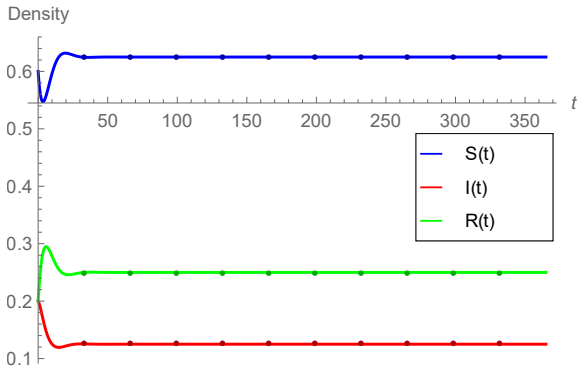
(b) Dynamics of infected population over time.



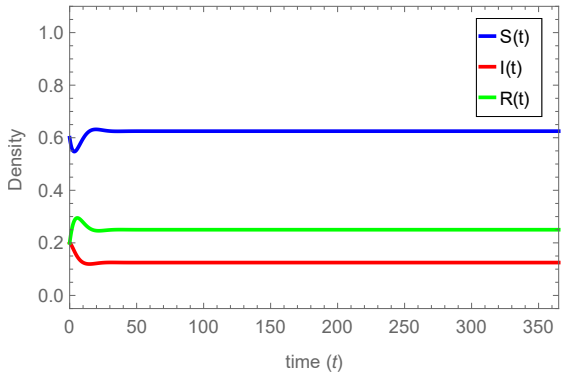
(c) Dynamics if ecovered population over time.

Figure 4.7: Effect of Yogachara on the dynamics of $S(t)$, $I(t)$, and $R(t)$.

Figures 4.10 (a),(b),and (c) further illustrate the relationship between the reproduction number and model parameters, demonstrating that awareness is effective in reducing disease transmission. Figure 4.10(a) shows that as awareness mass and recovery rate increase, the reproduction number decreases. When Yoga awareness is high and transmission rate is low, R_a is minimized; however, a high transmission rate can offset the benefits of awareness. Figure 4.10 (b), and (c) confirms that increased aware mass reduces the reproduction number. Overall, these results align with Figures 4.7 and 4.8, indicating that the model effectively captures the effect of Yoga awareness observed in real-world data. In summary, Yoga awareness reduces susceptibility, decreases infectivity, increases recovery rate, and helps to preserve immunity.



(a) Dynamic behaviour of state variables at DFE.



(b) Dynamic behaviour of state variables at EE.

Figure 4.8: Effect of Yogachara on disease transmission dynamics.

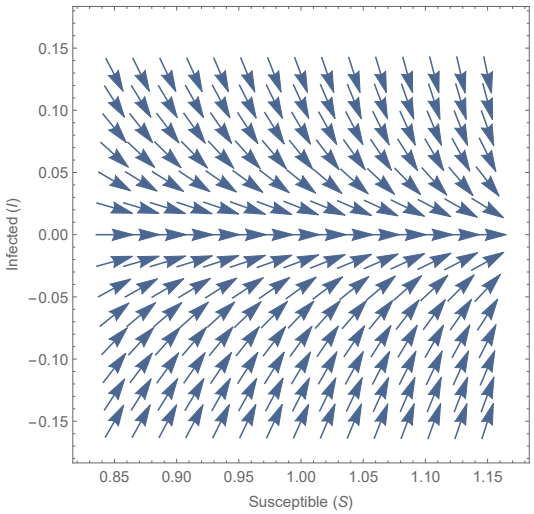
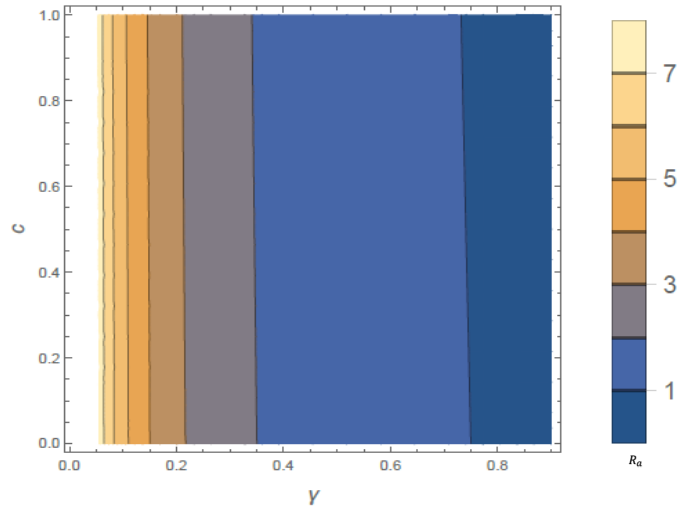
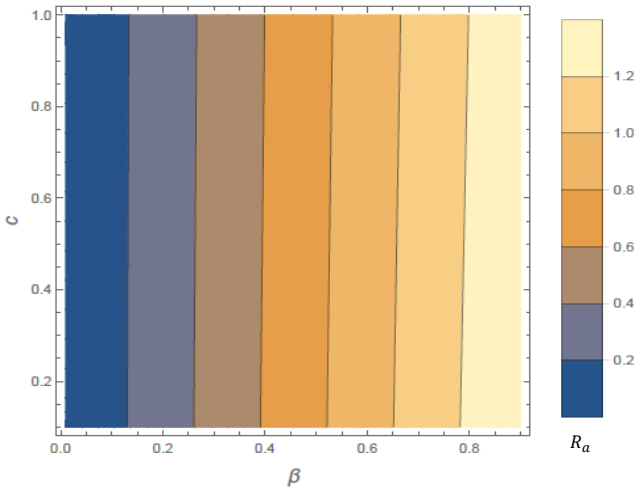


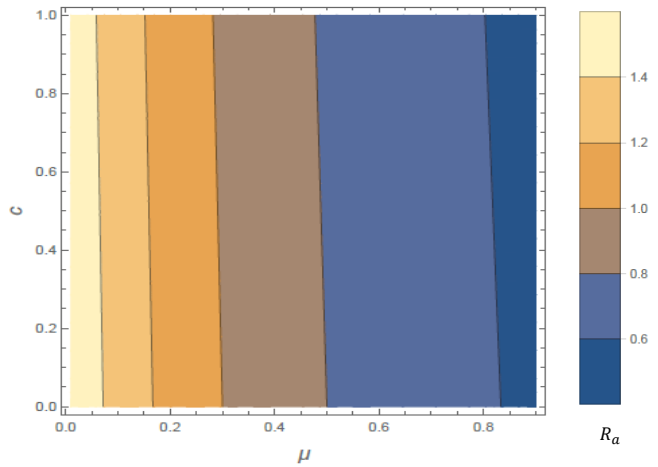
Figure 4.9: System stability as the number of infected individuals approaches zero.



(a) R_a variation with awareness efficacy and recovery rate.



(b) R_a variation with awareness efficacy and transmission rate.



(c) R_a variation with awareness efficacy and average life span.

Figure 4.10: Variation in R_a with parameters.

Discussion

Awareness plays a significant role in modern societies, particularly in the context of communicable diseases. In large and diverse populations, individuals often lack adequate information about disease prevention and holistic living. Within contemporary medical systems, physicians focus on physical treatment, social workers address emotional and social needs, and Yoga Sadhaka counselors provide guidance to purify both the body and the mind. Eastern philosophies, including Buddhism, Hinduism, and traditional Chinese medicine, emphasize a holistic conception of health as a harmonious equilibrium between the individual and the environment. Patanjali's *Yoga Sutra* outlines the eight limbs of yoga, which serve as guidelines for moral conduct, self-discipline, and overall well-being (Hartranft, 2003). Pranayama, described in the *Yoga Sutra*, connects breath, mind, and emotion, and is believed to refresh the body and extend life expectancy (Sivananda, 2019).

In this research, Yoga awareness is considered a control strategy that integrates Asana, Pranayama, and lifestyle practices. A long-term commitment to Yogachara - comprising Aahar (Food), Vihar Relaxation), Aachar (Routines), and Vichar (Thoughts)-aligns with the World Health Organization's definition of health as a state of physical, mental, and social well-being. Yoga Sadhakas provide information through Yoga centers, influencing individual behavior and community awareness. Previous studies have documented the health benefits of Yoga, including

improvements in diet, sleep, stress regulation, and physical fitness (Hagins, Selfe, Innes, et al., 2013; McCall, 2013; Nance, Sease, Crowe, Van Puymbroeck, & Zinzow, 2022; Ross et al., 2012; Sivaramakrishnan et al., 2019). The proposed mechanisms include hormonal regulation, modulation of the sympathetic nervous system, and enhancement of physical attributes such as balance, flexibility, strength, and cardiorespiratory health (Maehle, 2012; Omkar et al., 2011).

Unlike previous studies, this research incorporates Yoga awareness as a control measure that directly reduces the transmission rate (β), thereby influencing the reproduction number (R_a). When $R_a < 1$, the disease-free equilibrium remains stable; when $R_a > 1$, the endemic equilibrium prevails. Yoga awareness contributes to slowing disease spread, supported by both theoretical frameworks and empirical awareness coverage data.

Numerical simulations demonstrate that expanding Yoga awareness coverage lowers infectivity and reduces the final epidemic size. The model yields consistent outcomes across theoretical and data-driven frameworks under the parameters considered. For highly transmissible diseases, results may diverge, underscoring the need for empirical validation.

Conclusion

In this chapter, we developed an $SIRS$ epidemic model incorporating Yogachara (Yoga awareness) and analyzed its local and global stability

at both the disease-free and endemic equilibria. The disease-free equilibrium is locally asymptotically stable when the awareness reproduction number $R_a < 1$, whereas a stable endemic equilibrium emerges for $R_a > 1$, with a transcritical bifurcation occurring at $R_a = 1$. The Yoga awareness mass $w(t)$ influences both R_a and the basic reproduction number R_0, indicating that increased awareness reduces the effective reproduction number and mitigates disease transmission.

Sensitivity analysis and numerical simulations further reveal that Yoga awareness decreases susceptibility and infectivity, accelerates recovery, and extends the duration of immunity. The results illustrated in Figures (4.8)–(4.10) confirm that higher awareness coverage suppresses epidemic spread. Overall, Yoga awareness proves to be an effective behavioral control strategy for strengthening public health and reducing disease propagation.

Chapter 5

Analysis of an SIQS Epidemic Model with a Saturated Incidence Rate Incorporating Yogachara

Preamble

Effective methods for controlling disease spread include vaccination, treatment, and quarantine. Awareness programs can further reduce transmission, and in the previous chapter, Yoga awareness was introduced as a control strategy. This chapter focuses on diseases that do not confer immunity after infection, integrating quarantine with Yogachara (Yoga awareness) as a preventive measure, where Yogachara is considered a form of psychological counseling or lifestyle intervention.

Diseases without permanent immunity can be modeled using the SIS model, where S and I denote susceptible and infectious individuals. The SIS model is fundamental for studying infectious disease dynamics (Martcheva, 2015). Quarantine isolates infectious individuals voluntarily or by mandate and may extend to large populations, restricting movement and activities in crowded places, as determined by local authorities following health recommendations (X. Liu & Yang, 2012).

In the $SIQS$ model, susceptible individuals become infected, some remain in I, and others move into a quarantine class Q (Nuno et al., 2008; L.-I. Wu & Feng, 2000). Various incidence functions, including bilinear, nonlinear, and saturated forms, have been studied to capture real-world transmission dynamics (Alexander & Moghadas, 2004; Reno et al., 2020; X.-B. Zhang & Zhang, 2021). Motivated from previous studies conducted by (H. Hethcote et al., 2002; Joshi, 2020; Q. Zhang & Zhou, 2019), this chapter uses a saturated incidence rate

$$g(S)I = \frac{\beta SI}{1 + \alpha S},$$

which realistically models populations approaching saturation with infectives (R. Anderson & May, 1978; J. Wang & Jiang, 2014; W. Wang et al., 2013). This chapter presents the formulation of the $SIQS$ model with preventive measures, the derivation of equilibrium points and the basic reproduction number, the stability analysis of the disease-free and endemic equilibria, and numerical simulations supporting the theoretical results.

Assumptions and Model Formulation

In the study of epidemic models, the incidence rate plays a vital role. The incidence rate represents the rate at which susceptible individuals become infectious. Various forms of incidence rates have been proposed and analyzed by researchers, including bilinear, nonlinear, standard, sat-

urated, specific nonlinear, and non-monotone types. These formulations have been widely applied in compartmental epidemic models to conduct qualitative and bifurcation analyses. They also describe the psychological effects of severe diseases on the community, particularly when the number of infections increases substantially.

