SALISU MUHAMMAD MODELLING THE DYNAMICS OF TRACHOMA FOR CONTROL AND ELIMINATION MUHAMMAD PhD THESIS 2021

NEAR EAST UNIVERSITY

INSTITUTE OF GRADUATE STUDIES

DEPARTMENT OF MATHEMATICS

MODELLING THE DYNAMICS OF TRACHOMA FOR CONTROL AND ELIMINATION

PhD. THESIS

Salisu Muhammad, MUHAMMAD

Nicosia

October, 2021

NEAR EAST UNIVERSITY

INSTITUTE OF GRADUATE STUDIES

DEPARTMENT OF MATHEMATICS

MODELLING THE DYNAMICS OF TRACHOMA FOR CONTROL AND ELIMINATION

PhD. THESIS

Salisu Muhammad MUHAMMAD

Supervisor

Prof. Dr. Evren HINCAL

Nicosia

October, 2021

Approval

We certify that we have read the thesis submitted by Salisu Muhammad Muhammad titled "**Modelling the Dynamics of Trachoma for Control and Elimination**" and that in our combined opinion it is fully adequate, in scope and in quality, as a thesis for the degree of Doctor of Philosophy.

Examining Committee	Name-Surname	Signature
Head of the Committee:	Prof.Dr. Allaberen Ashyralyev	.....
Committee Member:	Assoc.Prof.Dr. Okan Gerçek	.....
Committee Member:	Assoc.Prof.Dr. Bilgen Kaymakamzade	.....
Committee Member:	Assoc.Prof.Dr. Tolgay Karanfiller	.....
Supervisor:	Prof.Dr. Evren Hınçal	.....

Approved by the Head of the Department

20/10/2021

Prof. Dr. Evren Hınçal
Head of Department

Approved by the Institute of Graduate Studies

...../...../2021

Prof. Dr. Kemal Hüsnü Can Başer
Head of the Institute

Declaration

I hereby declare that all information, documents, analysis and results in this thesis have been collected and presented according to the academic rules and ethical guidelines of Institute of Graduate Studies, Near East University. I also declare that as required by these rules and conduct, I have fully cited and referenced information and data that are not original to this study.

Salisu Muhammad Muhammad

15./11/2021.

Acknowledgments

egin with acknowledging the support and encouragement I received from my indefatigable supervisor, Prof. Dr. Evren Hincal, I thank you very much for injecting me with bunch of useful advice that made this work a reality today. I must acknowledge the support and assistance I received from Prof. Dr. Allaberan Ashyralyev, Prof. Dr. Adeguzel Dosiyev, Yard Doc Dr. Bilgen Kaymakamzade (My academic Advisor and vice chair of the department), Nezihal Gokbulut and all other staff in the Department of Mathematics NEU. The effort of my research collaborators is also worth mentioning and acknowledging.

I would like to extend my appreciation and prayers to my parents Alhaji Salisu Mu'azu Bungudu and Hauwa'u Usman, for their sacrifice, love, caring and moral support. On a personal note, I wish to thank my wives: Zainab Abdullahi Bunu and Khadija Sirajo Gulubba, my lovely children, Fadeela, Saleem and Ameena for their patience, support, prayers and well wishes. Special thanks and appreciations go to His Royal Highness the Emir of Bungudu Alh. Hassan Attahiru for his inspiring advice and useful suggestions.

I would like to thank in general all my colleagues in Federal University Gusau, Zamfara State especially Dr. Aminu Idris of the department of Political Science, Mansur Muhammad Bello the Head of Public Administration Department, and meny more. I owe special gratitude to Malam Lawal Muhammad Jabaka of the Department of Computer Science (FUGUS) for his inspiring pieces of advice especially during discussing issues related to this research work.

To my Family members, especially my immediate sister Shema'u Salisu, who has been with me in her prayers all the time, I say thank you for your encouragements, prayers and useful suggestions. In carrying out this work, I received much help and encouragement from many people whose efforts are worth acknowledging. I therefore wish to express my profound gratitude to those who in oway or another contributed towards the success of this work..

Salisu Muhammad Muhammad

Abstract

Modelling the Dynamics of Trachoma for Control and Elimination

Salisu Muhammad, Muhammad

PhD, Department of Mathematics

October, 2021, 100pages

Trachoma is an eye infectious disease caused by Chlamydia Trachomatis bacterium, which may lead to irreversible blindness. The disease is spread directly or indirectly by contacting a contaminated material, it can also be transmitted through the disease vector known as "musca sorbens" or "Bazaar fly". To curtail the spread of the disease in a population, a meaningful information on the spread and possible control of the disease is required. Mathematical modelling provides efficient tools that can be used to understand and analyse the dynamics of the disease and its control. Several compartmental epidemic models have been proposed in the literature to study the dynamics of trachoma; including SI, SIR and SEIR. However, majority of the existing trachoma models consider only person to person transmission. Thus, the information provided by such models is insufficient since they did not capture the disease vector transmission. The current study proposed a novel SEIR-SEI model that consider both person-person and vector transmission dynamics. The threshold quantity, basic reproduction number is obtained using the next generation matrix, and it was proved that the disease-free equilibrium is asymptotically stable when $R_0 < 1$, and the endemic equilibrium is globally asymptotically stable when $R_0 > 1$. Some simulation results with the aid of mesh plots for the reproductive number as a function of two different biological parameters was obtained. Furthermore, a comprehensive sensitivity analysis is conducted to identify the influence of the individual parameters on the R_0. Numerical results show that the vector contact rate σ_f has the highest sensitivity with respect to R_0, and the value of R_0 increases with increase in σ_f. hence, the disease can be control by decreasing the vector contact rate. Similarly, improving the rate of environmental hygiene and facial cleanliness will decrease the size of R_0 which results in the declination of the disease transmission. Moreover, a detailed parameter estimation of the model parameters and model fitting was presented with the use of field data cases from Northern Nigeria using least-square fitting method. The study provides alternative tools that can be used for planning trachoma control program to achieve global eradication of trachoma as a public heath challenge as targeted by WHO in 2030.

***Key Words**:* trachoma, reproduction number, chlamydia trachomatis, stability, sensitivity analysis

Abstract

Modelling the Dynamics of Trachoma for Control and Elimination

Salisu, Muhammad Muhammad

PhD, Department of Mathematics

Prof. Dr. Evren Hincal

October, 2021, 100pages

Trachoma is an eye infectious disease caused by Chlamydia Trachomatis bacterium, which may lead to irreversible blindness. The disease is spread directly or indirectly by contacting a contaminated material, it can also be transmitted through the disease vector known as “musca sorbens” or “Bazaar fly”. To curtail the spread of the disease in a population, a meaningful information on the spread and possible control of the disease is required. Mathematical modelling provides efficient tools that can be used to understand and analyse the dynamics of the disease and its control. Several compartmental epidemic models have been proposed in the literature to study the dynamics of trachoma; including SI, SIR and SEIR. However, majority of the existing trachoma models consider only person to person transmission. Thus, the information provided by such models is insufficient since they did not capture the disease vector transmission. The current study proposed a novel SEIR-SEI model that consider both person-person and vector transmission dynamics. The threshold quantity, basic reproduction number is obtained using the next generation matrix, and it was proved that the disease-free equilibrium is asymptotically stable when $R_0 < 1$, and the endemic equilibrium is globally asymptotically stable when $R_0 > 1$. Some simulation results with the aid of mesh plots for the reproductive number as a function of two different biological parameters was obtained. Furthermore, a comprehensive sensitivity analysis is conducted to identify the influence of the individual parameters on the R_0. Numerical results show that the vector contact rate σ_f has the highest sensitivity with respect to R_0, and the value of R_0 increases with increase in $\sigma_{f..}$ Hence, the disease can be control by decreasing the vector contact rate. Similarly, improving the rate of environmental hygiene and facial cleanliness will decrease the size of R_0 which results in the declination of the disease transmission. Moreover, a detailed parameter estimation of the model parameters and model fitting was presented with the use of field data cases from Northern Nigeria using least-square fitting method. The study provides alternative tools that can be used for planning trachoma control program to achieve global eradication of trachoma as a public heath challenge as targeted by WHO in 2030.

Keywords: trachoma, reproduction number, chlamydia trachomatis, stability, sensitivity analysis

ÖZET

Trachsummaroma, geri dönülemez körlüğe neden olabilen Chlamydia Trachomatis bakterisinin neden olduğu bulaşıcı bir hastalıktır. Hastalık doğrudan veya dolaylı olarak kontamine bir materyalle temas ederek veya "musca sorbens" ya da "Bazaar fly" olarak bilinen hastalık vektörü yoluyla da bulaşabilir. Bir popülasyonda hastalığın yayılmasını azaltmak için, hastalığın yayılması ve kontrolü hakkında anlamlı bilgi gereklidir. Matematiksel modelleme, hastalık dinamiğini ve kontrolünü anlamak ve analiz etmek için kullanılabilecek verimli bir yoldur. Literatürde Trahoma hastalığının dinamiklerinin incelendiği SI, SIR ve SEIR modelleri yer almaktadır. Bununla birlikte, mevcut Trahoma modellerinin çoğu, yalnızca kişiden kişiye bulaşmayı dikkate alır. Bu nedenle, bu tür modellerden elde edilen bilgiler, hastalık vektörü geçişini kapsamadığı için yetersizdir. Bu çalışmada, hem bireyden bireye hem de vektör aktarım dinamiklerini dikkate alan yeni bir SEIR-SEI modeli oluşturuldu. Temel üreme sayısının eşik değeri Next Generation Matris yöntemiyle elde edildi; $R_0 < 1$ durumunda hastalıksız denge noktasının, $R_0 > 1$ durumunda ise endemik denge noktasının küresel asimptotik olarak kararlı olduğu kanıtlandı. İki farklı biyolojik parametrenin bir fonksiyonu olarak üreme sayısı için ağ grafiği yardımıyla bazı simülasyon sonuçları elde edildi. Ayrıca, parametrelerin R_0 üzerindeki etkisini belirlemek için duyarlılık analizi yapıldı. Sayısal sonuçlar, R_0 üzerinde en yüksek duyarlılığa sahip parametrenin σ_f (vektör temas hızı) olduğu, σ_f'in artması sonucu R_0 değerinin de arttığı; vektör temas hızı azaltılarak hastalığın kontrol edilebileceğini gösterdi. Benzer şekilde, çevre hijyeni ve yüz temizliğinin iyileştirilmesi, R_0 değerini azaltacak ve bu da hastalığın bulaşma hızını azaltacaktır. Ayrıca, model parametrelerinin ayrıntılı bir tahmini ve model uyumu için en küçük kareler metoduyla Kuzey Nijerya'dan alınan veriler kullanıldı. Dünya Sağlık Örgütü, 2030 yılına kadar Trahomanın küresel eradikasyonunu sağlamayı hedeflemektedir. Tez'de yapılan çalışma, bu kontrol programının planlanmasında kullanılabilecek metodlar sunmaktadır.

Anahtar Kelimeler: Trahoma Modelleme, Üreme sayısı, Chlamydia Trachomatis, Denge, Duyalılık Analizi, Tahmin, Model Uyumu.