The proposed $SIQS$ model is an extended form of the classical SIS model. It includes four compartments: susceptible individuals (S), infectious individuals (I), and quarantined individuals ($Q(t)$) at any time t. The model is governed by the following nonlinear system of ordinary differential equations:

$$\begin{aligned} \frac{dS}{dt} &= b - \frac{\beta SI}{1+\alpha S} - \mu S + \gamma I + \theta Q \\ \frac{dI}{dt} &= \frac{\beta SI}{1+\alpha S} - (\gamma + \lambda + \mu + d)I \\ \frac{dQ}{dt} &= \lambda I - (\theta + \mu + d)Q \end{aligned} \tag{5.1}$$

The variables and parameters used in the model are defined in

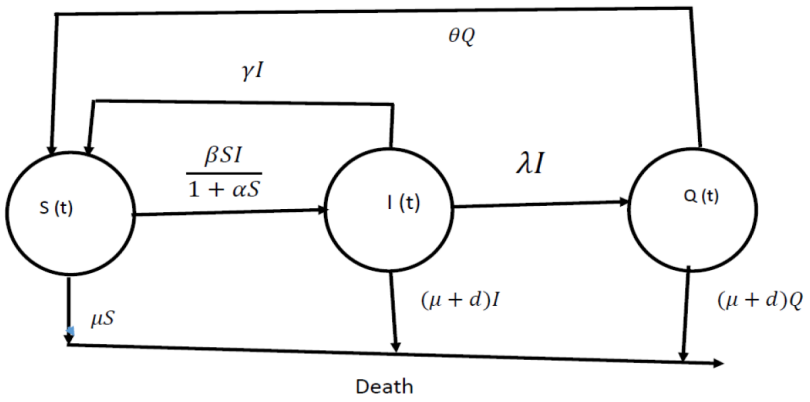


Figure 5.1: General transfer diagram for the $SIQS$ model.

Table 5.1. All parameters are assumed to be positive constants. In the

Variable/Parameter	Definition
b	Recruitment rate (including births and immigration) of susceptible individuals.
μ	Per capita natural death rate.
β	Average number of adequate (sufficient for transmission) contacts per person per unit time.
λ	Rate at which infectious individuals move to the quarantine compartment Q.
d	Disease-related death rate in compartments I and Q.
γ	Recovery rate of individuals returning from I to the susceptible compartment S.
θ	Recovery rate of individuals returning from Q to the susceptible compartment S.
α	Parameter representing preventive measures (e.g., yoga awareness) taken by the susceptible population for epidemic control.

Table 5.1: Description of variables and parameters used in the $SIQS$ model.

model system (5.1), the term $\frac{\beta SI}{1+\alpha S}$ represents a nonlinear saturated incidence rate. Here, βSI measures the force of infection, while $\frac{1}{1+\alpha S}$ captures the psychological effect due to behavioral changes among susceptible individuals (for example, behavioral modifications inspired by Yogachara philosophy). This formulation is essential because the number of effective contacts between infectious and susceptible individuals decreases when the infection level becomes high, either due to quarantine measures or protective behavior among the susceptible population. When $\alpha = 0$, the saturated incidence rate reduces to the standard bilinear form.

Let the total population be $N = S + I + Q$. Differentiating with respect to time gives:

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dI}{dt} + \frac{dQ}{dt} = b - \mu N - d(I + Q)$$

In the absence of disease-related deaths ($d = 0$), this simplifies to:

$$N' = b - \mu N$$

Solving this equation yields:

$$N = \frac{b}{\mu} + e^{-\mu t}\left(N_0 - \frac{b}{\mu}\right)$$

As $t \to \infty$, $N \to \frac{b}{\mu}$, indicating that the total population approaches the carrying capacity $\frac{b}{\mu}$.

Hence, the feasible region for the model (5.1) is defined as:

$$\Omega = \{(S, I, Q) \in \mathbf{R}^3_+ : S, I, Q \geq 0, S + I + Q \leq \tfrac{b}{\mu}\}.$$

Data Source and Parameter Estimation

The data analyzed in this study were primarily obtained from secondary sources, as summarized in Table 5.1. During the onset of the Delta variant wave, the government of Nepal initiated health screening at several border checkpoints, including the Gaddachauki and Trinagar checkpoints in Sudurpashchim Province. A province-wide lockdown commenced on 29 April 2021 and remained in effect until the resumption of intercity bus services and domestic flights. Most policy restrictions were lifted thereafter, except for the reopening of schools, which occurred on 1 September 2021.

The study period extends from 21 March 2021 to 31 August 2022. Throughout much of this time (April 2021–August 2022), many residents practiced *Yogachara* and self-quarantine. However, the Ministry of Health and Population of Nepal (MoHP, 2021a) did not record data specific to *Yogachara* participation or self-quarantine practices. To address this gap, supplementary primary data were collected directly from yoga centers through structured questionnaires administered to principal instructors.

The efficacy parameter for Yogachara is (α), representing a psychological preventive measure, was derived from these primary data. The disease transmission rate (β) was estimated using secondary data sources and contemporaneous published studies.

Results and Discussion

Equilibrium States and Quarantine Reproduction Number

For the equilibrium points of system (5.1), we have

$$\begin{aligned} b - \frac{\beta SI}{1+\alpha S} - \mu S + \gamma I + \theta Q = 0, \\ \frac{\beta SI}{1+\alpha S} - (\gamma + \lambda + \mu + d)I = 0, \\ \lambda I - (\theta + \mu + d)Q = 0. \end{aligned}$$

The system (5.1) always has the disease-free equilibrium (DFE) point

$$E_0 = \left(\frac{b}{\mu}, 0, 0\right).$$

The threshold quantity R_q is defined as the average number of secondary infections produced when one primarily infected individual is introduced into a fully susceptible population (Van den Driessche & Watmough, 2002). This quantity is called the *quarantine reproduction number* because the quarantine process is used to control disease spread. It is a key parameter for determining whether an infection will die out or persist in the population. The expression for R_q is derived following the method in (Van den Driessche & Watmough, 2002).

Let $X = (S, I, Q)$, then

$$X' = \mathcal{F} - \mathcal{V},$$

where

$$\mathcal{F} = \begin{bmatrix} 0 \\ \frac{\beta SI}{1+\alpha S} \\ 0 \end{bmatrix}, \quad \mathcal{V} = \begin{bmatrix} -b + \frac{\beta SI}{1+\alpha S} + \mu S - \gamma I - \theta Q \\ (\gamma + \lambda + \mu + d)I \\ -\lambda I + (\theta + \mu + d)Q \end{bmatrix}.$$

At the DFE $\left(\frac{b}{\mu}, 0, 0\right)$, the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ are

$$F_0 = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{\beta b}{\mu+\alpha b} & 0 \\ 0 & 0 & 0 \end{bmatrix}, \quad V_0 = \begin{bmatrix} \mu & \frac{\beta b}{\mu+\alpha b} - \gamma & -\theta \\ 0 & \gamma + \lambda + \mu + d & 0 \\ 0 & -\lambda & \theta + \mu + d \end{bmatrix}.$$

The next-generation matrix is

$$F_0V_0^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{\beta b}{(\mu+\alpha b)(\gamma+\lambda+\mu+d)} & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

The spectral radius of this matrix gives

$$R_q = \frac{\beta b}{(\mu + \alpha b)(\gamma + \lambda + \mu + d)}.$$

A unique positive equilibrium, known as the endemic equilibrium (EE) point $E^* = (S^*, I^*, Q^*)$, exists in Ω when $R_q > 1$, given by

$$\begin{aligned} S^* &= \frac{b}{\alpha b(R_q - 1) + \mu R_q}, \\ I^* &= \frac{b(\theta + \mu + d)(\alpha b + \mu)(R_q - 1)}{(\mu + \lambda)(\theta + \mu + d)[\alpha b(R_q - 1) + \mu R_q]}, \\ Q^* &= \frac{\lambda I^*}{\theta + \mu + d}. \end{aligned} \tag{5.2}$$

The total population at endemic equilibrium is

$$N^* = S^* + I^* + Q^* = \frac{bd}{(\mu + d)[\alpha b(R_q - 1) + \mu R_q]} + \frac{b}{\mu + d}.$$

When the disease-related death rate $d = 0$, the total population at EE approaches the disease-free carrying capacity:

$$N^* \to \frac{b}{\mu}.$$

Stability Analysis

This section establishes the local and global stability of the model (5.1).

Local Stability Analysis. The local stability of the $SIQS$ model refers to the behavior of the system near its equilibrium points. It describes whether small perturbations (small changes in the number of susceptible, infected, quarantined, or recovered individuals) will die out over time or grow.

Theorem 5.1. *If $R_q < 1$, the disease-free equilibrium E_0 of system (5.1) is locally asymptotically stable. If $R_q > 1$, E_0 is unstable.*

Proof. Let

$$\begin{aligned} M_1 &= b - \frac{\beta SI}{1+\alpha S} - \mu S + \gamma I + \theta Q, \\ M_2 &= \frac{\beta SI}{1+\alpha S} - (\gamma + \lambda + \mu + d)I, \\ M_3 &= \lambda I - (\theta + \mu + d)Q. \end{aligned}$$

The Jacobian matrix of system (5.1) is

$$V(X) = \begin{bmatrix} \frac{\partial M_1}{\partial S} & \frac{\partial M_1}{\partial I} & \frac{\partial M_1}{\partial Q} \\ \frac{\partial M_2}{\partial S} & \frac{\partial M_2}{\partial I} & \frac{\partial M_2}{\partial Q} \\ \frac{\partial M_3}{\partial S} & \frac{\partial M_3}{\partial I} & \frac{\partial M_3}{\partial Q} \end{bmatrix}.$$

At $E_0 = (\frac{b}{\mu}, 0, 0)$, the Jacobian becomes

$$V(E_0) = \begin{bmatrix} -\mu & -\frac{\beta}{\mu+\alpha b} + \gamma & \theta \\ 0 & \frac{\beta}{\mu+\alpha b} - (\gamma + \lambda + \mu + d) & 0 \\ 0 & \lambda & -(\theta + \mu + d) \end{bmatrix}.$$

The characteristic polynomial is

$$|V(E_0) - KI| = (\mu+K)[(\gamma+\lambda+\mu+d-\frac{\beta}{\mu+\alpha b}+K)(\theta+\mu+d+K)] = 0.$$

Two eigenvalues are clearly negative. The third is negative if

$$\frac{\beta}{\mu + \alpha b} < \gamma + \lambda + \mu + d \quad \text{i.e.,} \quad R_q < 1.$$

Hence, E_0 is locally asymptotically stable if $R_q < 1$ and unstable if $R_q > 1$. □

Theorem 5.2. *The endemic equilibrium E^* is locally asymptotically stable if $R_q > 1$.*

Proof. At $E^* = (S^*, I^*, Q^*)$, the Jacobian is

$$V(E^*) = \begin{bmatrix} -\frac{\beta I^*}{(1+\alpha S^*)^2} - \mu & -\frac{\beta S^*}{1+\alpha S^*} + \gamma & \theta \\ \frac{\beta I^*}{(1+\alpha S^*)^2} & \frac{\beta S^*}{1+\alpha S^*} - (\gamma + \lambda + \mu + d) & 0 \\ 0 & \lambda & -(\theta + \mu + d) \end{bmatrix}.$$

Let $A = \frac{\beta I^*}{(1+\alpha S^*)^2}$ and $B = \frac{\beta S^*}{1+\alpha S^*}$. Using the Routh-Hurwitz criteria, all eigenvalues have negative real parts provided $A - B > 0$. Thus, E^* is locally asymptotically stable when $R_q > 1$. □

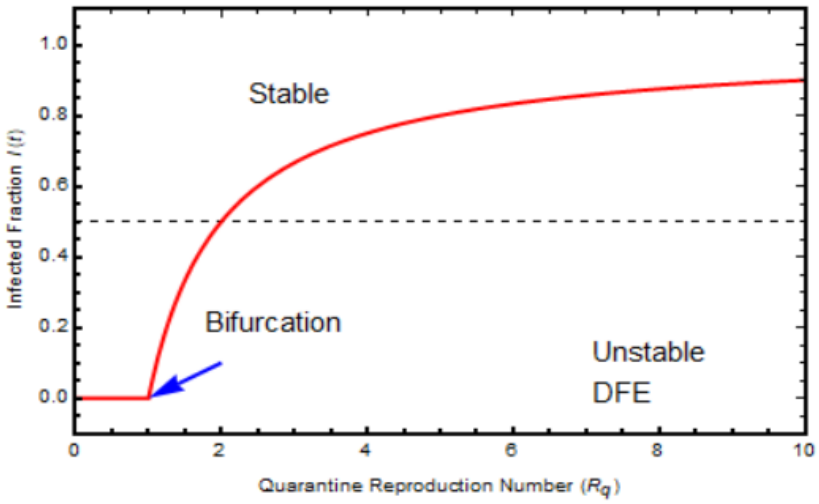


Figure 5.2: Stability of the system: DFE when $R_q < 1$, EE when $R_q > 1$.

Global Stability Analysis. We use Lyapunov global asymptotic stability theorem to test global stability of the system (5.1) (L.-X. Yang & Yang, 2015; Y. Yang & Zhang, 2020).

Theorem 5.3. *Consider nonlinear system x' = f(x), and suppose there exist a function* $L : R^n \to R$ *such that*

- *L is positive definite*
- $L'(z) < 0$ *for all* $z \neq 0$ *and* $L'(0) = 0$

then, every trajectory of $x' = f(x)$ *converges to zero as* $t \to \infty$ *(i.e., the system is globally asymptotically stable)(Nguyen & Nguyen, 2018).*

Theorem 5.4. *If* $R_q < 1$*, then the disease-free equilibrium* E_0 *of the system (5.1) is globally asymptotically stable in the region* Ω*. If* $R_q > 1$ *and* $d = 0$ *(i.e. there are no disease-related deaths), then the endemic equilibrium* E^* *is globally asymptotically stable in the region* $\Omega - (S, I, Q) : I = 0$.