TABLE OF CONTENTS

Approval…… 1

Declaration…… 2

Acknowledgements…….. 3

Abstract……. 4

Summary……... 5

Ozet ……..... 6

Table of Contents …… 7

List of Tables…… 11

List of Figures…….. 12

List of Abbreviations…… 15

CHAPTER 1

1.1 Introduction……..... 17

1.2 Background of Study…….. 17

1.2.1 Historical Perspective ……... 17

1.2.2 Clinical Features of Trachoma ……. 18

1.2.3 Chlamydia Trachomatis Infection Transmission.……. 20

1.2.4 Prevalence of Trachoma and Geographical Distribution…… 22

1.3 Controlling Trachoma: The SAFE Strategy …….. 23

1.3.1 Surgery for Trichiasis …… 23

1.3.2 Antibiotic …….. 24

1.3.3 Facial Cleanliness …….. 25

1.3.4 Environmental Improvement ……. 25

1.4 The SAFE Strategy: Putting the pieces together …… 26

1.5 Research Problem and Statement…… 27

1.6 Research Aim and Objectives …… 28

1.7 Scope of the Study ……. 30

1.8 Thesis Organization……. 31

CHAPTER 2

2.1 Literature Review……..... 32

2.2 Mathematical Modelling of Epidemiology……. 32

2.2.1 Basic mathematical epidemic models ……... 33

2.2.2 SIR Epidemic Model …… 36

2.2.3 SEIR Epidemic Model ……. 37

2.3 Basic Reproduction Number ……. 38

2.4 Model Fitting …….. 39

2.5 Sensitivity Analysis …… 42

2.6 Developmental Cycle of Chlamydia Trachomatis ……. 43

2.7 Epidemiology of Trachoma …… 45

CHAPTER 3

MATHEMATICAL MODELLING AND ANALYSIS FOR THE DYNAMICS OF BLINDING TRACHOMA

3.1 Methodology……..... 50

3.2 Formulation and Description of Trachoma Model …… 53

3.3 Basic Property of the Model …….. 57

3.4 Existence of Equilibria and Stability Analysis…… 58

3.4.1 Disease Free Equilibrium Point ……. 58

3.4.2 Local Stability Analysis of DFE …… 58

3.4.3 Endemic Equilibrium Point …… 60

3.4.4 Global Stability of Endemic Equilibrium Point ……. 62

3.5 Parameter Estimation ……. 67

3.6 Normalize Sensitivity Analysis …….. 70

CHAPTER 4

NUMERICAL SIMULATIONS AND DISCUSSION OF RESULTS

4.1 Numerical Simulations…… 77

4.2 Discussion of Results ……. 87

CHAPTER 5

CONCLUSION AND RECOMMENDATIONS

6.1 Conclusion……... 89

6.2 Recommendations……... 90

REFERENCES……. 91

APPENDIX : Ethical Similarity Report

LEST OF TABLES

Table 3.1: State Variables and parameters of the model 56

Table 3.2: Baseline values of the parameters used in the trachoma model 68

Table 3.3: Local Sensitivity indices and parameter values with respect to R0 73

LIST OF FIGURES

Figure 1.1: The life cycle of Trachoma …….. 21

Figure 1.2: WHO adopted SAFE Strategy ……..... 24

Figure 2.1: Developmental Stages of Epidemic model……... 35

Figure 2.2: Typical SIR epidemic model…… 37

Figure 2.3: Typical SIER epidemic model…….. 38

Figure 2.4: Flowchart for Model fitting ……. 41

Figure 2.5: Life cycle of Chlamydia Trachomatis ……. 44

Figure 2.6: Infectious Stages of Trachoma …… 46

Figure 3.1: Schematic diagram of the Model (3.1) …… 54

Figure 3.2: The Time series plot of the Model …….. 66

Figure 3.3: Model fitting for real Trachoma cases ……..……..... 69

Figure 3.4: Charts for the sensitivity indices…….. 74

Figure 4.1: Plot for R_0^T against parameter ψ_1 with ψ_1 from 0.100 to 0.700 while all other parameters are fixed……...……... 78

Figure 4.2: Plot for R_0^T against parameter ψ_2 with ψ_2 from 0.100 to 0.700 while all other parameters are fixed …… 78

Figure 4.3: Plot for R_0^T against parameter τ with τ from 0.100 to 0.700 while all other parameters are fixed …… 79

Figure 4.4: Plot for R_0^T against parameter σ_f with σ_f from 0.100 to 0.700 while all other parameters are fixed …… 79

Figure 4.5: Plot for R_0^T against parameter β_h with β_h from 0.100 to 0.700 while all other parameters are fixed …… 80

Figure 4.6: Plot for R_0^T against parameter β_f with β_f from 0.100 to 0.700 while all other parameters are fixed …… 80

Figure 4.7: Effects of SAFE control Strategies on human population (The behavior of Trachoma infection when $R_0 > 1$)……..…….. 81

Figure 4.8: Effects of SAFE control Strategies on human population (The behavior of Trachoma infection when $R_0 < 1$)……..……. 81

Figure 4.9: Effects of SAFE control Strategies on Flies population (The behavior of Trachoma infection when $R_0 > 1$)……..…….. 82

Figure 4.10: Effects of SAFE control Strategies on Flies population (The behavior of Trachoma infection when $R_0 < 1$)……..…….. 82

Figure 4.11: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of human transmission β_h …….. 83

Figure 4.12: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of flies transmission β_f ……..……. 83

Figure 4.13: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of flies progression rate from exposed class to infectious class τ ……..……. 84

Figure 4.14: Profile of Basic reproduction number with variation in human progression rates $\delta_1$and δ_2…….……..…… 84

Figure 4.15: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of Natural Death of human μ_h…… 85

Figure 4.16: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of Natural Death of flies μ_f……. 85

Figure 4.17: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of human progression rate from exposed class to early infectious class (I_{hs}) δ_1 ……. 86

Figure 4.18: Profile of Basic reproduction number with variation in contact rate of disease vector σ_f and that of human progression rate from exposed class to severe infectious class (I_{hc}) δ_2 …..…….. 86

LIST OF ABBREVIATIONS

WHO:	World Health Organization
GBD:	Global Burden of Disease
MTB:	Mycobacterium Tuberculosis
SEI:	Susceptible, Exposed, and Infectious
SEIR:	Susceptible, Exposed, Infectious, and Recovered
DFE:	Disease Free Equilibrium
EE:	Endemic Equilibrium
R_0:	Basic Reproduction Number
NGM:	Next Generation Matrix
ODE:	Ordinary Differential Equation
SSE:	Sum Squared Error
TF/TI:	Trachomatous Folliclar/Intense
TT:	Trachomatous Trachiasis
TS:	Trachomatous Scares
CO:	Corneal Opacity

HIV: Human Immunodeficiency Virus

ASMC: Adaptive Sliding Mode Control

EB: Elementary Body

RBs: Reticulate Bodies (RBs)

MDA: Mass distribution of Antibiotic

GET2020: Global Elimination of Trachoma 2020

SAFE: Surgery, Antibiotic, Facial Cleanliness, Environmental improvement

NTDs: Neglected Tropical Diseases

CHAPTER 1
INTRODUCTION

Trachoma is the global most common infectious cause of blindness. It is caused by Chlamydia trachomatis infection and is marked by inflammatory changes in the conjunctiva of children, followed by scarring, corneal opacity, and blindness in adults. In 2002, the World Health Organization (WHO) reported that 1.3 million people were blind due to trachoma (Resnikoff et al. 2004), with another 1.8 million possibly suffering from impaired vision (Frick et al. 2003a). In locations where trachoma is endemic, additional 1.9 million cases of blindness due to ”corneal opacities” were likely due to trachoma (Resnikoff et al. 2004). There are around 40 million people with active infection, and 8.2 million persons with trichiasis (Mariotti et al. 2009). Trachoma is an ancient disease that has long been a major public health issue in many parts of the world, including Europe and North America. Trachoma is now primarily seen in low-income nations, mainly in Sub-Saharan Africa, in poor, rural communities. The Alliance for the Global Elimination of Blinding Trachoma by 2020 was founded by the WHO in 1998. (GET2020). The SAFE Strategy (surgery for trichiasis, antibiotics for C. trachomatis infection, facial cleanliness, and environmental enhancement) is used to combat trachoma. There have been encouraging improvements in the prevalence of trachoma where control methods have been applied.

1.1 BACKGROUND OF THE STUDY

1.1.1 Historical Perspective

In the 27th century BC, trachoma was first mentioned in China (Al-Rifai 1988). The Ebers papyrus from Egypt, dating from the 15th century BC, and epilation forceps discovered in tombs dating from the 19th century BC both detailed trachoma symptoms (Maccallan 1931, Hirschberg 1982). Trachoma became a serious public health issue in Europe at the turn of the nineteenth century, when troops returning from Napoleon’s conquests in Egypt were thought to have brought the disease back with them. Many of the main ophthalmic facilities created in the nineteenth century, notably Moorfields Eye Hospital, were founded to cure trachoma. Immigrants to the U.s were consistently

examined for trachoma by the end of the nineteenth century, and those who showed signs of the disease were sent home. Trachoma has now vanished from developed countries (save for Aboriginal populations in outback Australia (Tellis et al. 2007)), owing to overall improvements in living and standards of hygiene.

1.2 CLINICAL FEATURES OF TRACHOMA

Trachoma is a keratoconjunctivitis caused by recurring infection with C. trachomatis serovars and Children are the ones who become infected the most. Some patients have scarring issues and blindness later in life as a result of frequent reinfections. The clinical manifestations of trachoma are classified into those related with 'active' illness, which is more commonly seen in children, and those associated with cicatricial or scarring sequelae, which are more commonly observed in late childhood and adults. Recurrent episodes of chronic follicular conjunctivitis are the hallmarks of active disease. Follicles are tiny, yellowwhite elevations on the conjunctiva of the everted upperlid that are sub-epithelial clusters of lymphoid cells. Papillary hypertrophy (small vessel enlargement with surrounding oedema) can also occur, and if severe enough, can cover the deep tarsal vessels. Active illness can also cause vascular infiltration of the top cornea (pannus), but this seldom impairs vision. Even when significant indicators of inflammation are present, many people are asymptomatic or have relatively mild symptoms. If redness, pain, tears, photophobia, and minimal muco-purulent discharge are present, they are comparable to those associated with any chronic conjunctivitis. Unlike follicles and papillae, conjunctival follicles at the top margin of the cornea create shallow depressions known as 'Herbert's pits,' which are a pathognomonic marker of trachoma. Scarring complications of trachoma can arise from repeated episodes of infection and inflammation. The subtarsal conjunctiva shows conjunctival scarring at first, which can range from a few linear/ stellate scars to extensive, distorting bands of fibrosis. Entropion (in-turning of the eyelids) and trichiasis (eyelashes touching the eyeball) are common side effects of scar tissue contraction. Corneal opacification eventually progresses to the blinding end-stage of the disease. Multiple insults to the cornea are most likely to blame: mechanical stress from lashes, subsequent bacterial or fungal infection, and a dry ocular surface.

Various trachoma grading systems have been proposed over the years. The 1987 WHO simplified grading system is the one being utilized by trachoma control programs (Thylefors et al. 1987). Preschool children have the highest prevalence of active disease, which falls to low levels in adulthood (Dawson et al. 1976; West et al. 1991b; Dolin et al. 1998). In other investigations, up to half of the community bacterial load was identified in children under the age of one year, which corresponds to the distribution of C. trachomatis infection (Solomon et al. 2003; Melese et al. 2004b). Adult bacterial loads are typically lower than those of children, and infection and disease duration decreases with age, possibly due to a developed immune response (Bailey et al. 1999; Grassly et al. 2008). In contrast, the scarring characteristics of trachoma increase with age, demonstrating the cumulative nature of the damage. Trichiasis was reported in 2–3percent of children under the age of 15 years in southern Sudan, while the frequency of active disease was 70–80percent. (Ngondi et al. 2006a; King et al. 2008). The evolution of the scarring process has been studied in cohort studies in trachoma-endemic groups in The Gambia and Tanzania:

- Scarring on the conjunctiva worsened in nearly half of scarred participants after 5 years (Tanzania) (Wolle et al. 2009).

- In Tanzania and The Gambia, 10percent of patients progressed from conjunctival scarring to trichiasis after 7 years, and 6percent after 12 years (Munoz et al. 1999; Bowman et al. 2001).

- Minor trichiasis (five or more lashes touching the eye) advanced to major trichiasis (five or more lashes touching the eye) in 33percent of patients after one year and 37percent after four years; unilateral trichiasis proceeded to bilateral trichiasis in 46percent of patients after one year (The Gambia) (Bowman et al. 2002b; Burton et al. 2006).

- Trichiasis is linked to corneal scarring: after four years, 8percent of patients with trichiasis developed incident corneal scarring, and 34percent had worsened established corneal scarring (The Gambia) (Bowman et al. 2002b; Burton et al. 2006).

The first Tanzanian study had a standardized, prospective design, but the others did not. There is a lot of variety in the reported progression rates, which could be due to both differences in progression rates in different populations and differences in methodology. Although direct evidence for this is sparse, the burden of C. trachomatis infection in a population over time is undoubtedly a crucial predictor of the rate of disease progression. Those with recurrent or persistent severe inflammatory trachoma are more likely to suffer scarring issues, according to several studies (Dawson et al. 1990; Munoz et al. 1999; West et al. 2001; Burton et al. 2006

1.3 CHLAMYDIA TRACHOMATIS INFECTION TRANSMISSION

Chlamydia trachomatis is shared between people through a variety of ways, including:

- Direct transmission from eye to eye during close contact, such as during play or sleep.
- Infected ocular or nasal secretions spread to fingers.
- Fomites, such as infected facecloths, spread the disease in an indirect manner. Transmission by flies that seek out people's eyes.

Most environments likely use a combination of these and other transmission modes, while their relative importance may vary. Eye-seeking flies, for example, are thought to have a role in the spread of infection in specific situations. In Ethiopia, Chlamydia trachomatis has been found in around 20percent of Musca sorbens caught on children's faces (Jones 1975; Miller et al. 2004a; Lee et al. 2007), and intervention trials to reduce fly density have been linked to a reduction in active trachoma in The Gambia (Jones 1975; Miller et al. 2004a; Lee et al. 2007). (Emerson et al. 1999, 2004). In other areas, however, the number of eye-seeking flies is negligible and does not appear to play a role in transmission (Taylor et al. 1985). C. trachomatis genital strains do not induce endemic trachoma, while they can infrequently cause a self-limiting conjunctivitis (Brunham et al. 1990).

Trachoma is a focused disease that has been observed to cluster at the community, household, and bedroom levels, confirming the infectious character of the disease and implying that infection transmission requires continuous personal contact (Dawson et al. 1976; Katz et al. 1988; Bailey et al. 1989; West et al. 1991b; Burton et

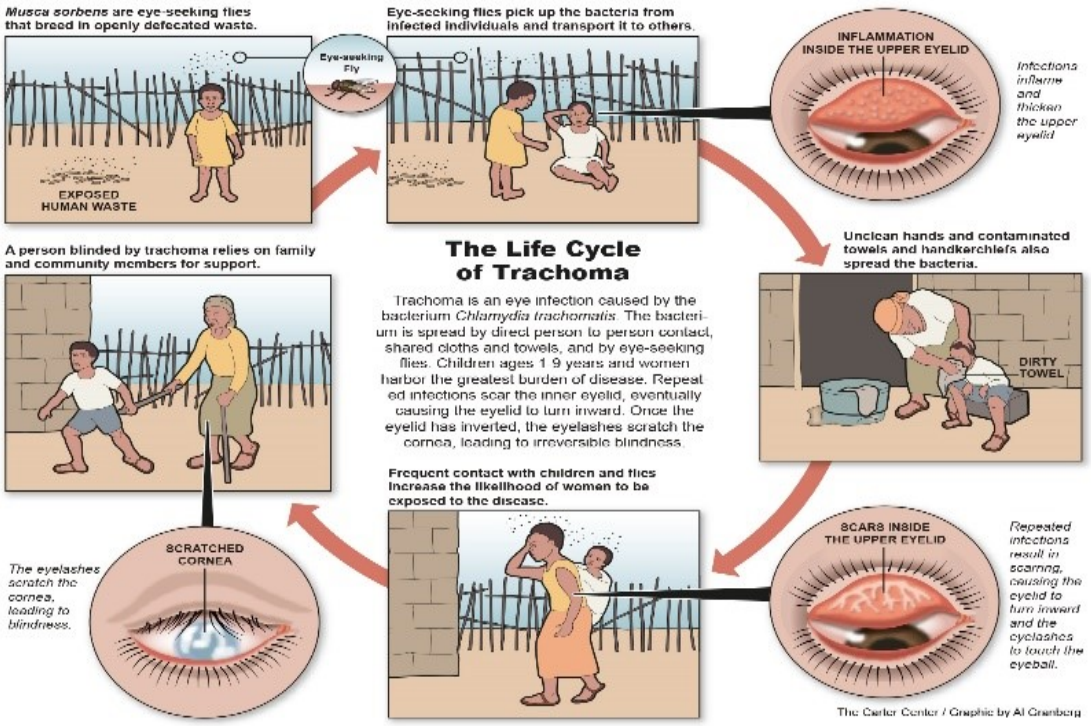


Figure 1.1. the life cycle of Trachoma. (Meraf et al. 2009)

al. 2003). This is especially essential for trachoma management programs since it raises the sample size required to estimate the prevalence within a region (Katz et al. 1988). The majority of transmission events occur within the home, and failing to treat all infected immediate relatives during mass antibiotic administration may result in a fast re-infection of that family, accompanied by a more slow spread across the community (Blake et al. 2009). There hasn't been any evidence of a non-human reservoir of infection, with flies serving only as passive vectors. The significance of infection locations outside of the eye has been questioned.

Chlamydia trachomatis can be found in nasopharyngeal secretions, and a new study finds that infected nasal discharge in children at baseline was linked to a higher risk of active illness and conjunctivitis two months following systemic treatment (Malaty et al. 1981; West et al. 1993; Gower et al. 2006). Nasal swabs, on the other hand, were only taken from children who had visible discharge and were of the discharge rather than nasal epithelium. Positive results could simply reflect a serious eye infection that didn't respond to a single antibiotic dosage, with contaminated fluids flowing through the nasolacrimal ducts. An earlier trial that used nose swabs on all children found that

new ocular infection following treatment had nothing to do with whether the nasal specimen was positive or negative at the start (West et al. 1993). In addition, genotyping of conjunctival and nasal samples from individuals with concurrent infection showed different genotypes to be present, suggesting that auto-infection was not an important factor (Andreasen et al. 2008).

1.4 PREVALENCE AND GEOGRAPHICAL DISTRIBUTION OF TRACHOMA

In many developing countries, particularly in rural regions, trachoma is a leading cause of blindness. Blinding trachoma is thought to be prevalent in more than 50 nations, with Africa having the highest frequency of active disease and trichiasis, primarily in the savannah regions of East and Central Africa and the Sahel of West Africa. It is also found in a number of Middle Eastern, Asian, Latin American, and Western Pacific countries (Polack et al. 2005). The prevalence of active illness, trichiasis, and blindness, according to WHO estimations, is much lower than prior estimates. Ethiopia, India, Nigeria, Sudan, and Guinea account for approximately half of the global burden of active trachoma. Due to the scarcity of current data from India and China, these figures are subject to high uncertainty (Mariotti et al. 2009). Trachoma is particularly common in southern Sudan, which was previously inaccessible due to the civil war: up to 80percent of children had active disease, and one-fifth of adults had trichiasis, according to recent studies (Ngondi et al. 2006a; King et al. 2008). Trachoma was shown to be responsible for 35percent of blindness, with 5percent of the population (including children) suffering from trachoma-related blindness (Ngondi et al. 2006b, 2007). The interpretation of global estimates of trachoma prevalence should be done with caution (Burton and Mabey 2009). These were mostly created using models based on the findings of a small number of surveys done in a few endemic countries.

The results from a single survey within a district is then extrapolated to obtain the district level prevalence, and national averages are calculated using available district prevalence data. The WHO's estimate of six million persons blinded by trachoma in the 1990s was most likely an overestimate, given the figures were based on questionnaires that reported numbers of people who may go blind if they didn't get

treatment (Thylefors et al. 1995). Recent estimations have relied on more accurate survey data. As with the departure of trachoma from industrialised countries a century ago, improved living standards in many countries are likely to account for at least some of this trend (Dolin et al. 1997; Hoechsmann et al. 2001). Although it is difficult to quantify, the creation of trachoma control programs is likely to have played a significant influence. Worryingly, the estimated number of patients with trichiasis has been relatively stable since 1991, with a little increase projected between 2003 and 2008. This implies that even after active illness and C. trachomatis infection have decreased significantly, progressive conjunctival scarring can develop.

The World Health Organization's most recent assessment puts the global impact of trachoma at 1.3 million disability-adjusted life years. This is the difference between a normal, healthy population and the disease's 'cost' in terms of premature mortality and disability (WHO 2008). Trachoma is expected to cost between 3 billion USD and 8 billion USD in lost productivity (Frick et al. 2003a,b). However, estimates of the worldwide burden of trachoma encounter a number of challenges, including a lack of reliable prevalence data and the decision to include different disease presentations (Burton and Mabey 2009).

1.5 CONTROLLING TRACHOMA: THE SAFE STRATEGY

Trachoma blindness is essentially irreversible, however it can be avoided. The WHO established the Alliance for the Global Elimination of Blinding Trachoma by 2020 (GET 2020) in 1997, and it recommends the SAFE trachoma control strategy, which includes surgery for trichiasis, antibiotics to treat C. trachomatis infection, personal hygiene, and environmental improvement through education and improved local economies.

1.5.1 Surgery for trichiasis

The goal of trichiasis surgery is to prevent corneal opacity and blindness from developing as a result of lashes abrading the cornea. In severe cases of trichiasis, surgery has been demonstrated to relieve comfort, reduce ocular discharge, and improve visual acuity (Reacher et al. 1992; Bowman et al. 2000a; Burton et al. 2005a). While there hasn't been any direct evidence that trichiasis surgery slows the

Figure 1.2. WHO adopted SAFE strategy. (Meraf et al. 2009)

advancement of corneal opacity (Bowman et al. 2001, 2002b), the general assumption is that it does. Regular surgical sessions at fixed sites once a week, with periodic outreach stations held in trachoma-endemic communities, according to the WHO, should be offered to anyone with trichiasis, regardless of the number of in-turned eyelashes, and should be offered to anyone with trichiasis, regardless of the number of in-turned eyelashes (WHO, 2006).

1.5.2 Antibiotics

The discovery that an oral dose dosage of azithromycin was as efficient as 6 weeks of regular tetracycline ointment in the management of acute infection (Bailey et al. 1993) was a huge breakthrough, and it paved the way for the worldwide eradication drive. It is advised that entire districts or towns be treated in bulk, as this is more effective in stopping re - infection than treating individual instances (Schachter et al. 1999). A district is a geographic location with a population of between 100 000 and 150 000 people.

The WHO advises either a single oral dose of azithromycin or a one percent

tetracycline eye ointment two times daily for 42 days. The two medicines are similarly effective in randomized controlled trials (Bailey et al. 1993; Tabbara et al. 1996; Dawson et al. 1997; Schachter et al. 1999), while azithromycin is more successful in operational use (Bailey et al. 1993; Tabbara et al. 1996; Dawson et al. 1997; Schachter et al. 1999). (Bowman et al. 2000b). Tetracycline is almost commonly available, however due to the time of dosage, difficulty and unpleasantness of application, and side effects such stinging and blurred vision, it has low compliance (West 1999; Kuper et al. 2003).

1.5.3 Facial cleanliness

By removing a potential source of infection, improving facial hygiene (the lack of ocular and nasal discharge) (Negrel and Mariotti 1999) seeks to prevent auto-transmission and transmission to others (Kuper et al. 2003). It is encouraged through health education and better water supply, however there is little evidence to support this management technique. As previously mentioned, gathering data on face cleaning is challenging since the validity of self-reporting is debatable, measures of a clean face are subjective, and some indications (discharge and flies) are more dependable than others (dust and food on the face) (Harding-Esch et al. 2008; Zack et al. 2008).

1.5.4 Environmental improvement

The SAFE strategy's 'E' component aims to prevent C. trachomatis transmission by fostering greater personal and environmental hygiene. Without any explicit intervention, trachoma was eradicated from Europe and North America in the nineteenth century, demonstrating the value of the SAFE strategy's environmental improvement components (Mabey et al. 2003). Trachoma transmission should be halted by improving water availability and quality, improving access to latrines, reducing fly density, reducing crowding, and giving health education (Kuper et al. 2003).

Until 2004, when Emerson et al. compared seven clusters that underwent spraying to seven clusters that did not, there was minimal evidence supporting the fly control component of the SAFE method. In the intervention clusters, insecticide spraying

resulted in an 88 percent reduction in fly-eye contacts and a significant 55.8 percent reduction in the prevalence of active illness (Emerson et al. 2004). Spraying is also time-consuming, expensive, and unsustainable (Rabiu et al. 2007). There is only one randomized controlled trial on latrine use. Emerson et al. compared seven clusters with latrines against seven clusters without (Emerson et al. 2004). The supply of latrines resulted in a 30 percent reduction in M. sorbens-eye contacts, as well as a 29.5 percent reduction in M. sorbens-eye infections. Despite a reported 98 percent use of latrines, the frequency of trachoma did not reach statistical significance. Latrines will only improve environmental cleanliness if a large proportion of the community uses them on a regular basis. As a result, latrine provision should be consistent with what is currently in place and what is considered appropriate in the community (WHO, 2006).

1.6 THE SAFE STRATEGY:

1.6.1 Putting the pieces together

Although the SAFE strategy's separate components have shown to be effective in preventing trachoma, the maximum success is expected when all four aspects are implemented together. Multiple components of the SAFE method have been studied in combination in some research. Receiving three rounds of azithromycin therapy, having a clean face, and increasing face-washing frequency were all independently linked to a lower prevalence of active disease in children in Ethiopia, according to a cross-sectional examination of implementation of the A, F, and E components (Ngondi et al. 2008). As a result, implementing the various SAFE components would have a compounding effect on trachoma control.