Proof. First, we prove the global stability at the disease-free equilibrium E_0 when $R_q < 1$. Consider a Lyapunov function $L = I$. Then the Lyapunov derivative will be

$L' = I'$

$L' = [\frac{\beta S}{1+\alpha S} - (\gamma + \lambda + \mu + d)]I^*$

$L' \leq [\frac{\beta b}{\mu+\alpha b}(1 - \frac{1}{R_q})]I^*$ since $S \leq \frac{\beta b}{\mu+\alpha b}$

Thus if $R_q < 1$, then $L' \leq 0$ and also $L' = 0$ if and only if $S = \frac{b}{\mu}$, I = 0 and Q = 0. Therefore, $\{E_0\}$ is the largest positive invariant set in $\{(S, I, Q) \in \Omega : L' = 0\}$, where $E_0 = (\frac{b}{\mu+\alpha b}, 0, 0)$ is disease free equilibrium. Thus by Lyapunov's invariant principle, E_0 is globally asymptotically stable in Ω.

To prove the global stability of E^*, when $R_q > 1$ and $d = 0$ (i.e. there are no disease-related deaths), we have $N' = b - \mu N$. This gives $N \to \frac{b}{\mu}$ as $t \to \infty$.

In this case the limit system of (5.1) is given by

$$N' = 0$$

$$I' = [\frac{(\frac{b}{\mu} - I - Q)}{1 + \alpha(\frac{b}{\mu} - I - Q)} - (\gamma + \lambda + \mu)]I \tag{5.3}$$

$$Q' = \lambda I - (\theta + \mu)Q$$

Now, we discuss in the first quadrant of $IQ - plane$. Using Dulac's criteria with multipliers $D_1 = \frac{1}{I}$.

Let

$$F_1 = [\frac{\beta(\frac{b}{\mu} - I - Q)}{1 + \alpha(\frac{b}{\mu} - I - Q)} - (\gamma + \lambda + \mu)]I,$$

$$F_2 = \lambda I - (\theta + \mu)Q.$$

Using multiplier D_1, we get

$$D_1F_1 = [\frac{\beta(\frac{b}{\mu} - I - Q)}{1 + \alpha(\frac{b}{\mu} - I - Q)} - (\gamma + \lambda + \mu)]$$

$$D_1F_2 = \lambda I - (\theta + \mu)\frac{Q}{I}$$

Now,

$$\frac{\partial(D_1F_1)}{\partial I} + \frac{\partial(D_1F_2)}{\partial(Q)} = -\frac{\beta}{[1 + \alpha(\frac{b}{\mu} - I - Q)]^2} - \frac{(\theta + \mu)}{I} < 0$$

Thus, there is no limit cycle, i.e., no periodic solutions exist in the region. Hence by Poincare-Bendixson theory (Kaur & Ahmad, 2014), endemic equilibrium E^* is globally asymptotically stable in the region $\Omega - \{(S, I, Q) : I = 0\}$ for the original system (5.1). □

Sensitivity Analysis and Numerical Results

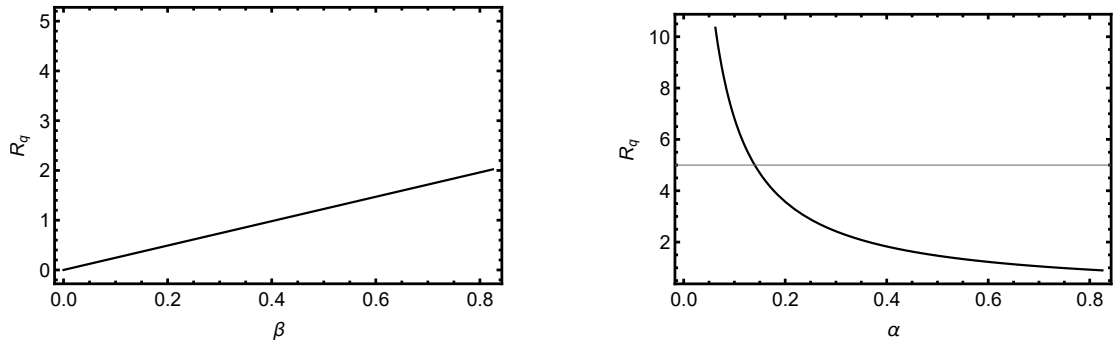
Sensitivity Analysis. Sensitivity analysis examines how changes in model parameters, such as transmission rate or recovery rate, affect the outcome of the model (the reproduction number R_q). It helps identify which factors have the greatest impact on disease spread and which are less influential, guiding better control strategies. For the sensitivity anal-

ysis, we use the parametric values given in Table 5.2.

Parameter	Value Range	Sensitivity Index of R_q	Numerical Value
α	[0.4–1]	$-\frac{\alpha b}{\mu+\alpha b}$	-0.93
β	[0.01–0.8]	1	1
λ	[0.1–0.5]	$-\frac{\lambda}{\gamma+\lambda+\mu+d}$	-0.5
b	[8–10]	1	1
γ	[0.1–0.5]	$-\frac{\gamma}{\gamma+\lambda+\mu+d}$	-0.33
d	[0–0.3]	$-\frac{d}{\gamma+\lambda+\mu+d}$	-0.22
μ	[0–0.2]	$-\frac{\beta b(\alpha b+\gamma+\lambda+2\mu+d)}{(\mu+\alpha b)^2(\gamma+\lambda+\mu+d)^2}$	-1.68
θ	[0.1–0.2]	N/A	N/A

Table 5.2: Parameters used in system (5.1) and corresponding sensitivity indices of R_q.

The sensitivity index of R_q with respect to each parameter is presented in Table 5.2. The relationship between the reproduction number and the parameters is illustrated in Figures 5.3 and 5.4.

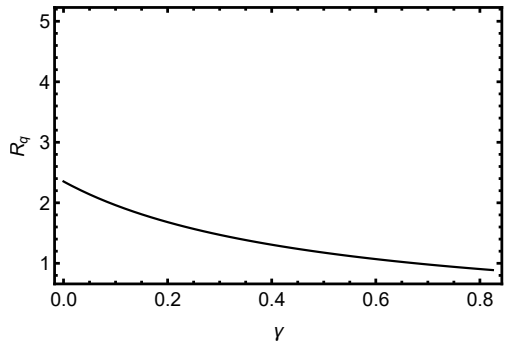


(a) Effect of β on R_q.

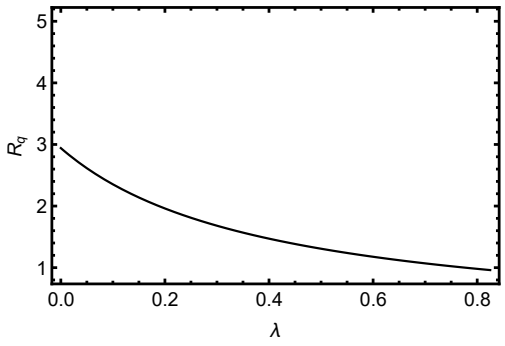
(b) Effect of α (awareness) on R_q.

Figure 5.3: Sensitivity of the quarantine reproduction number R_q to the parameters β and α.

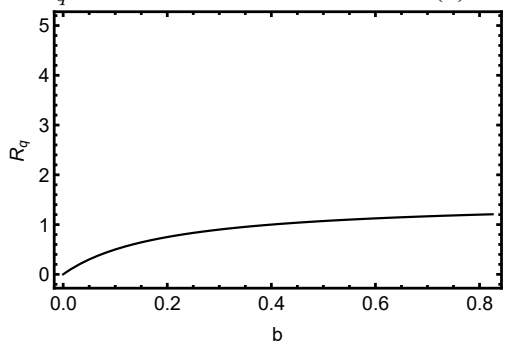
The sensitivity analysis shows that the reproduction number R_q is most influenced by the infection rate (β), the number of susceptible individuals (b), and preventive awareness (α). Increasing β, R_q increases, making the disease spread faster, while higher awareness (Yogachara practices) strongly reduces it. Quarantine rate (λ) and recovery rate (γ) have moderate effects, and other parameters like death rates have



(a) Effect of γ on R_q.



(b) Effect of λ on R_q.



(c) Effect of b on R_q.

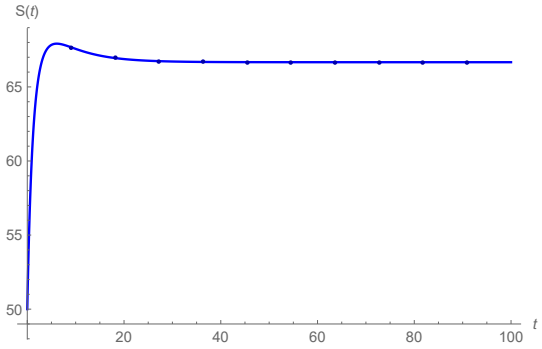
Figure 5.4: Sensitivity of the quarantine reproduction number R_q to the parameters γ, λ, and b.

smaller impacts. This indicates that focusing on reducing contacts, promoting preventive measures, and effective quarantine are the most effective strategies to control disease transmission. Figures 5.3(a) and (b), and 5.4(a), (b), and (c) visually confirm these relationships, showing steep changes in R_q for variations in β, b, and α, and gentler slopes for γ, λ, and d.

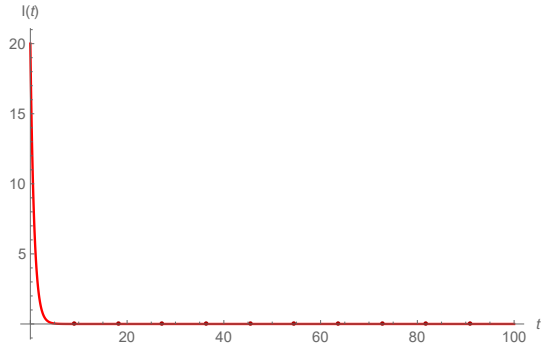
Numerical Simulations. We performed a global analysis of the $SIQS$ model and observed that the basic quarantine reproduction number, R_q, plays a vital role in controlling the disease. The main results indicate that if $R_q < 1$, the disease-free equilibrium is globally stable, whereas if $R_q > 1$, an endemic equilibrium exists and is globally stable. In addition to this analytical study, we provide numerical simulations to

illustrate these dynamics.

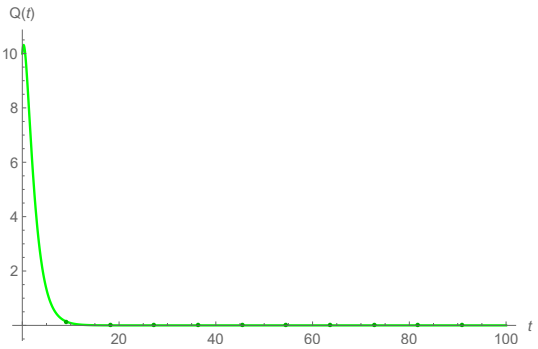
Disease-Free Equilibrium. Using the parameters $b = 10$, $\mu = 0.15$, $\beta = 0.02$, $\gamma = 0.6$, $\alpha = 1$, $\lambda = 0.4$, $d = 0.25$, $\theta = 0.25$, and initial conditions $(S(0), I(0), Q(0)) = (50, 20, 10)$ (from Table 5.2), we obtain $R_q < 1$. In this case, $S(t)$ approaches its steady-state value, while $I(t)$ and $Q(t)$ approach zero as $t \to \infty$, indicating that the disease dies out. The results are illustrated in Figures 5.5 and 5.6.



(a) $S(t)$ over time



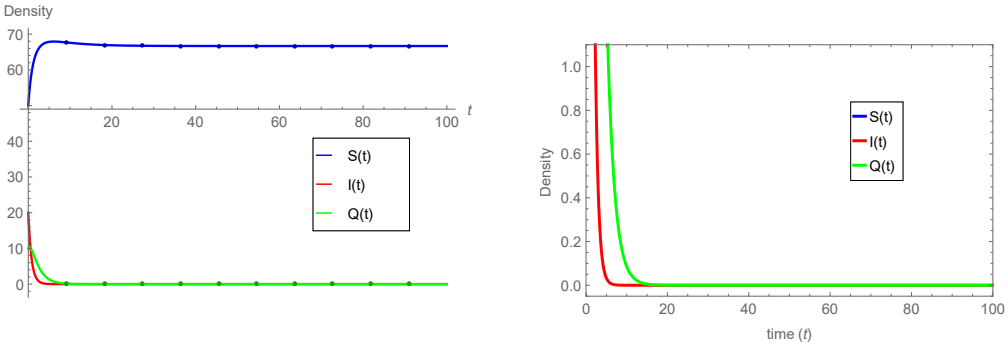
(b) $I(t)$ over time



(c) $Q(t)$ over time

Figure 5.5: Disease dynamics at disease-free equilibrium: $S(t)$ approaches steady state, while $I(t)$ and $Q(t)$ decline to zero, indicating disease die-out.