The whole SAFE method was implemented in five Ethiopian districts, with significant uptake of all components by the 3-year evaluation time-point (Ngondi et al. 2009a). In Zambia, SAFE methods reduced the prevalence of complete trachoma in children under the age of ten years from 55 percent at baseline to 10.6 percent after two years. In children, the prevalence of TF decreased from 24.9 percent to 4.5 percent, whereas the frequency of TT decreased from 0.6 percent to 0.3 percent in adults (Astle et al. 2006). In the lack of control groups, however, secular trend cannot be ruled out as a possible cause.

1.7 RESEARCH PROBLEM AND STATEMENT

The World Health Organization (WHO) has recognized trachoma among Seventeen neglected tropical diseases (NTDs) as a priority for surveillance and elimination through preventative treatments or better therapeutic and preventive strategies. (WHO2021,WHO2015a; Hotez et al., 2010). Poverty-related lifestyles, such as inadequate housing, poor hygiene, limited clean and safe water accessibility, and basic medical facilities, facilitate the spread of tropical diseases (Hotez et al. 2009). Trachoma is a leading cause of blindness, affecting millions of people across 51 endemic areas (WHO2021,WHO2012). It affects over 2.2 million people who have vision problems, with almost 1.2 million of them going blind permanently (WHO2012,WHO2015b). Active Trachoma occurs only when the Chlamydia trachomatis bacteria is infected, according to the WHO's simplified approach (Taylor et al. 2014). Long-term contact to these organisms causes redness or scarring of the tarsal conjunctiva, as well as twisting of the eyelashes, which can damage the cornea's surface. And this can lead to trachomatous trichiasis (TT), corneal opacity (CO), and lifelong and irreparable blindness. To foresee future characteristics of infectious illness and establish accurate control intervention methods to restrict disease breakout, a complete understanding of the dynamics of disease transmission among populations is essential (Lin et al., 2020; Yang et al., 2020). Mathematical modeling is one of the most important approaches for studying the dynamics and control of infectious diseases (Lin et al., 2020, Sofia et al., 2015). Designing and regulating epidemic models is a challenging and time-consuming effort because to the inherent nonlinearity, complexity, and parameter uncertainty associated with epidemic processes.

Simulation and modeling were used to study the dynamics of neglected tropical illnesses (specifically, Blinding diseases). These models not only represented the mathematical process of infectious disease, but they also provided useful information on the disease's possible control and spreadBrauer2012. However, the majority of current models in the literature focus on simple SI, SEI, and SEIR (human - human transmission) compartmental models.(Shattock et al 2015, Blake et al 2010, Gambhir et al2009, Pinsent et al 2018) Moreover, none of the models have ever examined a

trachoma Host-Vector transmission dynamic, in which the disease vector's contact rate plays a substantial role in reducing the prevalence of trachoma in the population. As a result, their analysis may have been too simple to be reliable in judging the situation. As a result, developing more advanced models that might give effective approaches for analyzing and describing the nature and control of trachoma remains a difficult undertaking. Nonetheless, creating a sophisticated model like this to emphasize management strategies for the trachoma epidemic is very consuming.

The current study proposed a novel multi-strain SEIR-SEI model that considers both person-to-person and disease vector transmission to provide some insights into the dynamics of trachoma and to propose appropriate control interventions in order to meet the World Health Organization's 2030 goals of eliminating trachoma as a global public health challenge. We used field data samples from Northern Nigeria to conduct parameter estimations and model fitting using the least-square fitting approach, and we achieved some simulation findings for the reproductive number as a function of two biological parameters using mesh plots.

Our new research report supplemented some of the prior studies in the literature (Shattock et al. 2015,Blake et al. 2010,Gambhir et al. 2009,Pinsent et al. 2018). Whereas the new $SEI_{h,c}R - SEI$ model includes the following additions: (i) Multi-strain infectious classes (ii) Disease-Vector population dynamics (iii) Model fitting and parameter estimates with real cases (iv) Sensitivity analysis to emphasize the impact of each parameter on epidemic control.

1.8 RESEARCH AIM AND OBJECTIVES

This study aims to design and scrutinize a systematic compartmental model for the transmission dynamics of trachoma epidemics in order to investigate how control measures can be put in place over time to minimize the incidence of active trachoma TF/TI to the lowest possible level while minimizing the cost of intervention.
The achievement of this goal would be aided by the following research objectives:

1 Develop a new mathematical model to investigate the dynamics of Blinding Trachoma with a saturation incidence rate.

2 Design and analysis of a Comprehensive Local Sensitivity Analysis to identify the impact of each parameter on modifying the size of the basic reproduction number so that management techniques may be prioritized.

1.9 SCOPE OF THE STUDY

The focus of this study is on analyzing the dynamics of the Trachoma epidemic. The research is based on the compartmental Kermack-Mackendrick model $SEI_{hc}R - SEI$ (susceptible-exposed-infectious-Recovered/ susceptible-exposed-infectious). The the whole populations are divided into eight groups in the proposed trachoma model: susceptible Humans (group of people who are healthy but can contract the disease), exposed Humans (group of people who have been in contact with the active trachoma agent but have not shown clinical symptoms of trachoma), and infectious Humans (individuals who have already developed the disease and now are infected).

There are also Susceptible Flies (healthy eye-seeking flies that can contract the disease), Exposed Flies (flies that have already been exposed to the disease but do not show clinical symptoms of trachoma), and Infectious Flies (Flies that have already developed the disease and can now spread). Two alternative scenarios will be used to address the research objectives. The first refers to the efforts of the World Health Organization (WHO) and other health professionals to ensure that the trachoma epidemic is properly controlled. The importance of disease vector contact rate will be statistically demonstrated. The Lyapunov Function Theory is used to demonstrate the system's asymptotic stability analytically, MATLAB/SIMULINK 2019b will be used to carry out all of the numerical simulations.

The research is restricted to the deterministic compartmental model, which takes into account the disease's characteristics at the population level. The Trachoma dynamics are studied in eight compartments, with two population groups (Human population and Vector population), the vulnerable human class, exposed human class, early infected human class, severe infectious human class, and recovered human class. There are three groups of flies in the vector population: susceptible flies, exposed flies, and infected flies. As part of the practical and practicable design process, certain estimations, choices, and assumptions are made.

1.10 THESIS ORGANIZATION

This thesis report is divided into five chapters, with the study objectives being thoroughly discussed in chapters 3 and 4, that could be studied separately. The following is a list of the chapters:

Chapter 1: The study's background is described in this chapter. The statement of the problem, objective, and scope of the study are also discussed in this chapter.

Chapter 2: The basic theories of epidemic modeling are reviewed in Chapter 2. It also includes a thorough and concise summary of the Trachoma pandemic and related research.

Chapter 3: This chapter depicts the construction of a new Trachoma dynamics model that takes into account the saturation incidence rate, the stability analysis of the equilibria, extensive local sensitivity analysis, and model fits of the proposed model are also included in this chapter.

Chapter 4: This chapter describes the numerical simulations of the Trachoma models step by step, as well as a detailed discussion of the results.

Chapter 5: The conclusion of the thesis work, as well as recommendations for future work, are presented in this chapter.

CHAPTER 2
LITERATURE REVIEW

2.1 INTRODUCTION

The literature review of the pertinent studies is offered in this chapter. The chapter opens with a review of the core theories of mathematical epidemiological modeling and basic mathematical epidemic models. The chapter also highlights research organizations' and public health programs' efforts to control and eradicate blinding trachoma as a public health issue. Finally, a detailed and concise review of existing research on mathematical models for trachoma control and elimination is presented.

2.2 MATHEMATICAL MODELLING OF EPIDEMIOLOGY

Epidemiology is the study of health and illness trends, as well as related factors, in a specific population. The word ”epidemiology” comes from the Greek terms ”epi” which means ”over,” ”demos” which means ”people,” and ”logos” which means ”study.” As a result, epidemiology is defined as the study of disease trends across human groups. The term ”epidemiology” is thought to have been coined in 1802 by a Spanish physician named de Villalba to describe the study of epidemics (Martcheva, 2013). Hippocrates (460-377 B.C.E.) is regarded as the father of epidemiology due to his pioneering work in establishing the disease-environment relationship. Heart attacks, cancer, and stroke have all gotten a lot of attention in epidemiology recently (WHO, 2018). It's important to define the phrases endemic, epidemic, and pandemic in epidemiological terms. Endemism refers to a disease outbreak that continues, epidemic refers to a sudden growth in the disease population, and pandemic refers to a worldwide sickness that affects a large number of people (Brauer, 2017). Infectious disease is defined as a clinically proven sickness caused by the infection of a pathogenic microbial agent, which can be parasitic, fungal, bacterial, or toxic protein. Bacterial agents, for example, cause pneumonia and tuberculosis; fungal illnesses, such as dermatomycoses; parasitic infections, such as helminth and protozoa; and virus infections, such as influenza and HIV, cause disorders. Several factors influence the disease's spread among a community, including population growth, poor

sanitation in poorer countries, and contemporary infrastructure that allows for cross-border travel.

The disease is frequently spread from person to person, either directly or indirectly. Indirect contact occurs when infectious material, is passed via the body. Physical contact or sexual engagement are examples of direct interaction. Influenza is conveyed by indirect contact, whereas the human immunodeficiency virus (HIV) is spread directly. When a healthy individual inhales infectious air, another form of transmission occurs. Tuberculosis, smallpox, chickenpox, pneumonia, and measles are examples of infectious diseases that fall within this category. In addition, for modeling purposes, multiple types of transmission are assumed: direct, where the pathogen is transferred from human to human; vector-spread, where the pathogen is transferred from human to human; vertical, where the pathogen is transferred from mother to child at birth; and environmental, where the pathogen is transferred from human to human through the environment (Hethcote, 2000; Sokat et al., 2019).

2.3 BASIC MATHEMATICAL EPIDEMIC MODELS

Epidemic models are mathematical models that depict the transmission of disease infections in humans. The phrase ”mathematical model” refers to the depiction of a system using mathematical terms and methods. Mathematical models may be used to any technical or natural science activity, including epidemiology, biology, or other well-defined systems. A mathematical model is created to accurately characterize a system, assess the effects of its different components, provide a rational understanding of experimental results, interpret the system's response pattern, predict future behavior, and improve system performance (Martcheva, 2013).

The development of epidemic models is seen in Figure 2.1. The first step is to get a scientifically correct description of the system. The system behavior is then represented using mathematical equations (typically differential equations). Following that, a thorough mathematical study is carried out to demonstrate the existence, uniqueness, and stability of the model's solution. The model is validated using reliable experimental data through model fitting, and the model's parameters

may be calculated once the model is developed and the mathematical analysis is finished. This is followed by a sensitivity study to determine the impact of different system parameters on system performance. Simulation results can also provide a lot of helpful insights (Martcheva, 2013).

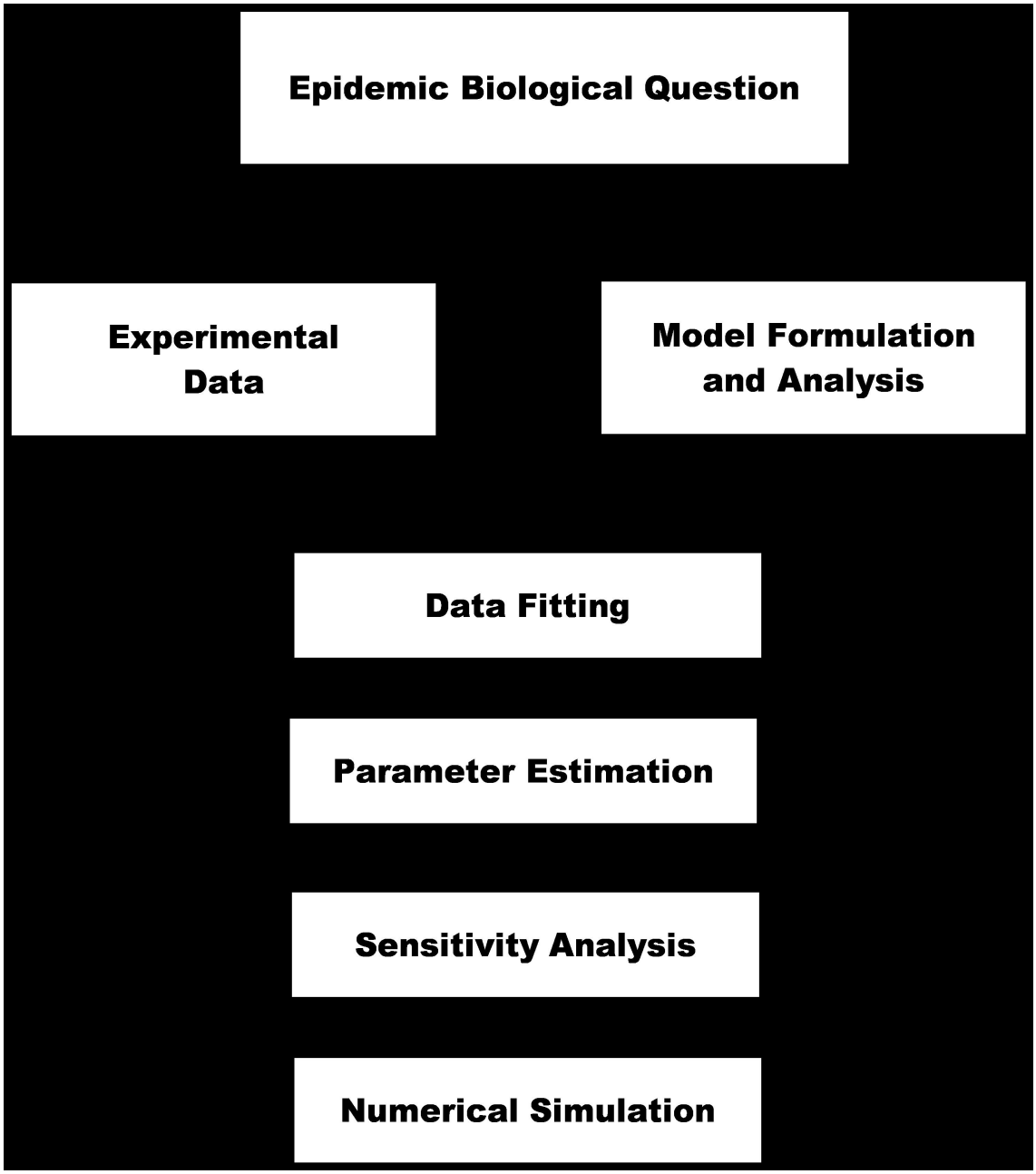


Figure 2.1. Developmental Stages of a typical Epidemic model. (Martcheva, 2013)

The focus of this research was on a deterministic compartmental epidemic model. Stochastic models, on the other hand, have been designed and used in the literature (Allen, 2017; Britton, 2010; Rao, 2014). The state variables in stochastic models are represented by probability distributions, whereas in deterministic models, the state variables are uniquely defined by model parameters and the starting states of the variables (Allen, 2017). The origins of compartmental epidemic models may be traced back to Kermack and McKendrick's efforts in the 1920s (Wilkinson, Ball, and Sharkey, 2016). The nature of a disease epidemic at the population level is taken into account in these models. Separate segments are developed for each individual in the population. Individuals were transferred from one group to another based on a set of mathematical parameters that served as a connection between compartments. Differential equations are used to evaluate compartmental models, which are used to predict disease progression behavior such as prevalence, mortality, and epidemic length. It also teaches you how to handle the condition most effectively. The primary forms of compartmental models are described in the following subsections; however, for a detailed analysis of the subject, see (Keeling Rohani, 2008).

2.3.1 SIR epidemic model

Kermack and McKendrick established the SIR model in 1927, and it is one of the most fundamental epidemic models (Wilkinson et al., 2016). In the literature, there are several variations of the SIR model, most of which change the primary model to accommodate extra data (Ameen, Baleanu, Ali, 2020; Wang, 2015; Zhang, 2015). The SIR model divides people into three categories to describe how the disease spreads. The first category, referred to as susceptible (abbreviated as S), consists of persons who are healthy yet vulnerable to the disease. Individuals who have already developed the illness and are now infected make up the second category, infectious (indicated by I). Individuals who have recovered from the illness are placed in the recovered compartment (designated by R).

Figure 2.2 depicts the process of moving from one group to another. The transmission rate is provided by β when susceptible individuals become infected and migrate from the S to I compartments. The recovery rate, indicated by α, represents the rate at which

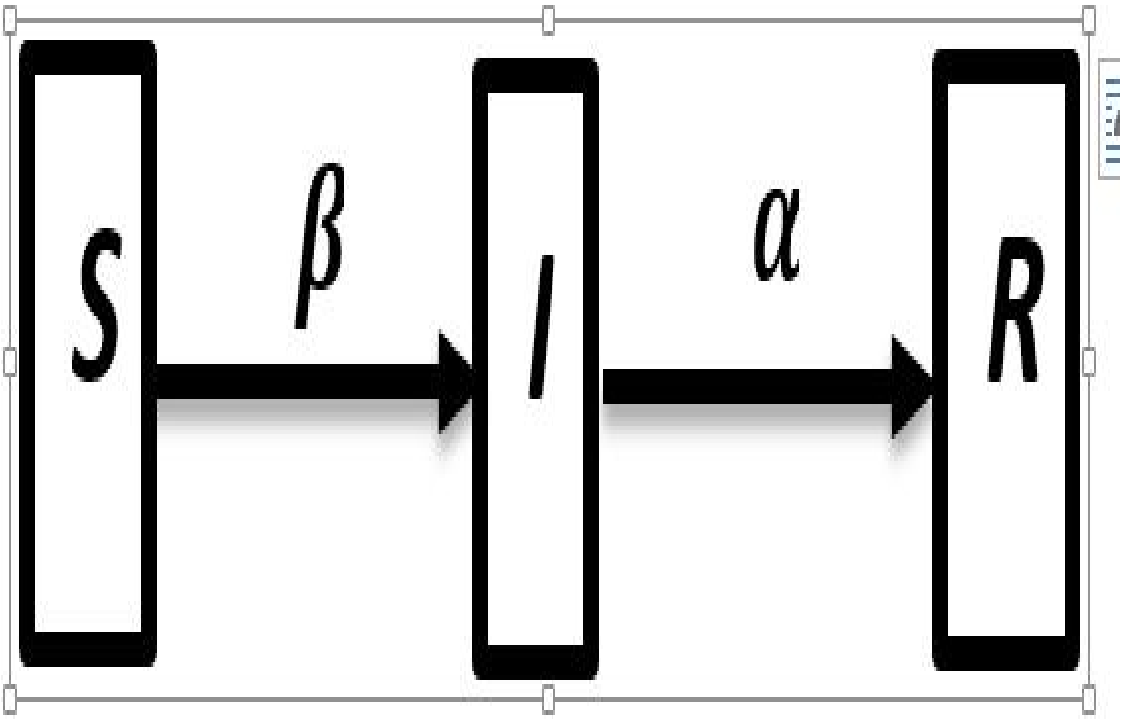


Figure 2.2. A Typical SIR model. (Meraf et al. 2009)

people who have recovered or died shift from the infectious to the recovered groups. S(t), I(t), and R(t) represent the number of individuals in the three compartments as a function of time (t). As a result, the entire population size N(t) is equal to the sum of the numbers of individuals in each of the three compartments, i.e. $N = S(t) + I(t) + R.(t)$. (Wilkinson et al., 2016)

2.3.2 SEIR epidemic model

The SEIR model, in which an extra compartment, exposed, indicated by E(t), is placed between the susceptible and infected classes, is another important form of epidemic model. This is because, unlike in the SIR model, when susceptible people come into touch with the disease agent (are exposed), they do not immediately progress to the infected class (Huang, 2008). The pathogen needs time to proliferate and establish itself in the host. The latent stage refers to when a person becomes sick but is not yet contagious.

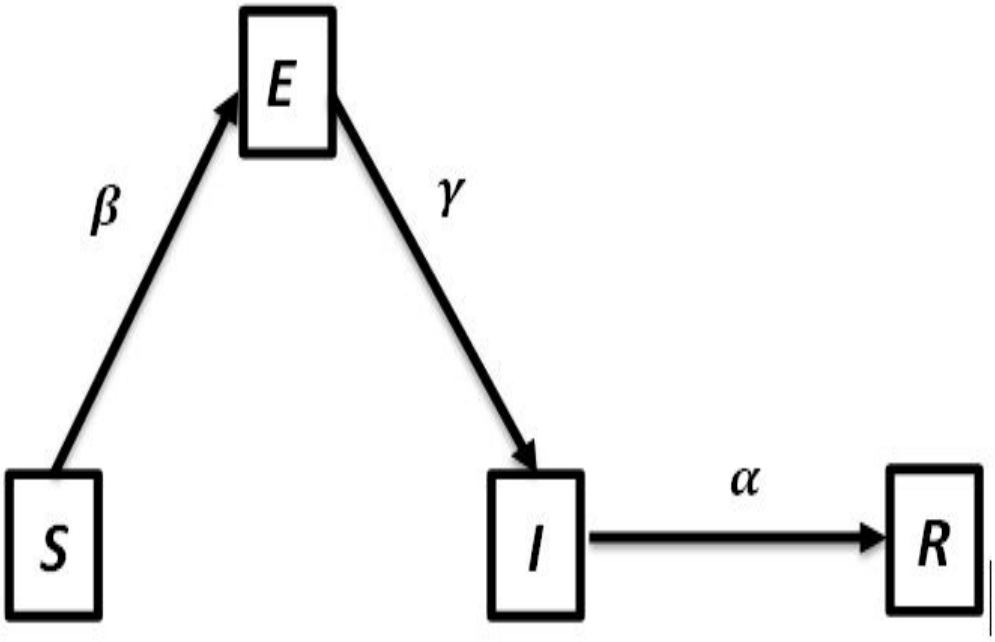


Figure 2.3. A simple SEIR model (Huang, 2008)

2.4 BASIC REPRODUCTION NUMBER

Sir Ronald Ross initially proposed the concept of a basic reproduction number in his book "The Prevention of Malaria," published in 1911. In this study, the scientist found that malaria is transferred between mosquitos and people. He subsequently developed a mathematical model to characterize malaria dynamics and calculated a threshold quantity, which is now known as the basic reproduction number (Hardy Magnello, 2002). During the infectious cycle, the basic reproduction number, represented as R_0, is defined as the expected number of new infections that might be caused by only one infected person in a populace of completely susceptible people. For basic models, R_0 may be calculated by tracking new cases in the populace caused by a single infectious agent and employing the formula given:

$$R_0 = (rate of contact) \times (probability of infection in a contact) \times (period of infectiousness)$$

However, adopting the new instances technique is not possible for complicated epidemic models with heterogeneity or seasonality, as well as varied susceptibilities. To address these flaws, a more generic approach based on a specific square matrix

known as the next-generation matrix (NGM) was developed by (Diekmann, Heesterbeek, Metz, 1990; Roddam, 2001). R_0 is the spectral radius of the'next-generation operator' in the next-generation matrix method. The operator creation entails identifying two compartments from the model: infected and non-infected compartments (Roddam, 2001). Consider a compartmental epidemic model with m compartments and n infected compartments to illustrate this idea. Let x_i for $i = 1, 2, ..., m$ denote the number of people in the ith compartment, $F_i(xi)$ denote the rate at which new infections occur in compartment i and $V_i(x_i) = V_i^-(x_i) - V_i^+(x_i)$ denote the rate at which individuals are moved from the i^th compartment and V_i^+ denote the rate at which individuals are moved to the i^th compartment.

The NGM operator G can be described as a product of two partial derivatives matrices of F_i and V_i :, according to Hefferman (Heffernan et al., 2005).

$$G = FV^{-1} \tag{2.1}$$

and

$$R_0 = \rho(FV^{-1}) \tag{2.2}$$

Where $\rho(G)$ is the dominant eigenvalue (spectral radius) of matrix G,

$$F = \left[\frac{(dF_i(x_0)}{(dx_j)}\right], \qquad V = \left[\frac{(dV_i(x_0)}{(dx_j)}\right], \tag{2.3}$$

x_0 = disease free equilibrium and $i, j = 1, ..., n$ During Epidemiological modelling, R0 is considered to be the threshold quantity to monitor the dynamics of an infectious disease (Hardy and Magnello, 2002).