Endemic Equilibrium. For the parameters $b = 10$, $\mu = 0.1$, $\beta = 0.6$, $\gamma = 0.3$, $\alpha = 0.5$, $\lambda = 0.4$, $d = 0$, $\theta = 0.1$, and initial conditions $(S(0), I(0), Q(0)) = (50, 20, 10)$, we obtain $R_q > 1$. In this case, all compartments $S(t)$, $I(t)$, and $Q(t)$ approach their steady-state values as



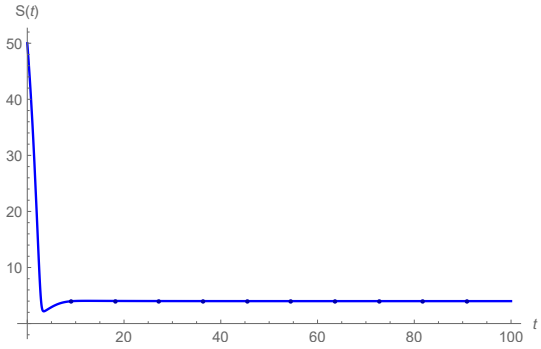
(a) Density plot 1 at disease-free equilibrium (b) Density plot 2 at disease-free equilibrium

Figure 5.6: Density distribution of S, I, and Q at disease-free equilibrium.

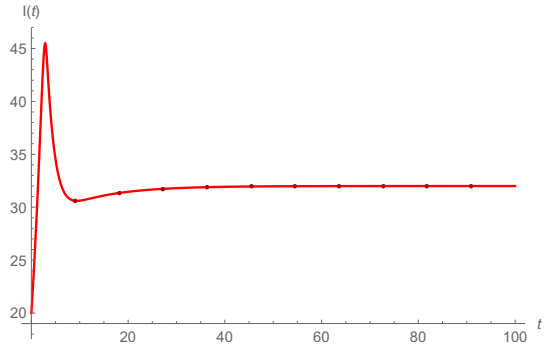
$t \to \infty$, indicating an endemic state. Figures 5.7 and 5.8 illustrate this behavior.

The quarantine process reduces the average infectious period, $\frac{1}{\gamma+\mu+\lambda+d}$, by isolating infectives and preventing transmission. Since the mean residence time in the quarantine class Q is $\frac{1}{\theta}$ and R_q is independent of θ, it confirms the model assumption that individuals in quarantine do not transmit infection. Furthermore, R_q decreases as α increases, indicating that higher disease awareness reduces transmission.

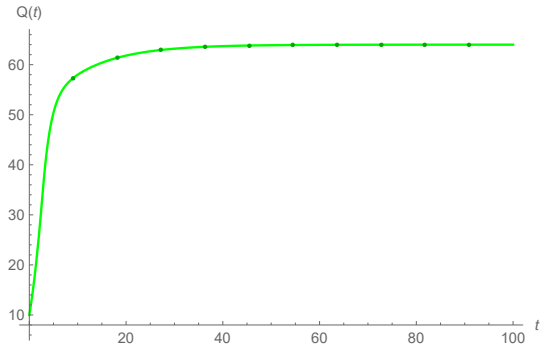
Interpretation of Numerical Results. The numerical simulations of the $SIQS$ model illustrate the role of the quarantine reproduction number, R_q, in determining disease dynamics. When $R_q < 1$, the system converges to the disease-free equilibrium, as shown in Figures 5.5 and 5.6. In this scenario, the susceptible population stabilizes, while the infected and quarantined populations decline to zero, indicating that the disease dies out over time. This result confirms that effective quarantine measures and increased awareness can successfully control disease transmission.



(a) $S(t)$ over time



(b) $I(t)$ over time



(c) $Q(t)$ over time

Figure 5.7: Disease dynamics at endemic equilibrium: all compartments approach steady-state values, indicating disease persistence ($R_q > 1$).

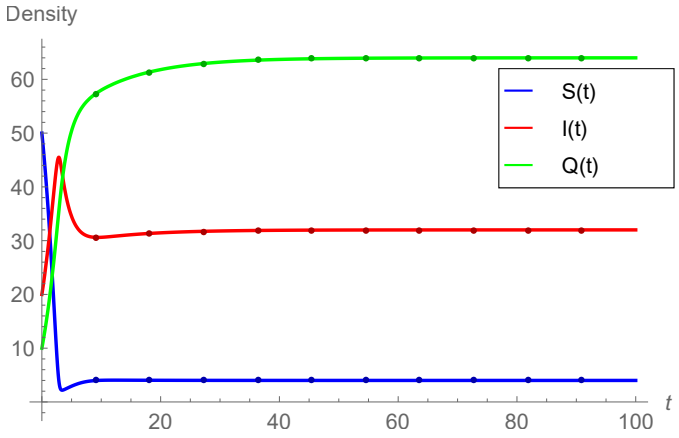


Figure 5.8: Density distribution at endemic equilibrium ($R_q > 1$).

Conversely, when $R_q > 1$, the system reaches an endemic equilibrium, as illustrated in Figures 5.7 and 5.8. In this case, all compartments—susceptible, infected, and quarantined—approach steady-state values, showing persistent disease presence. The simulations demon-

strate that higher values of α, representing increased awareness or preventive measures, reduce R_q and consequently the infection prevalence. Similarly, increasing the quarantine rate decreases the effective infectious period, limiting transmission.

Overall, these results highlight the importance of both quarantine and awareness-based interventions in controlling disease spread. The simulations align with the analytical findings: controlling R_q below unity ensures disease elimination, while $R_q > 1$ leads to a stable endemic state. These insights provide practical guidance for public health strategies aimed at mitigating communicable diseases.

Discussion

In this study, a mathematical model was formulated using ordinary differential equations. Specifically, an $SIQS$ epidemic model incorporating a quarantine strategy and Yogachara under a saturated incidence rate was developed and analyzed. The threshold parameter, the quarantine reproduction number (R_q), was derived to predict the impact of quarantine and disease outcomes within the population. Numerical simulations were performed and verified using *Mathematica* software. The analytical and numerical results are summarized as follows:

(a) When the reproduction number $R_q < 1$, a disease-free equilibrium was observed, indicating that the disease could be effectively controlled through the implementation of quarantine measures combined with Yogachara interventions.

(b) The reproduction number R_q was reduced following the introduction of quarantine as a control measure. Similarly, a negative association between Yogachara and R_q was observed, consistent with the findings of (G. Agaba et al., 2017a).

(c) An endemic equilibrium was identified when $R_q > 1$, particularly in the absence of disease-induced mortality.

(d) Sensitivity analysis indicated that the transmission rate β had a direct positive effect on disease propagation, whereas parameters such as λ, γ, α, and μ exerted negative effects. This implies that increases in the transmission rate lead to greater disease spread, while enhanced awareness (Yogachara) and quarantine measures reduce the reproduction number and, consequently, control the disease. These relationships are illustrated in Figures 5.3 and 5.4.

These findings support the principle that control measures are essential for reducing the reproduction number. When $R_q < 1$, the disease can be eradicated. Therefore, a combined intervention strategy incorporating quarantine and Yogachara was found to be more effective in curtailing epidemics with saturated incidence rates. The model accounted for the isolation of infected individuals in quarantine, thereby preventing their migration and subsequent transmission, which reduces the societal disease burden. These conclusions are in agreement with previous studies (Shah et al., 2021; Q. Zhang & Zhou, 2019; X.-B. Zhang & Zhang, 2021).

Furthermore, preventive measures such as awareness campaigns or psychological interventions were shown to further mitigate disease transmission. Increasing public awareness was found to enhance the immunity of susceptible individuals, thereby lowering infection rates. These results are consistent with prior research (G. Agaba et al., 2017a; G. Agaba, Kyrychko, & Blyuss, 2017b; G. O. Agaba & Soomiyol, 2020; Cui & Wu, 2014; Funk, Gilad, & Jansen, 2010).

It should be noted that the model incorporated a mass-action saturated incidence rate. Analytical results were obtained regarding invariant regions, as well as the existence and stability of equilibria, while numerical simulations provided additional insights into the dynamics of the model that could not be easily derived analytically.

Conclusion

In this study, an $SIQS$ epidemic model with a saturated incidence rate and quarantine strategy was developed and analyzed. The threshold parameter, the quarantine reproduction number (R_q), was derived to evaluate the impact of quarantine measures on disease dynamics.

The analysis shows that when $R_q < 1$, the disease-free equilibrium exists and is globally stable, indicating that the disease will eventually die out. Conversely, when $R_q > 1$, a unique endemic equilibrium exists and is globally stable, indicating that the disease becomes persistent in the population. The model demonstrates that both the effective infectious period and R_q decrease as the quarantine rate λ increases. This implies

that faster isolation of infectious individuals accelerates disease elimination. Since the mean residence time in the quarantine class Q is given by $\frac{1}{\theta}$ and the expression for R_q is independent of θ, the assumption that individuals in quarantine do not transmit the infection and are non-infectious upon leaving the quarantine class is validated.

Furthermore, R_q was found to decrease as the parameter α increases, highlighting that the implementation of psychological protective measures, such as Yogachara (Pranayama and Yoga awareness), reduces disease transmission.

Numerical simulations complement the analytical results (Figures 5.5–5.8), showing that with increased quarantine rates and awareness measures, the susceptible population $S(t)$ stabilizes at its steady-state value, while the infected population $I(t)$ and quarantined population $Q(t)$ approach zero over time. These results collectively indicate that the disease can be effectively controlled and ultimately eradicated through a combination of quarantine interventions and awareness programs.

Chapter 6

Dynamics of Susceptible and Hyper-Susceptible Populations in an Epidemic Model

Preamble

Hyper-susceptibility refers to a condition in which individuals are more prone to infection due to weakened immunity or reduced resistance. In this chapter, the concept is introduced to distinguish between individuals with normal immunity (susceptible) and those with lower immunity (hyper-susceptible). This distinction allows a more realistic representation of disease transmission dynamics, as variations in immunity significantly influence the spread and persistence of infections.

Infectious diseases account for a major share of global morbidity and mortality, particularly in developing countries. These communicable diseases spread through various routes, including air, water, body fluids, and insect vectors, but can be prevented or controlled through vaccination, quarantine, medication, and awareness programs (Castilho, 2006; d'Onofrio, Manfredi, & Salinelli, 2007; Gaff & Schaefer, 2009; Gumel et al., 2004; Kassa & Ouhinou, 2015). Mathematical modeling

plays an important role in understanding disease transmission and guiding control strategies. Numerous epidemic models have been developed to predict outbreaks and inform public health policies. The rate at which susceptible individuals become infected, known as the *incidence rate*, has been represented in several forms—mass action, nonlinear, standard, saturated, and general incidence—to capture diverse epidemic dynamics (Alexander & Moghadas, 2004; Capasso & Serio, 1978; Ruan & Wang, 2003; D. Xiao & Ruan, 2007). Stability analysis of equilibrium states is main part to epidemiological modeling. The Lyapunov method is widely used for this purpose but requires constructing complex auxiliary functions. Alternative approaches include the Poincaré–Bendixson theory and its generalization by Dulac (1937); Y. Li and Muldowney (1993), which eliminate periodic solutions, as well as geometric methods as described by M. Y. Li and Muldowney (1996); Y. Li and Muldowney (1993). The technique proposed by Busenberg and Van den Driessche (1990), based on demonstrating the nonexistence of periodic solutions within the feasible region, is adopted in this study to establish the model's global properties.

Epidemic models that incorporate multiple groups, age structures (often described by partial differential equations), or varying levels of susceptibility have been extensively studied. These models typically include several susceptible classes within a single framework, making the analysis of global stability more challenging than in single-group models. In the present study, the susceptible population is divided into two

compartments: susceptible and hyper-susceptible, based solely on differences in immunity levels, without considering demographic factors such as age or sex. Individuals transition from the susceptible to the hyper-susceptible class as their immunity decreases. Each compartment is assumed to be internally homogeneous, but the level of susceptibility differs between them (Maehle, 2012; Nadholta et al., 2023; Sivananda, 2017; Yeun & Kim, 2021).

Accordingly, the hyper-susceptible group is modeled as a separate compartment. The proposed model, formulated through a system of ordinary differential equations, is structurally analogous to the classical SIS model. It incorporates mass-action interactions among susceptible, hyper-susceptible, and infective populations, along with immigration into both susceptible classes. The primary objective of this chapter is to examine the dynamics of the susceptible and hyper-susceptible populations under these assumptions.

This chapter includes the formulation of the model, calculation of the reproduction number, stability analysis of disease-free and endemic equilibria, and sensitivity analysis. It also presents numerical results, a brief discussion, and concluding remarks.

Assumption and Model Formulation

Assumptions

Epidemic models with Yogachara as a control strategy were discussed in earlier chapters; however, such strategies may not affect all individuals equally due to variations in immunity and personal factors such as age, health status, hygiene, vaccination, and Yoga awareness. In general, non-infected individuals are considered susceptible, possessing normal immune responses to infections. Yet, some individuals have weakened immunity, making them more prone to disease. These are classified as the hyper-susceptible group, which may include the elderly, chronically ill, or immunocompromised persons. Immunocompromised persons are individuals whose immune systems are weakened or not functioning properly, making them more susceptible to infections and certain diseases. Their reduced immune capacity leads to prolonged infections and weaker responses to treatment or vaccination. Regular Yogachara practice, especially Pranayama, can potentially strengthen their immunity and improve health outcomes.