2.5 MODEL FITTING

A mathematical model is validated via model fitting. It is a procedure for determining the conformance of a mathematical model to real-world facts (Halder Bhattacharya, 2011). Model fitting is frequently used to obtain parameter estimates after designing , and it also gives high model dependability. It is assumed that data for at least one class in the model is provided in time-series format in order to fit an epidemiological model. The least-square methodology is the most often used model fitting method in epidemiology. The model response curve is fitted thru the data points in the least-squares technique such that the sum of the squares of the residual between the data

points and the points on the fitted curve is as little as possible. For example, if we want to fit the incidence of a disease I(t), and we have real data in the form of time series like $(t_1, Y_1), \ldots\ldots\ldots\ldots, (t_N, Y_N)$, we should minimize the sum-squared error (SSE) as follows:

$$SSE = \sum_{i=1}^{N} [Y_i - I(t_i)]^2 \tag{2.4}$$

Trachoma prevalence data is obtained for fitting the suggested model (Sight savers and carter center 2018). However, because the epidemic model is given by nonlinear differential equations, minimization of (2.4) is a nonlinear optimization problem that cannot be addressed explicitly. Meanwhile, we use the Matlab2019b program to calculate the result for our research.

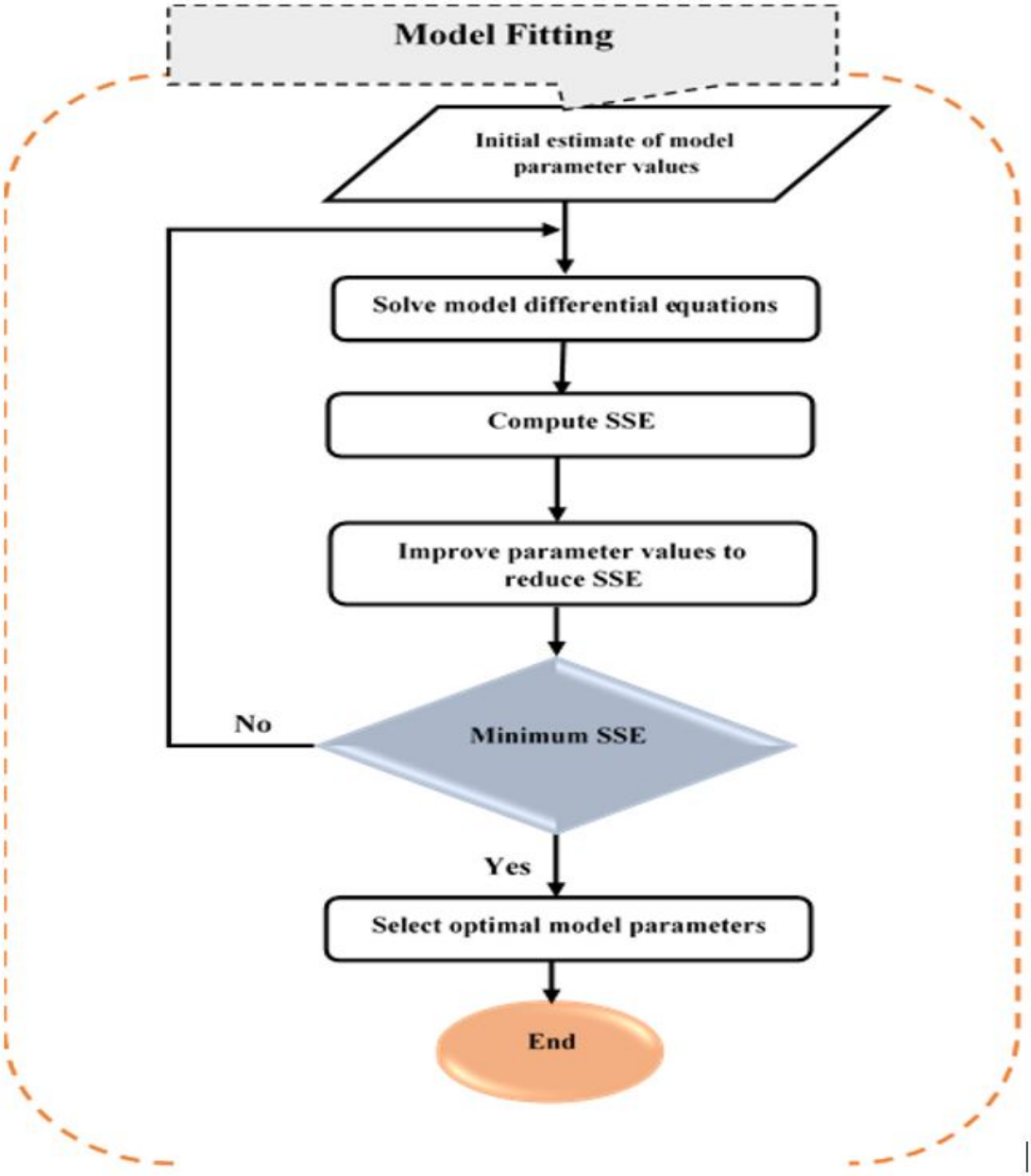


Figure 2.4. A Flowchart for model fitting(Marcheva, 2013)

There are two steps to the computation: first, the differential equation is solved, then the error function is iteratively minimized. The differential equations are solved with the initial values and an initial estimate of the parameter values is given. After that, the SSE is assessed, and the settings are changed to minimize the SSE. The differential equation is re-solved with the new parameter values, and the process is continued until the convergence of SSE (Martcheva, 2013).

In Summary, the steps for the whole process are as follows:

Stage A: Initially, the parameters of the model is estimated.

Stage B: use the estimated parameters to solve the differential equations of the model.

Stage C: Computation of the Sum-Square Error.

Stage D: Interchange or adjust the parameter values to ensure reduction in the Sum-Square Error.

Stage E: Re-run Stages B to D iteratively using the new parameters till when you obtain a minimal Sum-Square Error.

2.6 SENSITIVITY ANALYSIS

After estimating the model parameters, it is critical to discover the relevant model parameters that impact disease transmission when researching infections quantitatively. As a result, sensitivity analysis is used in computational modeling. This is crucial in identifying the main input parameters that should be the focus of attention if the disease is to be controlled. The sensitivity analysis is used in this work to determine the uncertainty of the parameters in the proposed Trachoma model using the basic reproduction number R_0 as a threshold quantity. The normalized sensitivity index is used in the sensitivity analysis. A local elasticity indices for the output y to input x is generally given by Ω_x^y (Woldegerima et al., 2018):

$$\Psi_x = \Omega_x^y = \frac{x}{y}\frac{dy}{dx} \tag{2.5}$$

2.7 DEVELOPMENTAL CYCLE OF CHLAMYDIA TRACHOMATIS

The developmental cycle of C. trachomatis is biphasic. The extracellular elementary body (EB), a tiny non-dividing environmentally stable infectious form of C. trachomatis seen in infectious discharge, is the initial stage of development. EBs seek for epithelial cells for entrance, which they require to gain access to nutrients and begin their metamorphosis into reticulate bodies (RBs). The ability of EBs to bind to the host cell and become absorbed into endocytic vacuoles is well known. Intracytoplasmic inclusions are formed when these vacuoles join. EBs begin to convert into RBs and begin replication within these inclusions.) The RBs are a non-infectious variant of C. trachomatis with a strong replicative capacity. RBs gain access to nutrients necessary for life within the cytoplasm of their hosts. Within the inclusion, RBs divide and reproduce. The popular belief is that they divide via binary fission (Kuper et al. 2003, Hu et al. 2010), however investigations have shown that they divide by polarized cell division, which is comparable to budding (Polack et al. 2005).

After numerous rounds of division, RBs are transformed back to EBs; however, this process is slow and asynchronous. The exact mechanism by which the RB cell decides to transition from splitting into two RBs to transforming into an EB has yet to be discovered. According to one theory, the RB size must reach a specific threshold below which conversion happens; hence, RB size may govern RB to EB conversion (Hu et al. 2010). Another idea is that the separation of RBs from the inclusion membrane stimulates their transformation to an EB; the fundamental assumption is that conversion is hindered when the RB is in touch with the inclusion membrane through its numerous projections (Mariotti et al. 2009), Thylefor et al. 1987). Only EBs are infectious, therefore converting RBs to EBs is crucial (Mariotti et al 2009). The EBs are subsequently freed from their confinement and discharged into the extracellular environment. The entire developmental cycle takes around 48–72 hours, and an one infected cell can produce up to 1000 ‘progeny’ (Mariotti et al 2009).

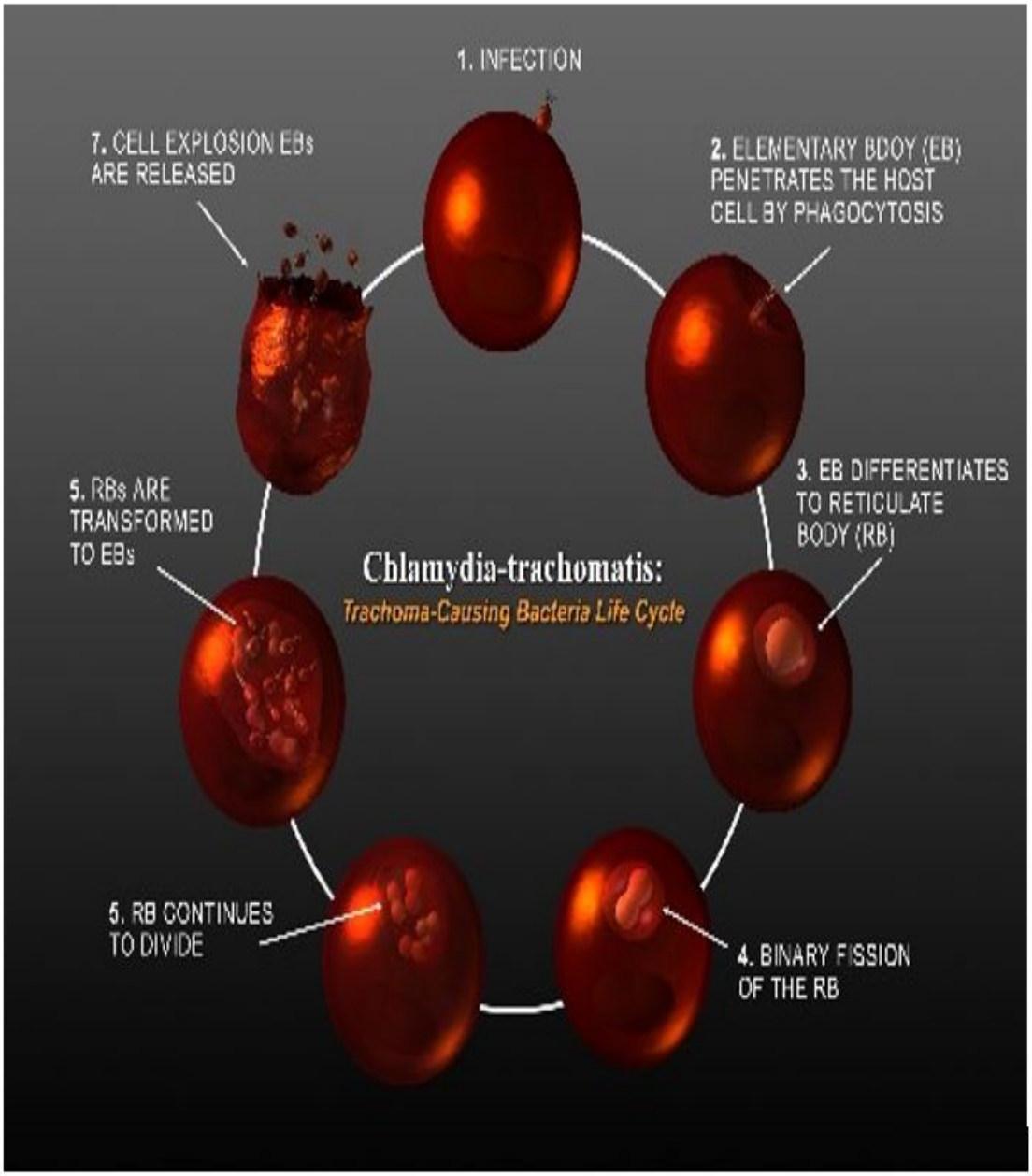


Figure 2.5. the life cycle of Chlamydia. Source: Raulston, Jane E., and Priscilla B. Wyrick."Chlamydia." Encyclopedia of Microbiology. 2nd edition.2000.

2.8 EPIDEMIOLOGY OF TRACHOMA

Epidemiology of trachoma C. trachomatis infections in the eyes are common. Newborn ocular infections are caused by passage via a birth canal contaminated with genital strains in industrialized countries. Because the danger of re-infection and subsequent scarring and entropion is practically non-existent in these cases of ophthalmia neonatorum, they are not trachoma. Trachoma necessitates the existence of C. trachomatis infection as well as an environment that permits children to acquire many infections over years of exposure. Trachoma-endemic areas in low-income countries are marked by poverty and marginalization, with poor access to water and sanitation.; This condition promotes the transmission of infected ocular and nasal secretions, increasing the risk of recurrent infections, protracted inflammation, and blinding complications (Wand et al. 1967, Kari et al 2011). Trachoma does not have an obligatory intermediate host like onchocerciasis (black flies) or malaria (mosquitos); transmission is only from person to person via contaminated secretions. Eye-seeking flies may also transfer ocular secretions in some situations, although flies are not an obligatory vector, and trachoma may be found in many places without flies.