Model Formulation

The dynamics of susceptible, hyper-susceptible, and infected populations are described by the system of ordinary differential equations:

$$\begin{aligned}
\frac{dS}{dt} &= (1-p)A - (1-\pi)\beta SI - \theta S + \gamma I - \mu S, \\
\frac{dH}{dt} &= pA - \beta HI + \theta S + \delta I - \mu H, \\
\frac{dI}{dt} &= (1-\pi)\beta SI + \beta HI - (\mu + \gamma + \delta + d)I
\end{aligned} \tag{6.1}$$

where, S, H, and I denote the susceptible, hyper-susceptible, and infected populations, respectively. All variables and parameters are assumed to be non-negative. The infection rate of susceptibles is reduced

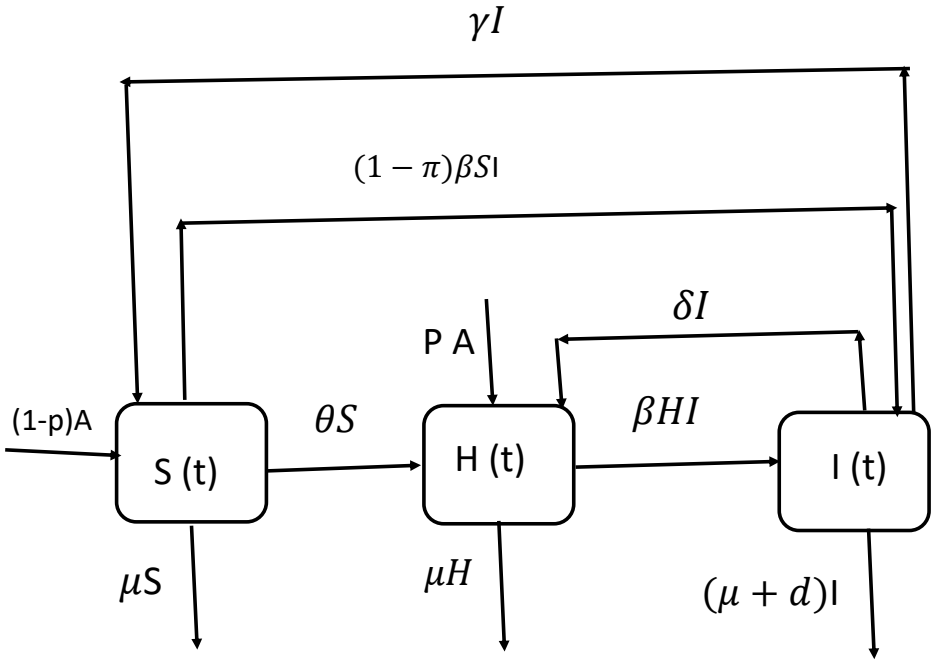


Figure 6.1: Flow diagram of the epidemic model.

by a factor $(1-\pi)$ due to their higher immunity. The total population at

Parameter	Description
A	Recruitment rate of individuals into the population.
p	Proportion of recruited individuals who are hyper-susceptible $(0 \leq p \leq 1)$.
μ	Natural death rate, assumed equal across all compartments.
β	Transmission rate of infection for hyper-susceptible individuals.
θ	Rate at which susceptibles become hyper-susceptible.
π	Protection level of susceptibles due to higher immunity.
γ	Recovery rate of infected individuals returning to susceptible compartment.
δ	Rate at which infected individuals improve and move to hyper-susceptible compartment.
d	Disease-induced death rate in the infected compartment.

Table 6.1: Parameters used in the epidemic model.

any time t is

$$N(t) = S(t) + H(t) + I(t),$$

which evolves as

$$\frac{dN}{dt} = A - \mu N - dI. \tag{6.2}$$

It follows that

$$\lim_{t\to\infty} \sup N(t) = \frac{A}{\mu},$$

ensuring that all solutions of system (6.1) remain bounded within the biologically feasible and positively invariant set

$$\Omega = \{(S, H, I) \in \mathbf{R}^3_+ : S, H, I \geq 0,\ S + H + I \leq \frac{A}{\mu}\}.$$

Results and Discussion

Equilibrium Points and Reproduction Number

This section discusses the two equilibrium points of the model: the *disease-free equilibrium* (DFE) and the *endemic equilibrium* (EE). At the DFE, system (6.1) reduces to

$$\begin{aligned} (1-p)A - (1-\pi)\beta SI - \theta S + \gamma I - \mu S = 0, \\ pA - \beta HI + \theta S + \delta I - \mu H = 0, \\ (1-\pi)\beta SI + \beta HI - (\mu + \gamma + \delta + d)I = 0. \end{aligned} \tag{6.3}$$

Solving at $I = 0$ gives the DFE:

$$S_0 = \frac{(1-p)A}{\mu + \theta}, \quad H_0 = \frac{(p\mu + \theta)A}{\mu(\mu + \theta)}.$$

The reproduction number R_h represents the average number of secondary infections produced by a single infected individual in a fully susceptible population (Van den Driessche & Watmough, 2002). Following the next-generation matrix approach (Van den Driessche & Watmough, 2002), let $X = (S, H, I)$, then

$$X' = \mathcal{F} - \mathcal{V},$$

where

$$\mathcal{F} = \begin{bmatrix} 0 \\ 0 \\ (1-\pi)\beta SI + \beta HI \end{bmatrix},$$

$$\mathcal{V} = \begin{bmatrix} -(1-p)A + (1-\pi)\beta SI + \theta S - \gamma I + \mu S \\ -pA + \beta HI - \theta S - \delta I + \mu H \\ (\mu + \gamma + \delta + d)I \end{bmatrix}.$$

The Jacobian matrices at the DFE are

$$F_0 = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & (1-\pi)\beta S_0 + \beta H_0 \end{bmatrix}, \quad V_0 = \begin{bmatrix} \theta + \mu & 0 & (1-\pi)\beta S_0 - \gamma \\ -\theta & \mu & \beta H_0 - \delta \\ 0 & 0 & \mu + \gamma + \delta + d \end{bmatrix}.$$

The next-generation matrix is $F_0 V_0^{-1}$, and its spectral radius gives the reproduction number:

$$\begin{aligned} R_h &= \frac{\beta H_0 + (1-\pi)\beta S_0}{\mu + \gamma + \delta + d} \\ &= \frac{(p\mu + \theta)\beta A}{\mu(\mu + \theta)(\mu + \gamma + \delta + d)} + \frac{(1-\pi)(1-p)\beta A}{(\mu + \theta)(\mu + \gamma + \delta + d)}. \end{aligned} \tag{6.4}$$

For the endemic equilibrium (EE), solving (6.3) for $I > 0$ gives

$$S = \frac{(\mu + \gamma + \delta + d) - \beta H}{(1-\pi)\beta}$$

$$H = \frac{(1-\pi)\beta(pA + \delta I) + \theta(\mu + \gamma + \delta + d)}{\beta[(1-\pi)(\beta I + \mu) + \theta]}.$$

The infected population I^* satisfies the quadratic equation

$$b_1 I^2 + b_2 I + b_3 = 0, \tag{6.5}$$

where

$$b_1 = (1-\pi)\beta^2(\mu+d),$$
$$b_2 = \beta(1-\pi)\mu(\mu+\delta+d) + \mu(\mu+\gamma+\theta+d) + \theta d - (1-\pi)\beta A,$$
$$b_3 = \mu(\mu+\theta)(\mu+\gamma+\delta+d)(1-R_h).$$

Clearly, $b_1, b_2 > 0$. Thus, no positive endemic equilibrium exists when $R_h \leq 1$. If $R_h > 1$, a unique endemic equilibrium $E^* = (S^*, H^*, I^*)$ exists.

$$\begin{aligned} S^* &= \frac{(\mu+\gamma+\delta+d) - \beta H^*}{(1-\pi)\beta}, \\ H^* &= \frac{(1-\pi)\beta(pA+\delta I^*) + \theta(\mu+\gamma+\delta+d)}{\beta[(1-\pi)(\beta I^*+\mu)+\theta]}, \\ I^* &= \frac{-b_2+\sqrt{B}}{2b_1}, \end{aligned} \tag{6.6}$$

with

$$B = b_2^2 - 4b_1b_3 > 0 \quad \text{for } R_h > 1.$$

Stability Analysis

Stability Analysis at Disease-Free Equilibrium (DFE).

Theorem 6.1. *The disease-free equilibrium (DFE) E_0 of the model (6.1) is locally asymptotically stable if $R_h < 1$, and it is unstable if $R_h > 1$.*

Proof. The Jacobian (variation) matrix of system (6.1) is

$$V(E) = \begin{bmatrix} -(1-\pi)\beta I - \mu - \theta & 0 & -(1-\pi)\beta S + \gamma \\ \theta & -\beta I - \mu & -\beta H + \delta \\ (1-\pi)\beta I & \beta I & (1-\pi)\beta S + \beta H - (\mu + \gamma + \delta + d) \end{bmatrix}.$$

At DFE, we have

$$V(E_0) = \begin{bmatrix} -\mu - \theta & 0 & -(1-\pi)\beta S_0 + \gamma \\ \theta & -\mu & -\beta H_0 + \delta \\ 0 & 0 & (1-\pi)\beta S_0 + \beta H_0 - (\mu + \gamma + \delta + d) \end{bmatrix}.$$

The characteristic equation is

$$(-K-\mu)(-K-\theta-\mu)(-K-d-\gamma-\delta-\mu+\beta H_0+(1-\pi)\beta S_0) = 0.$$

Two eigenvalues are $K_1 = -\mu$ and $K_2 = -(\mu + \theta)$, which are always negative. The third eigenvalue is

$$K_3 = \beta H_0 + (1-\pi)\beta S_0 - (\mu + \gamma + \delta + d),$$

which is negative if and only if

$$\frac{\beta H_0 + (1-\pi)\beta S_0}{\mu + \gamma + \delta + d} = R_h < 1.$$

Hence, the DFE is locally asymptotically stable when $R_h < 1$ and unstable when $R_h > 1$. □

The global stability of the DFE can be established using the method given by Chavez, Feng, and Huang (2002). Rewriting system (6.1) as

$$\begin{aligned} \frac{dX}{dt} &= F_1(X, Y), \\ \frac{dY}{dt} &= F_2(X, Y), \quad F_2(X, 0) = 0, \end{aligned} \tag{6.7}$$

where $X = (S, H)^T$ represents the uninfected population and $Y = I$ represents the infected population. Let $D_0 = (X_0, 0)$ denote the DFE.

Lemma 6.2. *If the conditions*

(C_1) *X is globally asymptotically stable for $\frac{dX}{dt} = F_1(X, 0)$,*

(C_2) *$F_2(X, Y) = DY - \overline{F_2}(X, Y)$ with $\overline{F_2}(X, Y) \geq 0$ and D is an M-matrix,*

are satisfied with $R_h < 1$, then the DFE D_0 is globally asymptotically stable.

Theorem 6.3. *The DFE E_0 of system (6.1) is globally asymptotically stable if $R_h < 1$.*

Proof. Setting $X = (S, H)$ and $Y = I$, and comparing system (6.1) with

(6.7), we have

$$F_1(X,Y) = \begin{bmatrix} (1-p)A - (1-\pi)\beta SI - \theta S + \gamma I - \mu S \\ pA - \beta HI + \theta S + \delta I - \mu H \end{bmatrix},$$

$$F_2(X,Y) = (1-\pi)\beta SI + \beta HI - (\mu+\gamma+\delta+d)I.$$

At $X \to X_0$, $F_2(X,Y) = -(\mu+\gamma+\delta+d)(1-R_h)I - \overline{F_2}(X,Y)$, where $\overline{F_2}(X,Y) \geq 0$. By Lemma 6.2, E_0 is globally asymptotically stable for $R_h < 1$. □

Stability of Endemic Equilibrium (EE).

Lemma 6.4. *(McCluskey & Driessche, 2004) Let T be a 3×3 real matrix. If trace$(T) < 0$, $\det(T) < 0$, and $\det(T^{[2]}) < 0$, then all eigenvalues of T have negative real parts.*

Theorem 6.5. *The endemic equilibrium E^* is locally asymptotically stable if $R_h > 1$.*

Proof. The Jacobian at E^* is

$$V(E^*) = \begin{bmatrix} -(1-\pi)\beta I^* - \mu - \theta & 0 & -(1-\pi)\beta S^* + \gamma \\ \theta & -\beta I^* - \mu & -\beta H^* + \delta \\ (1-\pi)\beta I^* & \beta I^* & (1-\pi)\beta S^* + \beta H^* - (\mu+\gamma+\delta+d) \end{bmatrix}$$

Using $(1-\pi)\beta S^* + \beta H^* = \mu+\gamma+\delta+d$, it follows that trace$(V(E^*)) < 0$, $\det(V(E^*)) < 0$, and $\det(V(E^*)^{[2]}) < 0$. By Lemma 6.4, all eigenvalues have negative real parts, implying that E^* is locally asymptotically stable. □

Moreover, the system (6.1) is uniformly persistent for $R_h > 1$, and the endemic equilibrium E^* is globally asymptotically stable on the invariant set Ω following the method used by Busenberg and Van den Driessche (1990), as no periodic orbits exist within this region.

Sensitivity Analysis and Numerical Results

Sensitivity Analysis. The sensitivity index $S_{R_h}^{y_i}$ quantifies the relative change in the basic reproduction number R_h with respect to a relative change in a parameter y_i:

$$S_{R_h}^{y_i} = \frac{\partial R_h}{\partial y_i} \cdot \frac{y_i}{R_h}.$$

Based on relation (6.4) and the parameter values provided in Table 6.2, the resulting sensitivity indices are also summarized in the same Table 6.2.

Parameter	**Sensitivity** $S_{R_h}^{y_i}$
$\beta = 0.0005$	0.9
$\gamma = 0.05$	-0.4
$\delta = 0.02$	-0.2
$d = 0.01$	-0.1
$\pi = 0.8$	-0.3
$\theta = 0.001$	0.004
$p = 0.2$	0.4
$A = 10$	1.000
$\mu = 0.02$	-0.6

Table 6.2: Sensitivity indices of R_h with respect to model parameters.

The sensitivity analysis shows that R_h is most influenced by the transmission rate β, recruitment rate A, natural death rate μ, recovery rate

γ, and immunity level π. Increasing γ, δ, or π lowers R_h, helping control the disease, while the transition rate θ has little effect. This suggests that interventions should focus on the most sensitive parameters to reduce disease spread effectively.

Numerical Simulations. In this chapter, an epidemic model incorporating a hyper-susceptible compartment with mass-action interactions is formulated and analyzed. The reproduction number, R_h (equation 6.3), is used to govern the global dynamics of the disease within the invariant region Ω. Analytically, it has been shown that when $R_h < 1$, the disease-free equilibrium (DFE) E_0 is globally asymptotically stable, whereas when $R_h > 1$, the endemic equilibrium (EE) E^* is globally stable.

Numerical results obtained using *Mathematica* are presented to support these analytical findings:

(a) For the parameters $A = 10$, $\mu = 0.02$, $\gamma = 0.05$, $\delta = 0.02$, $\beta = 0.0005$, $p = 0.2$, $\theta = 0.001$, $d = 0.01$, and $\pi = 0.8$, R_h is calculated as $0.9762 < 1$. A globally stable DFE, $E_0 = (381, 119, 0)$, is observed, indicating that the disease is eradicated (Figure 6.2).

(b) For the parameters $A = 10$, $\mu = 0.02$, $\gamma = 0.04$, $\delta = 0.02$, $\beta = 0.0006$, $p = 0.2$, $\theta = 0.001$, $d = 0.02$, and $\pi = 0.6$, R_h is calculated as $1.8095 > 1$. A globally stable EE, E^*, is observed, and the disease is maintained in the population (Figure 6.3).

(c) Sensitivity analysis (Table 6.2) indicates that an increase in π reduces R_h, thereby decreasing the infected population. When sus-

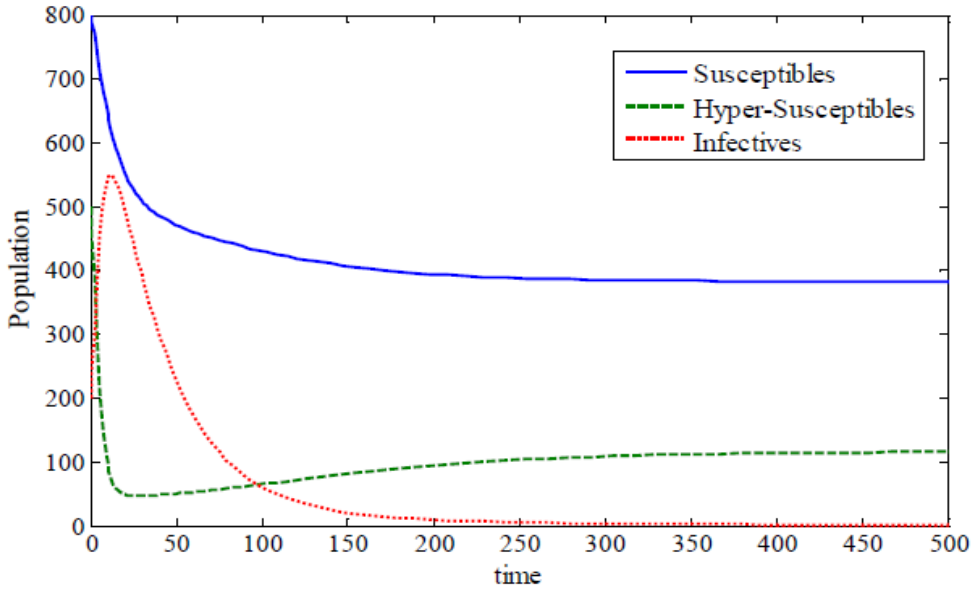


Figure 6.2: Time evolution of the sub-populations for parameters $A = 10$, $\mu = 0.02$, $\gamma = 0.05$, $\delta = 0.02$, $\beta = 0.0005$, $p = 0.2$, $\theta = 0.001$, $d = 0.01$, $\pi = 0.8$, with $R_h = 0.9762 < 1$.

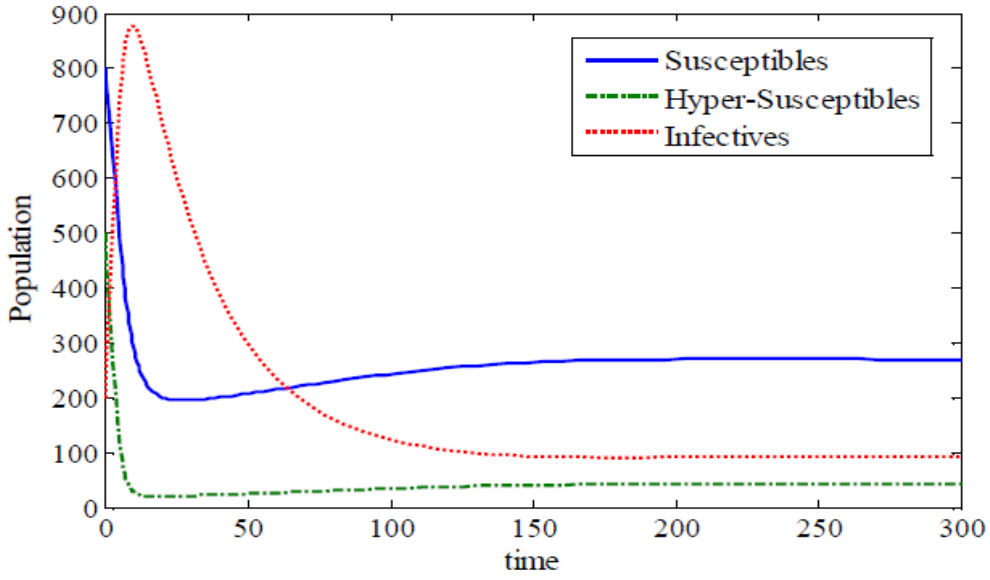


Figure 6.3: Time evolution of the sub-populations S, I, and H for the parameter values $A = 10$, $\mu = 0.02$, $\gamma = 0.04$, $\delta = 0.02$, $\beta = 0.0006$, $p = 0.2$, $\theta = 0.001$, $d = 0.02$, and $\pi = 0.6$, with $R_h = 1.8095 > 1$.

ceptibles recover at a higher rate than hyper-susceptibles, effective disease control is achieved. A decrease in θ, the rate at which susceptibles become hyper-susceptible, is also found to reduce the

equilibrium infection level. These results emphasize the importance of stronger immunity among susceptibles.

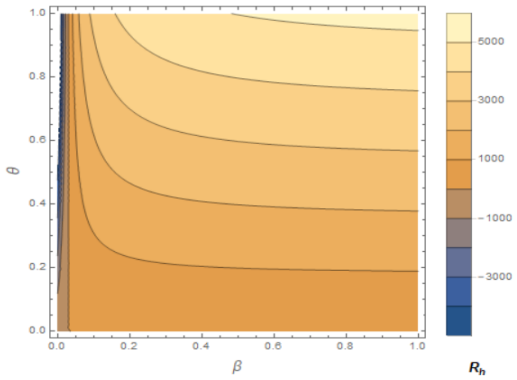
(d) Graphs (Figure 6.2–6.3) and counter plots (Figures 6.4–6.5) illustrate the effects of parameters on R_h:

- An increase in β or θ is observed to elevate R_h, enhancing disease spread (Figures 6.4a–6.4b).
- Higher protection of susceptibles (π) combined with lower hyper-susceptibility reduces R_h (Figures 6.4c–6.4d).
- A restriction in the proportion of hyper-susceptible recruits (p) is found to mitigate disease propagation (Figures 6.5a and 6.5c).
- Variation in β and δ affects transmission intensity (Figure 6.5b).

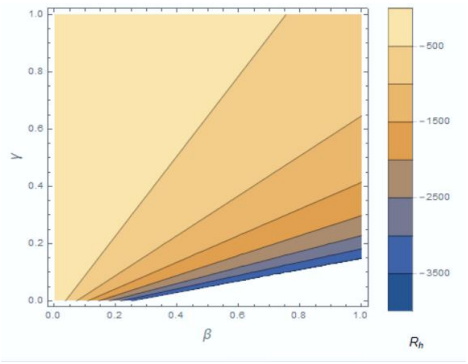
In summary, it is observed that increasing protection (π) and reducing the transition rate (θ) significantly decrease R_h, lowering the infected population and promoting disease control. Furthermore, the restriction of the proportion of hyper-susceptible recruits (p) contributes to suppression of disease spread. The counter plots collectively illustrate the combined effects of parameters on disease transmission dynamics.

Discussion

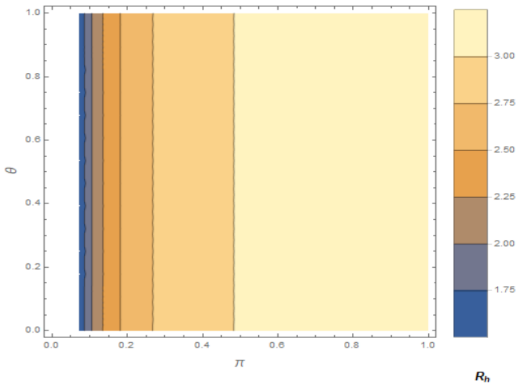
An epidemic model with two different stages of susceptibility (susceptible and hyper-susceptible) has been formulated and analyzed qualitatively. The model incorporates mass-action incidence and immigration



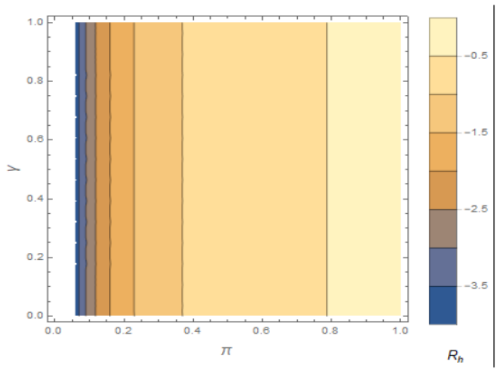
(a) Effect of β and θ on R_h



(b) Effect of β and γ on R_h

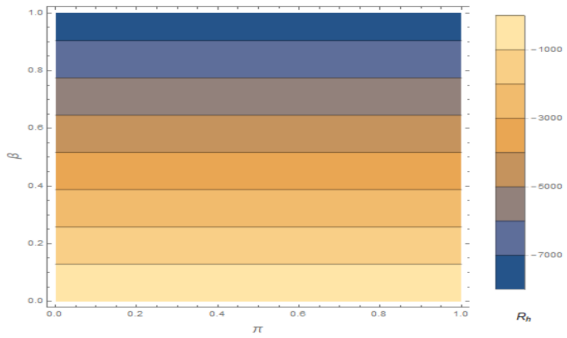


(c) Effect of π and θ on R_h

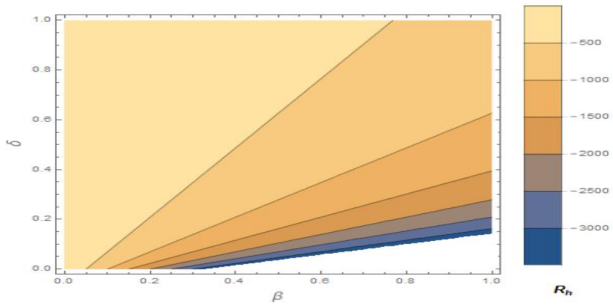


(d) Effect of π and γ on R_h

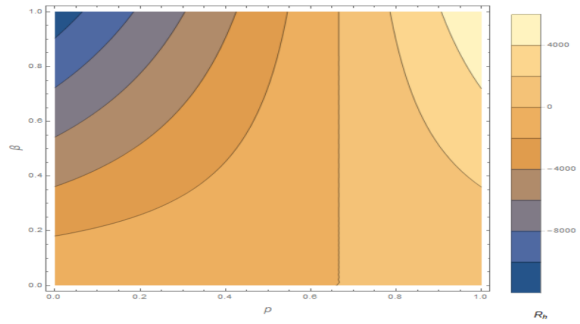
Figure 6.4: Effects of transmission and protection parameters on R_h.



(a) Effect of p and β on R_h



(b) Effect of β and δ on R_h



(c) Effect of p and β on R_h

Figure 6.5: Effects of recruitment, transmission, and removal parameters on R_h.

in both compartments. The reproduction number (threshold), R_h, was derived, and a rigorous mathematical analysis of the proposed model was conducted. It was shown that if this threshold number is less than one, the disease-free equilibrium is globally stable; if it is greater than one, the endemic equilibrium is globally stable. Analytical and simulation results demonstrated the effect of stronger immunity among the susceptible population compared with the hyper-susceptible population. The results also indicate that immigration into the hyper-susceptible class should be restricted to control the spread of the disease. This can be achieved through protective measures such as Yoga Pranayama practices, awareness campaigns, isolation, quarantine, and vaccination of immigrants. If $\pi = 0$, then

$$R_h = \frac{\beta A}{\mu + \gamma + \delta + d}.$$

In this case, R_h is independent of p and θ, and the model reduces to a simple SIS model. The model was deliberately kept simple, focusing only on the effects of immunity levels and immigration in the hyper-susceptible compartment, which facilitated a tractable mathematical analysis. Incorporating additional factors such as age or sex would have required a more complex multi-group modeling approach, as a hyper-susceptible group could not be addressed with a single equation.