Ocular chlamydia infection is a chronic conjunctivitis in trachoma-endemic cultures, and trachoma clinical symptoms vary with age (Figure 2.6). Conjunctival infection, follicles, and inflammation are most commonly observed in preschool-aged children, who are thought to be the community's infection reservoir (Kari et al. 2011). Children may have stellate scars, although trachoma scarring is more common in young adults, and females are more likely to have it (Keenan et al. 2011). Entropion and trichiasis, which are the result of continuous scarring, are considerably more frequent in women and visible in people in their middle and later years. The most frequent age group for trachoma-related corneal damage is the elderly once. There is presently no therapy or intervention available for corneal opacification or scarring. Stopping the active illness in youngsters is the most effective strategy for preventing these late trachoma symptoms.

The World Health Organization (WHO) simplified grading scheme which is used to report the prevalence of active trachoma and the sequelae in population based surveys

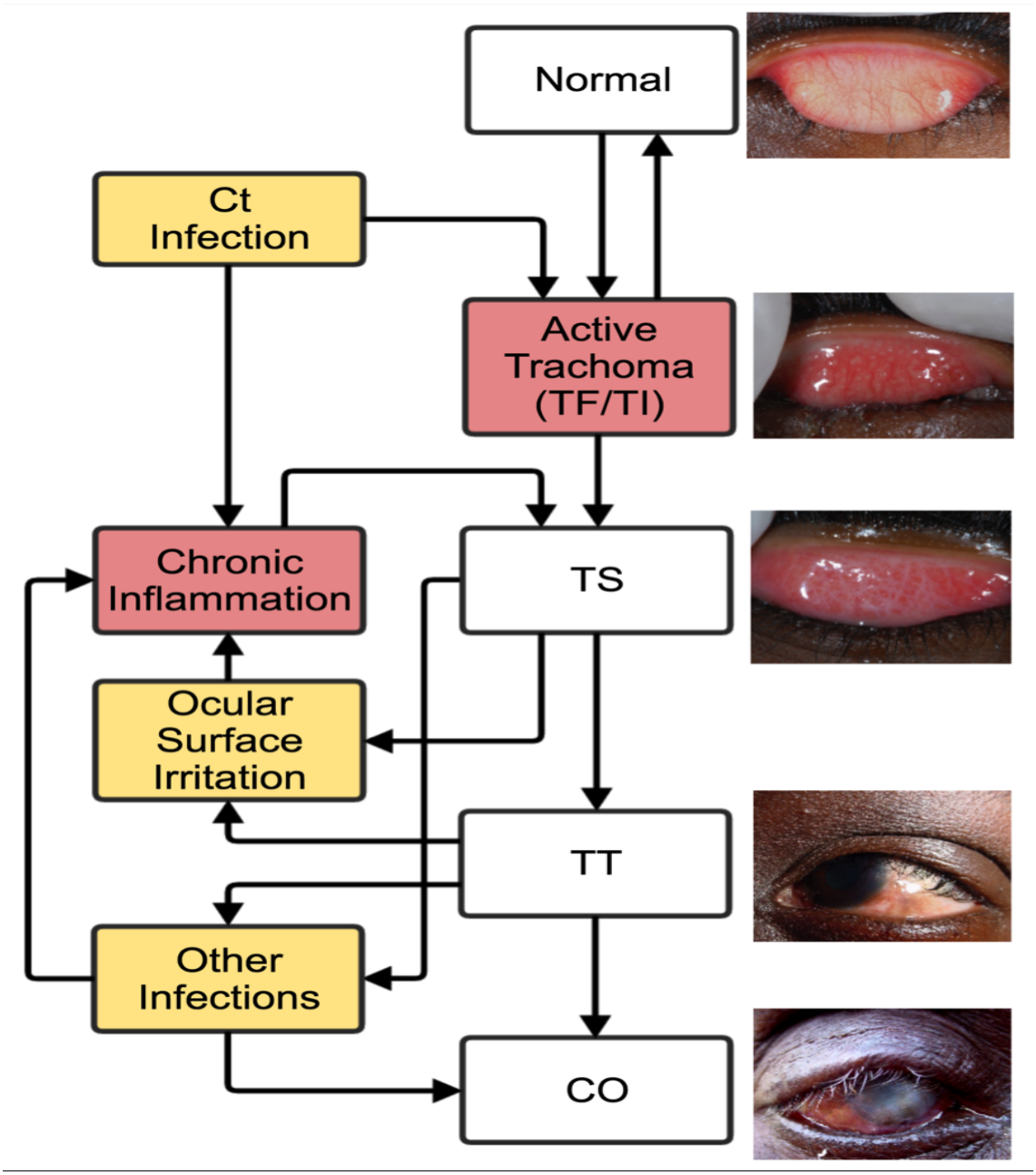


Figure 2.6. The infectious stages of trachoma (Meraf et al. 2009)

(Meraf et al. 2009, Munoz et al. 2011).

In population-based surveys, the World Health Organization (WHO) has established a simple grading scheme use to assess the incidence of active trachoma and its consequences (Munoz et al. 2011). Follicular trachoma (TF) is described as a condition in which the upper tarsal conjunctiva contains at least five follicles measuring 0.5 mm in diameter (within a defined area for grading). Trachoma-intense (TI) is present when there is enough inflammatory thickening to conceal at least half of the deep tarsal arteries. The conjunctiva is classified as having trachomatous scarring if the scarring is plainly apparent (TS). The presence of epilation or at least one lash contacting the globe is considered trachomomatous trichiasis (TT). When the opacity is sufficient to conceal the pupillary edge, it is called corneal opacity (CO).
These trachoma clinical symptoms represent the disease's normal course and have implications for trachoma eradication initiatives. The first two, TF and TI, indicate the amount and severity of trachoma that is "active." The WHO now advises evaluating the incidence of TF in children aged 1 to 9 years to assess the need for MDA, evaluate the efficacy of elimination programs, and monitor for re-emergence following program termination. Scarring is a predictor of trichiasis risk in the long run. The incidence of trichiasis is essential to plan surgical treatments, and the prevalence of trachoma-related corneal opacity is an indicator of the public health impact of trachoma-related vision loss.

After the infection has been eradicated, the clinical symptoms of active trachoma require time to disappear. In children with active trachoma, the incidence of infection varies based on the intensity of the inflammation, the amount of trachoma endemic regions in the society, and the period after treatment. In highly endemic regions, nucleic acid amplification tests show that 30 to 50 percent of children with TF have signs of infection, while up to 80 percent of children with TI may have infection. When grading for active trachoma is done immediately after MDA, the link between clinical symptoms and infection is poor (Stare et al. 2018, Michel et al. 2011).

Furthermore, as the infection grows increasingly rare, evaluating the clinical

symptoms of active trachoma becomes more challenging. As a result, interest in other potential techniques than clinical evaluation that trachoma programmes may employ to gauge progress toward eradication is rising. A test for infection is one tool being investigated. For C. trachomatis, there are extremely sensitive and specific nucleic acid amplification assays, as well as one that may be used in the field (Solomon et al. 2006, Keenan et al. 2012, See et al. 2011, Yang et al. 2007). Even with specimen pooling techniques (Harding et al. 2011), the expense of collecting, storing, and evaluating ocular specimens is considerable. Furthermore, there are inadequate statistics on the frequency of infection in the community, indicating that re-emergent trachoma is unlikely.

Several studies have found trachoma re-emergence months after populations had been free of infection, while others have found no persistent trachoma re-emergence even after infection was re-introduced into the community (Hu et al. 2011, Weiss et al. 2011). Even with a 1 percent incidence of infection in children aged 1–9 years, a monitoring study done 4 years after the program's end showed no trachoma (West et al. 2005).

Antibodies to C. trachomatis antigens have emerged as a potential technique for trachoma monitoring (Burton et al. 2005). Serologic testing in children might represent cumulative exposure to C. trachomatis within communities since antibodies to chlamydial antigen pgp3, a plasmid antigen, appear to be very long-lasting (West et al. 2005). Antibodies in young children that are absent or have a low frequency after years of trachoma program efforts may suggest that transmission has been interrupted. This situation was found in Tanzania and Nepal, where the incidence of clinical disease was less than 5percent in children in areas where program operations had ceased between 2 and 10 years ago; surveillance surveys indicated a very low frequency of antibodies (Burton et al. 2005, West et al. 2011). Sero-reversion may also occur in the absence of continuing transmission, according to the evidence (Gebre et al. 2011). Unfortunately, despite its high immunogenicity, the pgp3 antigen is not specific to C. trachomatis ocular strains Children exposed at birth in locations with high incidence of genital infections may test positive as a result of a protracted respiratory illness. On order to identify the role of genital chlamydia infection in the

seropositivity of children in a trachoma-endemic area, more research is needed.

Trachoma's global profile has undoubtedly altered over time. Trachoma was formerly prevalent in most nations, but it was eradicated from Europe and North America long before antibiotics were developed. By the early twentieth century, the disease had vanished due to socio - economic progress, particularly improved water and sanitation, which reduced C. trachomatis transmission to the point where it could no longer be sustained all through the society. Trachoma remains endemic in Africa, Asia, and the Middle East's poorest and most remote regions. Trachoma did affect the most vulnerable people in society of those communities, women and kids, who often have the fewest resources to address health issues. In 2016, an estimated 190 million people lived in trachoma-endemic areas across 37 countries, with 1.9 million suffering from trachoma-related vision loss (West et al. 2011).

In conclusion, the epidemiology of trachoma is marked by an active, infectious stage in children, followed by scarring and trichiasis in older people, especially women. It's a communicable disease spread by contaminated ocular and nasal secretions from person to person. To be successful, trachoma eradication necessitates a multifaceted public health approach that involves widespread medication administration of antibiotics.

CHAPTER 3
MATHEMATICAL MODELLING AND ANALYSIS OF THE SEIR-SEI MODEL FOR THE DYNAMICS OF BLINDING TRACHOMA

An SEIR-SEI model is develop in tis chapter to analyse the transmission dynamics of blinding trachoma, considering both the effect of human tohuman transmission and vecto-human transmission. In human pupulation, we divide the infection into two groups : Trachomatous Folliclar (TF), Trachomatous Intense (TI) and Trachomatous Scares (TS) belongs to the 1st group of infection, while Trachomatous Trachiasis (TT) and Coneal Opacity (CO) belongs to the 2nd group infection. By vector-human transmission we mean the spred of Chlamydia Trachomatis through the disease vector called (musca sorbens) or eye seeking fly. During the analysis, it is prove that , disease-free equilibrium point and endemic equilibrium points exist, where the local assymptotic stability of the DFE is confirm at a poimt when $R_0 < 1$ (i.e the basic Reproduction number is less than unity) and not stable if its greater than unity. Similarly, The global stability of the endemic equilibrium point is guaranteed unsing the idea of Lyapunov function when $R_0 > 1$. In addition, some parameters of the model are estimated and fitted using the real cases of trachoma form nothern nigeria, we also presented a local sensitivity analysis to monitor the influence of the individual parameters in changing the size of basic reproduction number for feasible control and elimination of the epidemic.

3.1 INTRODUCTION

The blinding trachoma has been a primary source of visual impairment which reportedly affects millions of people in 51 endemic settings (WHO 2012). It affects approximately 2.2million individuals with visual impairment, of which nearly 1.2 million remain permanently blind (WHO 2012,WHO 2015b). Active Trachoma, as reported in the simplified scheme of the world health organization(Taylor et al 2014), arises only when infection with the Chlamydia trachomatis bacterium occurs. Prolonged exposure to these organisms result in an immunopathological reaction identified through redness or scaring of tarsal conjunctiva, and subsequent twirling of

the eyelashes that damage the surface of the cornea. And this may contribute to trachomatous trichiasis (TT), opacity of the cornea (CO), as well as permanent and irreversible blindness.

Trachoma is also found to be significantly associated with morbidity and mortality (Hotez et al 2010). The Disability Adjusted Life Years "DALY" estimates that were attributable to trachoma seem to be dynamic. The 1990 Global Burden of Disease (GBD) relevant literature reveal that the trachoma prevalence (all age groups) of about 144000.00 [95 percent interval (95 percent U.I), 104000.00 to 189000.00] DALY.