Extending this study to emerging viral diseases could improve predictions and strengthen healthcare system preparedness for future pandemics.

Conclusion

In this chapter, an epidemic model with mass-action interactions, consisting of susceptible, hyper-susceptible, and infected compartments, was investigated. The threshold number, R_h, was identified as the key parameter governing the global dynamics of the disease, determining whether it is eliminated or persists in the population. Mathematical analysis of the model revealed that increases in π result in a decrease in R_h, thereby reducing the size of the infected population and facilitating disease control. It was also observed that the equilibrium level of the infected population decreases as θ decreases, that is, as the flow of individuals from the susceptible to the hyper-susceptible compartment is reduced. These findings highlight the effect of stronger immunity in the susceptible population relative to the hyper-susceptible population.

Furthermore, it was shown that restricting the immigration rate, p, into hyper-susceptible communities can reduce disease transmission. Protective measures, such as isolation and vaccination of immigrants, can be implemented to achieve this reduction. Simulation results were found to be consistent with the theoretical findings and illustrated the dynamics of both susceptible and hyper-susceptible populations.

The global stability of the model was analyzed, and it was demonstrated that stability depends critically on the reproduction number, R_h, which plays a central role in disease control. The main results indicate that when $R_h < 1$, the disease-free equilibrium, E_0, is globally asymp-

totically stable. When $R_h > 1$, E_0 becomes unstable, and the endemic equilibrium, E^*, exists and is globally asymptotically stable. Analytical findings were further supported by numerical simulations, which confirmed the predicted dynamics of disease spread and control.

Chapter 7

Findings and Conclusions

Preamble

This chapter summarizes the principal findings of the dissertation and outlines several prospective research directions for future investigation, as well as potential extensions of the present work in areas of national significance. The dissertation primarily investigates the dynamics of communicable diseases and proposes effective prevention strategies to mitigate their spread through the formulation and analysis of deterministic compartmental epidemic models. Additionally, the potential role of Yoga philosophy in disease prevention has been examined, highlighting its relevance as a complementary preventive approach.

The transmission dynamics of communicable diseases are influenced by several factors, including the population growth rate of a given community or nation, the incidence rate, the recovery rate of infected individuals (through natural recovery, treatment, or awareness), the rate at which recovered individuals lose immunity, and the mortality rate of the population (both natural and disease-induced). Considering these factors, four distinct epidemic models were formulated and analyzed in this

study. Specifically, Chapters 3 and 4 focus on the $SYIRS$ model and an improved SIR model incorporating Yogachara principles, whereas Chapters 5 and 6 address the $SIQS$ and SIS models, which incorporate saturated incidence rates and population hyper-susceptibility, respectively. The primary focus of this research is to examine the impact of yoga on the transmission dynamics of communicable diseases through mathematical modeling. The major findings of this study are summarized below.The primary focus of this research is to examine the impact of yoga on the transmission dynamics of communicable diseases through mathematical modeling. The major findings of this study are summarized below.The primary focus of this research is to examine the impact of yoga on the transmission dynamics of communicable diseases through mathematical modeling. The major findings of this study are summarized below.

Findings

The primary focus of this research is to examine the impact of yoga on the transmission dynamics of communicable diseases through mathematical modeling. The major findings of this study are summarized below.

1. Yoga Pranayama has been found to decrease disease transmission dynamics. It reduces susceptibility and infectivity, accelerates recovery, and helps maintain immunity for a longer duration.

2. Yoga awareness reduces the disease transmission rate. Therefore,

incorporating Yoga awareness practices can contribute significantly to controlling the spread of infectious diseases.

3. Quarantine serves as one of the most effective non-pharmaceutical interventions for disease control. The expression for the quarantine reproduction number, R_q, indicates that its value decreases as the awareness parameter α increases. Hence, the spread of disease can be further controlled by integrating Yogachara (or psychological protective measures) alongside quarantine.

4. Individuals in society do not possess the same level of immunity to combat disease. Some individuals are hyper-susceptible. Stronger immunity among susceptible individuals facilitates faster recovery compared to hyper-susceptible ones. Immunity can be enhanced through protective measures such as Yogachara, awareness, isolation, and vaccination.

5. Non-pharmaceutical interventions such as awareness and self-quarantine, when combined with the Yoga philosophy (Yogachara), can effectively prevent or control the propagation of disease. These findings also contribute to the ongoing discourse among public health policymakers regarding the integration of traditional practices with modern preventive strategies.

Conclusions

This dissertation explores the contemporary application of mathematical modeling in analyzing the causes, genesis, rate of spread, and prevention strategies of epidemics. As part of this applied research, it aims to integrate Yoga and Yoga awareness among policymakers to facilitate the development of effective policies for reducing the transmission dynamics of infectious diseases. In this respect, the work initiates a discourse among researchers and professionals involved in public health policy, health improvement programs, control strategies, and human development challenges.

A wide range of literature on "Mathematical Modeling in Epidemiology" has been reviewed and critically evaluated prior to undertaking this research. The present work explores the application of mathematical modeling in epidemiology in conjunction with the efficacy of Yoga. In contemporary scientific inquiry, mathematical biology has emerged as a crucial discipline for validating and parameterizing the dynamics of communicable diseases. Mathematical models serve as essential tools for understanding real-world problems, developing predictive frameworks, and providing quantitative insights that complement experimental and empirical studies.

In the context of mathematical modeling, the SIR model has been applied in Chapters 3 and 4. The SIR model is further extended to an $SYIRS$ model in Chapter 3 by introducing a new Yoga Sadhaka com-

partment and by incorporating the Yoga awareness effect term $\beta e^{-cM(t)}$ into the transmission rate of communicable diseases in Chapter 4. Extended forms of the SIS epidemic model are used in Chapters 5 and 6. The analysis of these mathematical models employs advanced mathematical techniques such as the next-generation matrix operator, Routh–Hurwitz stability criteria, Lyapunov theorem, and Dulac's criterion. The simulation tool *Mathematica* has been used to obtain numerical results and graphical representations.

Mathematical modeling has been utilized to study the impact of control and prevention strategies on reducing mortality caused by communicable diseases. This approach contributes to achieving the objectives of the research and addresses related questions concerning human health improvement and epidemiological demography. The study is directly linked to issues associated with declining immune function in populations. Further comprehensive and multidisciplinary studies are needed to enhance public health programs, strategies, and policies by integrating them with the philosophy of Yoga. The spread of communicable diseases has long posed a serious threat to public health, affecting not only human survival but also economic and social development. The incorporation of Yoga efficacy as a preventive tool in this study highlights its potential significance for public health management.

The primary focus of this dissertation has been to explore the efficacy of Yoga in preventing communicable diseases. Yoga has been employed as a complementary health practice and is considered a holis-

tic remedy addressing physical, mental, and emotional well-being. In the modern world, where lifestyles have become increasingly complex, people suffer from numerous ailments and disorders. The concept of Yogachara used in this research has proven beneficial as a preventive strategy that is widely applicable, easy to implement, and cost-effective. Unlike allopathic or ayurvedic treatments that may not be universally effective, yogachara offers a holistic paradigm—viewing an individual as an integrated entity comprising physical, vital, emotional, and spiritual dimensions (*Pancha Kosha*). Mathematical modeling has been used to examine the impact of control and prevention strategies on reducing mortality from communicable diseases. This approach supports the objectives of the research and addresses questions related to human health improvement and epidemiological demography. The study is directly connected to issues arising from declining immune function in populations. Further comprehensive and multidisciplinary research is needed to strengthen public health programs, strategies, and policies by integrating them with the philosophy of Yoga. Communicable diseases have long posed a serious threat to public health, affecting not only human survival but also economic and social development. Incorporating Yoga efficacy as a preventive tool in this study underscores its potential significance for public health management.

The first chapter introduces the basic and philosophical concepts of Yoga, along with the terminology and foundational ideas used throughout the dissertation. Chapter 2 presents mathematical preliminaries and

the essential concepts of mathematical epidemiology, including key terminology relevant to communicable diseases.

In Chapter 3, an $SYIRS$ epidemic model incorporating Yoga Pranayama has been developed. The disease-free and endemic equilibria have been analyzed in terms of the basic and Yoga reproduction numbers. In this model, the parameter σ represents the efficacy of Yoga, while m denotes the rate of increase of Yoga Sadhaka individuals. Sensitivity analysis and simulation results indicate that the steady-state values of infected individuals (I^*) decrease as the number of Yoga Sadhaka increases. Both R_e and I^* are found to decrease with increasing m, implying that disease spread diminishes with higher Yoga participation. These findings confirm that Pranayama is an effective means of disease prevention and control.

Chapter 4 extends the SIR epidemic model by incorporating a Yoga awareness term in the transmission rate, assuming that it decreases as awareness increases. The modified transmission rate $\beta_1 = \beta e^{-cM}$ is introduced, where β_1 declines as the Yoga awareness mass $w(t)$ increases, thereby reducing disease transmission. The reproduction number R_a is derived, governing the disease dynamics: if $R_a < 1$, the disease-free equilibrium is stable and infection vanishes; if $R_a > 1$, an endemic equilibrium exists and infection persists. The mathematical analysis demonstrates that the implementation of a Yoga awareness program effectively reduces the reproduction number, leading to a lower average number of secondary infections. Numerical simulations validate this model, showing that without awareness, the infected population re-

mains high, while increasing awareness significantly reduces infection levels. These results establish that Yoga awareness plays a crucial role in preventing and controlling diseases in modern society.

Certain communicable diseases do not confer lasting immunity, allowing reinfection. In such cases, SIS-type epidemic models are appropriate. Accordingly, Chapter 5 formulates an $SIQS$ model by introducing a quarantine compartment Q into the SIS epidemic framework, incorporating Yogachara (psychological protective measures) at a saturated incidence rate. The quarantine reproduction number R_q is derived to analyze the effects of quarantine and awareness on disease transmission. If $R_q < 1$, the disease-free equilibrium exists and is both locally and globally stable, implying eradication of the disease. If $R_q > 1$, an endemic equilibrium arises and remains stable, indicating persistence of the disease. Analysis shows that both the effective infectious period and R_q decrease as the quarantine rate λ increases, meaning that rapid quarantine leads to disease extinction. Since the mean residence time in the quarantine class Q is $\frac{1}{\theta}$ and R_q is independent of θ, individuals leaving quarantine are assumed non-infectious. Furthermore, R_q decreases as the awareness parameter α increases, demonstrating that enhanced psychological protective measures (such as Yoga and awareness) reduce disease transmission. Numerical simulations support the analytical results.

Chapters 3, 4, and 5 analyze epidemic models incorporating Yoga Pranayama, Yoga awareness, and quarantine with Yogachara, respectively. However, in real-world settings, these control measures are not

equally effective for all individuals due to variations in health conditions and immunity levels. Consequently, Chapter 6 develops a dynamical model to study hyper-susceptibility versus susceptibility among individuals. This model, an extension of the SIS framework, accounts for differences in immunity and protective behavior. The threshold number R_h governs the global disease dynamics and determines whether infection is eliminated or persists. Mathematical analysis reveals that increasing the protective level π decreases R_h, thereby reducing the size of the infected population. Similarly, the equilibrium level of infection declines as θ decreases, meaning that a reduced flow from the susceptible to the hyper-susceptible class mitigates disease spread. The results highlight that improved immunity among susceptible individuals reduces infection levels. Moreover, limiting the immigration rate p into the hyper-susceptible class can further control disease spread—achievable through protective measures such as Yoga awareness, isolation, and vaccination. Simulation outcomes align with theoretical findings and clearly demonstrate the dynamics of susceptible and hyper-susceptible populations.

Future Research Directions

The present study entitled "Mathematical Modeling and Epidemic Analysis of Communicable Disease with Yoga" is an attempt to develop new mathematical models that contribute to the prevention of communicable diseases through the application of the profound Eastern philosophy of Yoga. In today's globalized world, people frequently migrate across countries for employment, business, and other human needs, resulting in cultural exchange as well as increased risk of disease transmission. It is, therefore, imperative to recognize that outbreaks of communicable diseases originating in a specific region or country can easily spread worldwide due to high population mobility.

Nepal, being geographically diverse and situated near the tropics with both plains and mountainous terrains, experiences distinct climatic conditions influenced by the monsoon cycle. Seasonal variations such as winter and summer create different environmental settings not only for the survival of viruses (the primary cause of many diseases) but also for their transmission across different topographical regions.

The present research, which integrates mathematical modeling with Yoga awareness, provides a foundation for future studies among mathematical researchers, medical practitioners, and national health policymakers. Although this dissertation has made significant progress in improving existing mathematical models, much remains to be explored. The following directions may serve as potential avenues for future in-

vestigation:

1. Future research can focus on extending these models to study specific communicable diseases, incorporating their unique transmission and recovery characteristics.

2. This dissertation employs deterministic models only. Future work should involve the development and analysis of stochastic epidemic models that incorporate randomness and uncertainty, particularly with Yoga-related behavioral parameters.

3. Further studies should examine the quantitative impact of Yoga practices on the recovery rates of specific diseases through the inclusion of empirical and clinical data.

4. In Chapter 4, the incidence rate is modeled as $\beta e^{-cM} SI$. Future research could explore generalized incidence functions with higher powers of M, or other nonlinear forms, by fitting more comprehensive and authentic datasets that include various protective and behavioral measures.

5. The modeling framework presented here can be extended to analyze the effect of Yoga Pranayama on non-communicable diseases such as diabetes, cancer, and asthma, thereby broadening its interdisciplinary applicability.

6. Similar modeling approaches can be applied to social issues such as corruption, alcoholism, and drug addiction, using dynamical models

to study their transmission and control mechanisms.

Future Research Topics

In this dissertation, SIR and SIS epidemic models have been employed with standard incidence rates. In future studies, modified SIR models can be used to investigate the effects of Yogachara under saturated incidence rates. Additionally, non-monotone incidence rates can be incorporated into various epidemic models, including SIR, SI, $SIRS$, $SEIR$, and SIS, to examine the influence of awareness and behavioral interventions more comprehensively. Some possible directions for future research include:

1. Mathematical Modeling and Analysis of Non-Communicable Diseases with Yoga.
2. Effect of Yoga Awareness on Recovery Rate of Infectious Diseases
3. Modeling the Impact of Mass Education and Yogachara on Corruption Dynamics

Contribution of the Research to National Health Policymakers

This research can support the improvement of public health through coordinated policy actions across sectors and the expansion of preventive, promotive, curative, palliative, and rehabilitative Yoga services delivered through public health institutions (e.g., Yoga centers), with an emphasis on enhancing quality of life. The key contributions of this research to

health policymakers are summarized as follows:

1. Communicable diseases not only pose serious threats to human survival but also adversely impact the economic and social development of society. The spread of such diseases has always been a significant public health challenge. Mathematical modeling of disease dynamics can help minimize social and economic burdens and guide policymakers in formulating effective economic strategies, providing reliable predictions, and supporting evidence-based decision making.

2. The research informs policies that emphasize investment and action in school health by integrating Yoga into health education curricula, promoting hygiene and safe health practices, and establishing schools as sites for primary health care. Promotion of healthy living strategies and Yoga in workplaces is also crucial, particularly in the Nepalese context. Expanding Yoga related interventions in school education can aid in the prevention of both communicable and non-communicable diseases.

3. Analysis of these models provides policymakers with insights into the sociological, biological, and environmental factors influencing disease transmission. Consequently, policies can be designed to incorporate institutional mechanisms that foster social movements for health promotion and preventive care.

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Annex A: List of Publications out of the Dissertation

S.N.	Title of Paper	Journal Details (Country, ISSN, DOI)
1	Impact of Yoga Awareness on Transmission Dynamics of Communicable Diseases: SIR Model Analysis	Communications on Applied Nonlinear Analysis (Q_4), USA, ISSN: 1074-133X
2	Impact of Yogachara on Transmission Dynamics of Infectious Diseases: SIQS Model Analysis at Saturated Incidence Rate	Journal of Jilin University (Engineering and Technology Edition), Vol. 43, Issue 09-2024 (Q_2), China, ISSN: 1671-5497, DOI: 10.5281/zenodo.13859350
3	Mathematical Modeling and Dynamic Systems in Epidemiology	AMC Journal (Dhangadhi), Vol. 5 (Peer-reviewed Category B by UGC Nepal), Research Management Cell, AMC, ISSN: 2661-6114, DOI: https://doi.org/10.3126/amcjd.v5i1.69086
4	Mathematical Modeling of Communicable Diseases Transmission Dynamics: Yoga Efficacy as Prevention Strategy	Applied Science and Technology Annals. Vol.2, No.1 (2025); ISSN: 2717-5014 (Print).
5	Compartmental Epidemiological Models and Their Associated Incidence Rate	Journal of NAST College (JONC), Volume 1, Number 1-2, 2025, ISSN: 3102-0283
6	Mathematical modeling of the impacts of mass education and Yogachara on corruption dynamics	Kathmandu Universily Journal of Science, Engineering and Technology (KUSET), Volume l9 Number 3(2025); Manuscript Submitted
7	Dynamics of an SIQS Epidemic Model with Saturated Incidence and Yogachara Influence	Discover Applied Sciences Springer Nature ;in communication

Table 1: List of articles published or submitted in journals

Annex B: Oral Presentations in National / International Conferences

The following oral presentations were delivered by the author at national and international conferences:

1. April 9–11, 2021: **Modelling of Epidemics Using Differential Equations**, International Conference on Analysis and Its Application-2021 (ICAA-Nepal-2021), organized by Nepal Mathematical Society, Nepal.

2. June 11–13, 2022: **Modeling of Transmission Dynamics of Communicable Diseases with Yoga**, National Conference on Mathematics and Its Applications, organized by Nepal Mathematical Society, Illam, Nepal.

3. May 25–28, 2023: **The Impact of Yoga Awareness on the Dynamics of a Time-Delayed Communicable Disease: A SIR Model Analysis**, Third International Conference on Applications of Mathematics to Nonlinear Sciences (AMNS-2023), Pokhara, Nepal.

4. September 28–29, 2024: **Impact of Yogachara on Communicable Disease Dynamics: SIQS Model Analysis at Saturated Incidence Rate**, International Conference on Theory and Application of Mathematics, organized by Tripura Mathematical Society, Agartala, India.

5. December 13–15, 2024: **Mathematical Modeling, Analysis, and Yogic Prevention of Infectious Diseases Dynamics**, Second Inter-

national Conference on Mathematics and Its Applications (ICMA-2024), Kathmandu, Nepal.

6. January 18–19, 2025: **Mathematical Modeling of Infectious Disease Transmission Dynamics at Saturated Incidence Rate**, JMC First International Conference 2025 on Quality Assurance in Higher Education: Challenges and Prospects, organized by JMC, Kathmandu, Nepal.

7. May 8–10, 2025: **Mathematical Modeling: Effect of Mass Education and Yogachara on Corruption Dynamics**, International Conference on Non-linear Analysis and Optimization (ICAN-OPT NEPAL 2025), organized by Kathmandu University in collaboration with NSU, NMS, and TU, Nepal.

Annex C: Data collected from Yoga Centers in Sudurpashchhim Province

The data were collected from various Yoga centers in Sudurpashchhim Province between March 2021 and August 2022. At each center, the main Yoga teacher was responsible for completing the questionnaire, which provided information on the number of trained Yoga teachers, Yoga Sadhaka (participants), and changes in participation and their health condition over the period.

Center Code	Yoga Sadhaka Individuals	Infected Yoga Sadhaka Individuals
1SK	20	2
2NK	30	3
GK	50	4
TK	60	5
OGK	80	9
M1K	70	6
M2K	60	5
M3K	20	1
D1	30	3
B1	35	3
B2	35	3
Da1	15	1
Do1	20	1
A1	35	2
Ba1	30	2
K1bazar	100	12
Online	1005	20
R1Oso	250	8
R2	12	1
R3	25	3

Table 2: Annex C

Annex D: Data collected from Yoga Centers: Yoga Sadhaka (Trained Yoga Teachers)

The following table presents the number of trained Yoga teachers in 2020/2021, the number of Yoga Sadhaka (participants) in 2022/2023, the increase in Yoga Sadhaka over two years, and the corresponding percentage increase across 16 Yoga centers:

Center Code	**Trained Yoga Teachers (2020/2021)**	**Yoga Sadhaka (2022/2023)**	**Increase in Yoga Sadhaka**	**Percentage Increase**
1SK	18	30	12	66
2NK	20	35	15	75
3GK	40	64	24	60
4TK	60	95	35	58
5GK	80	129	49	61.13
6M1K	70	116	46	65.71
7M2K	50	85	35	70
8M3K	20	35	15	75
9D1	30	53	23	76.66
10B1	35	63	28	80
11B2	35	63	28	80
12Da1	15	28	13	86.66
13Do1	20	38	18	90
14A1	35	62	27	77.14
15Ba1	30	52	22	73
16KA1	35	55	25	71.43
Total	593	1003	410	69.13

Table 3: Annex D

Appendix E: Questionaire

The following questionnaire was used to collect data from Yoga centers:

नेपाल संस्कृत विश्वविद्यालय, बेलझुण्डी, दाङ
अनुसन्धान केन्द्र
सुदूरपश्चिम प्रदेशका विभिन्न योग केन्द्रहरूमा तथ्याङ्क सङ्कलनका लागि प्रयोग भएको प्रश्नावली

क) प्रारम्भिक खण्ड

अनुसन्धान उद्देश्य:
सरुवा रोग नियन्त्रणमा योग शिक्षा (प्राणायाम) तथा योगाचार/योग चेतना को प्रभावकारिता अध्ययन गर्नका लागि यो प्रश्नावली प्रयोग भएको हो।
विगत एक वर्षको अवधिमा (२०७७ फाल्गुनदेखि २०७९ जेठसम्म) तपाईंको योग केन्द्रमा कुनै सहभागीले सरुवा रोग (जस्तै: रुघाखोकी, कोभिड, हैजा आदि) भोगेका भए, तलका प्रश्नहरूको आधारमा उत्तर दिनुहोस्।

अन्तर्वार्ता मिति: _____
केन्द्रको नाम: _____
योग प्रशिक्षकको नाम: _____ योगमा संलग्न अवधि: _____
योग साधक/साधिकाको नाम: _____फर्म नम्बर: _____
अन्तर्वार्ता मिति: _____
केन्द्रको नाम: _____
योग प्रशिक्षकको नाम: _____ योगमा संलग्न अवधि: _____
योग साधक/साधिकाको नाम: _____

क्र.स.	प्रश्न	विकल्प वा उत्तर लेख्ने स्थान
1	तपाईंको योग केन्द्रमा के नियमित योगका कक्षाहरू सञ्चालन हुन्छन् ?	☐ हुन्छन् ☐ हुँदैनन्
2	यदि हुन्छन् भने कुन समयमा कक्षाहरू सञ्चालन हुन्छन् ?	_____
3	विगत एक वर्षदेखि कति जना योगमा सहभागी हुनुहुन्छ ?	_____
4	तपाईंको केन्द्रमा नियमित आउने योग साधकहरू मध्ये कुनैलाई कुनै पनि प्रकारको सरुवा रोग लागेको कुरा थाहा छ ?	☐ छ ☐ छैन
5	यदि छ भने विगत एक वर्षमा कति जना बिरामी हुनुभयो ?	_____
6	ती बिरामीहरूलाई मुख्य रूपमा कुन स्वास्थ्य समस्या भएको थियो ?	☐ रुघाखोकी ☐ जरो ☐ कोभिडका लक्षण ☐ अन्य: _____
7	ती लक्षणहरू कति दिनसम्म देखिएका थिए ?	☐ एक हप्ता भन्दा कम ☐ एक हप्तादेखि दुई हप्तासम्म ☐ दुई हप्ताभन्दा बढी

Figure 1: Annex E: Questionaire used for data collection

Appendix E cont . . .

क्र.स.	प्रश्न	विकल्प वा उत्तर लेख्ने स्थान
8	पहिले (लगभग एक वर्षअघि) तपाईंको योग केन्द्रमा योग साधक/साधिकाको सङ्ख्या कति थियो ?	_____
9	अहिलेको सङ्ख्या उल्लेख गर्नुहोस् ।	_____
10	कोभिड अघि र पछि योग साधकहरूको सङ्ख्यामा फरक पाउनुभएको छ ?	☐ छ ☐ छैन
11	यदि छ भने फरक कस्तो देखिन्थ्यो ?	☐ बढेको सङ्ख्या: _____ ☐ घटेको सङ्ख्या: _____
12	तपाईंको अनुभवमा, घर वा केन्द्रमा क्वारेन्टाइनमा रहेका व्यक्तिहरूमा नियमित योग अभ्यास (योगाचार) ले शारीरिक वा मानसिक स्वास्थ्यमा कस्तो प्रभाव पार्‌यो ?	☐ अत्यधिक सकारात्मक प्रभाव ☐ मध्यम सकारात्मक प्रभाव ☐ कुनै विशेष प्रभाव छैन ☐ नकारात्मक प्रभाव (व्याख्या गर्नुहोस्): _____
13	क्वारेन्टाइन अवधिमा योग अभ्यास गर्दा छिटो निको हुन कुन तत्व बढी सम्बन्धित देखिन्थ्यो ?	_____
14	तपाईंको अवलोकन अनुसार, योग अभ्यास गर्ने व्यक्तिहरूमा रोग निको हुन र आइसोलेशन अवधिमा स्वास्थ्य सुधारमा योगले कसरी सहयोग गर्‌यो ?	_____
15	योगाचारका तत्वहरू ,आहार (Aahar), विहार (Vihar), आचार (Aachar) र विचार (Vichar) ले सहभागीहरूको रोगावस्थामा कस्तो प्रभाव पार्छ ?	☐ अत्यधिक सकारात्मक प्रभाव ☐ मध्यम सकारात्मक प्रभाव ☐ कुनै विशेष प्रभाव छैन ☐ नकारात्मक प्रभाव (व्याख्या गर्नुहोस्): _____

तपाईंको समय र सहयोगका लागि धेरै धेरै धन्यवाद।