While 2010 "GBD" research recorded about 334000.00 [95 percent U.I 243000.00 to 438000.00] (Murray et al 2012). Some scholars determine the number as minimum DALY of 1 million (Evans et al 1995). Sub-Saharan Africa produced as much as 3.6 million, with the highest percentage (72 percent) (Frick et al 2003). In 2010, Trachoma accounted for 5.2 percent Africa's cause of visual impairment (Naidoo et al 2014). Nonetheless, a reliable quantitative estimation of the trachoma load persists and is attributed to a numerous of factors, such as the limited data that restricts the ability to get reliable estimation on the number of infected individuals and the unanswered question about the status of trichiasis, "a sequela for debilitating disease?" (Burton et al 2009).

Chlamydia T. can be transmitted via 2 main ways. One of those is direct physical contacts with infectious person or by interaction with clothes that encountered contaminated eye discharges (Burton et al 2009). Other path includes transmission through an eye-seeking fly [musca sobens] wich touched the discharge from the eyes or nose of an infected individual (Emerson et al 2004). In order to sustain infection transmission it shall constantly be transmitted between individuals. The conditions of untreated patents depends on such factors like aging as well as the length of exposure to the infection (Bailey et al 1999,Grassly et al 2008). The rate and the spread of the C. Trachomatis is also age-dependent; thus, at the childhood stage infections are higher (Bailey et al 1999,Grassly et al 2008)..Repetitive aged-related inflammation contributes to scar on the tarsal conjunctiva and eventually to traciasis, then Opacities of the cornea and total blind conditions as described earlier(Burton et al 2009,West et al 1991).

Modeling and simulation have been used to capture the dynamics of the neglected tropical diseases (specifically, Blinding Trachoma). These models described not only the infectious disease mathematical process but this do provide meaningful information on the possible control and spread of the disease(Brauer et al 2012). However, majority of the existing models found in the current literature concentrate their studies only on simple SI, SEI, SEIR (human – human transmission) compartmental models (Shattock et al 2015,Blake et al 2010,Gambhir et al 2009,Pinsent et al 2018), and none of the model has ever considered a Host-Vector transmission dynamic of trachoma, where the contact rate of the disease vector plays a significant role in curtailing the prevalence of trachoma in the populace. This might have made their analysis trivial and thus, not reliable enough to judge the situation. Therefore, it remains a challenging task designing more sophisticated models that can provide useful methods to analyze and describe the nature and control of trachoma. Nevertheless, it is tedious to design a complicated model of this kind to highlight management strategies for trachoma epidemic.

The current study proposed a novel multi-strain SEIR-SEI model that consider both person-person and disease vector transmission to provide some insights on the dynamics characteristics of the trachoma and propose suitable control interventions toward achieving the 2030 goals set by World Health Organization to walkaway trachoma as world's public health challenge. we have also performed parameter estimation of the model parameters and model fitting with the use of field data cases from Northern Nigeria using least-square fitting method, Some simulation results produced with the aid of mesh plots for the reproductive number as a function of two different biological parameters was obtained. Our new research article complimented some of the earlier mentioned studies in the literature (specifically, (Shattock et al 2015,Blake et al 2010,Gambhir et al 2009,Pinsent et al 2018) Where, the novel $SEI_{(h,c)}R - SEI$ model incorporates the following extensions: (i) Multi strain infectious classes (ii) Disease-Vector population dynamic (iii) Model Fitting and parameter estimation with real cases (iv) The sensitivity analysis, to highlight the influence of each parameter in controlling the epidemic.

3.2 FORMULATION AND DESCRIPTION OF THE TRACHOMA MODEL

The model is considered to be a compartmental model comprising of two groups population with two strain of infectious stages: Population of human and population of eye-seeking flies.The human population at time t, $N_h(t)$ is sub divided into five classes. viz; $S_h(t)$ Class of Susceptible human who are healthy but can become infected through direct or indirect contact with infectious individual or infectious flies. This class is increased through the recruitment of individuals at the rate Π_h and by the loss of infection-acquired temporary immunity at the rate φ and decreased by progression to exposed class at the rate λ_h and by natural death at μ_h.

$E_h(t)$ the exposed class of human population generated through the infection of the susceptible individuals at the rate λ_h and decreased by progression to show the symptoms of active trachoma at the rate δ_1 and decreased by progression to the recovered class at the rate ψ_1 and by natural death at the rate μ_h. $I_{hs}(t)$, the stage one of infectious class of human population which comprises (i) trachomatous inflammation follicle/intense stage of the trachoma (TF/TI), and (ii) trachomatous scarring stage (TS), this class is generated by population of exposed individuals who develop symptoms of the chlamydia trachomatis at δ_1 and decreased by progression to the next infectious stage at the rate δ_2 and by natural death μ_h its also decreased through progression to the removed class at rate ψ_2.

$I_{hc}(t)$, the stage two of infectious class, comprises Trachomatous trichiasis (TT) and Corneal opacity (CO), this class is generated by the progression from stage one of infectious class at the rate δ_2, and decreased by progression to removed class at the rate ψ_3 also reduced by natural death at the rate μ_h. $R_h(t)$, the population of recovered individuals generated by the removal of individuals from infectious classes at the end of infectiousness due to the application of one of the control measures mentioned above (SAFE) at rates ψ_1, ψ_2, and ψ_3. This class is decreased by loss of temporary immunity at rate φ and natural death at rate μ_h. Assuming the infection does not confer permanent immunity to re-infection in the recovered class (i.e $\varphi \neq 0$).

Similarly the flies population $N_f(t)$ is sub-divided into three disjoint compartment namely; Susceptible flies $S_f(t)$ generated by the recruitment rate Π_f and decreased by

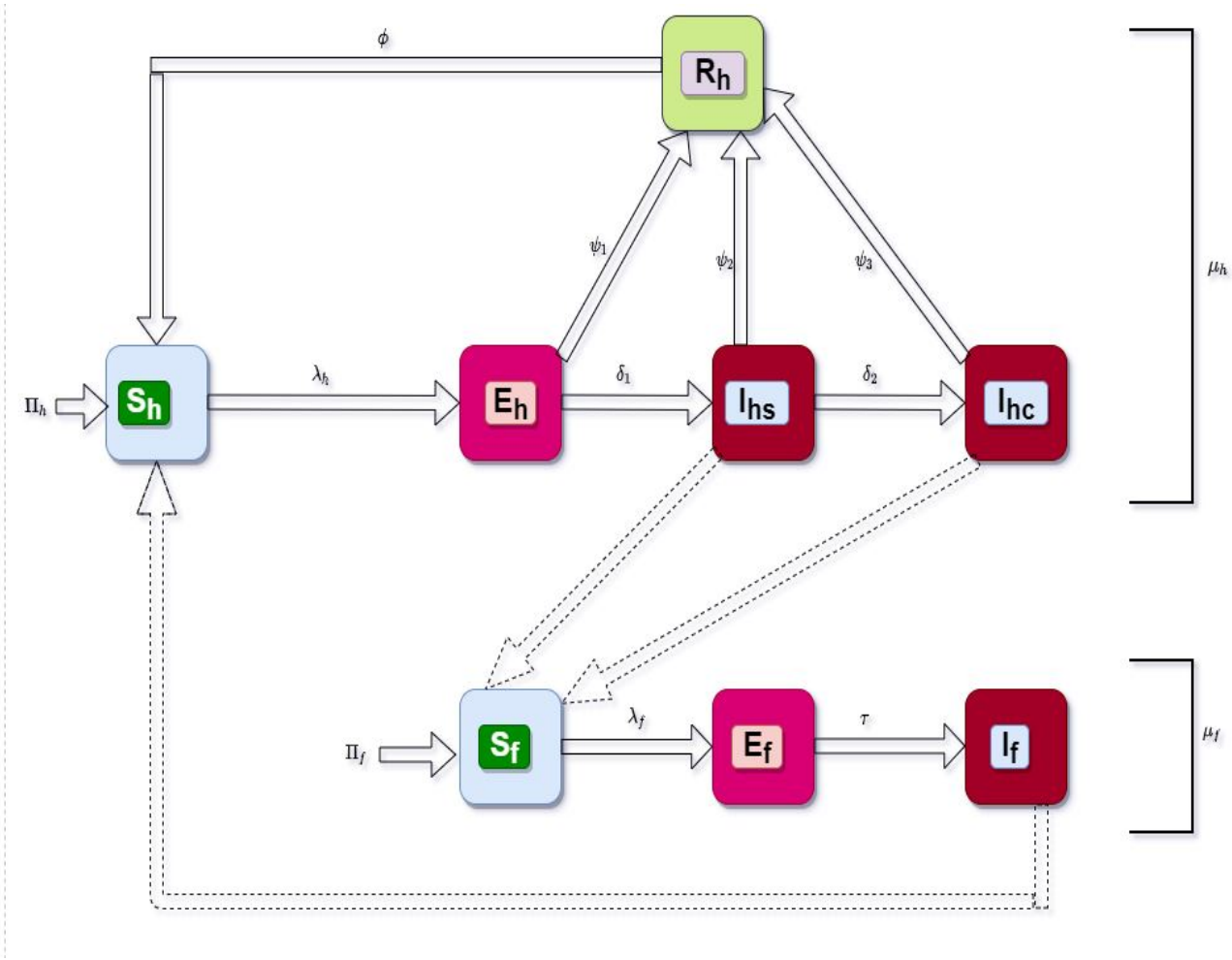


Figure 3.1. Schematic Diagram of the model

progression to exposed class at the rate λ_f and natural death at μ_f, the exposed flies $E_f(t)$ which is increased by susceptible flies population at the rate λ_f and decreased by progression to infectious class and natural death at the rates τ and μ_f respectively, Infectious flies $I_f(t)$ this is produced when the exposed class shows clinical symptoms of Chlamydia trachomatis and reduced by natural death at the rate τ and μ_f respectively. the period of infection of flies usually ends with their natural death due to nature of their petite life-cycle(i.e they dos'nt recover from infections), the reason why immune class does not appear in the flies population.

The total human and flies population of this model is presented as:

$$N_h(t) = S_h(t) + E_h(t) + I_{hs}(t) + I_{hc}(t) + R_h(t),$$

and

$$N_f(t) = S_f(t) + E_f(t) + I_f(t).$$

Using the above description and assumptions the model for the transmission dynamic of blinding trachoma, the model's associated deterministic system of non-linear ordinary differential equation is presented as:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Pi_h - \lambda_h S_h + \varphi R_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \lambda_h S_h - \delta_1 E_h - \mu_h E_h - \psi_1 E_h, \\
\frac{dI_{hs}}{dt} &= \delta_1 E_h - \delta_2 I_{hs} - \mu_h I_{hs} - \psi_2 I_{hs}, \\
\frac{dI_{hc}}{dt} &= \delta_2 I_{hs} - \psi_3 I_{hc} - \mu_h I_{hc}, \\
\frac{dR_h}{dt} &= \psi_1 E_h + \psi_2 I_{hs} + \Psi_3 I_{hc} - \varphi R_h - \mu_h R_h, \\
\frac{dS_f}{dt} &= \Pi_f - \lambda_f S_f - \mu_f S_f, \\
\frac{dE_f}{dt} &= \lambda_f S_f - \tau E_f - \mu_f E_f, \\
\frac{dI_f}{dt} &= \tau E_f - \mu_f I_f,
\end{aligned} \tag{3.1}$$

where

$$\lambda_h = (\beta_h \sigma_f \frac{I_f}{N_h}),\ \lambda_f = \sigma_f \beta_f (\frac{I_{hs} + I_{hc}}{N_h}),$$

are the forces of infection.

Table 3.1. **The state variables and parameters values used in the Trachoma model**

Variable	**Interpretation**
$S_h(t)$	Population of susceptible humans
$E_h(t)$	Population of exposed humans
$I_{hs}(t)$	Class of infective individuals with early stages of trachoma
$I_{hc}(t)$	Class of infective individuals with TT and CO stages of trachoma
$R_h(t)$	Population of Recovered humans
$S_f(t)$	Class of susceptible flies
$E_f(t)$	Population of exposed flies
$I_f(t)$	Class of infective flies
Parameter	**Description**
Π_h	Human recruitment rate
μ_h	Natural death rate of human
μ_f	Natural death rate of eye-seeking fly(musca sorvens)
Π_f	Flies recruitment rate
β_h	Rate of transmission from vector to host
β_f	Rate of transmission from host to vector
δ_1	Progression rate from E_h to I_{hs}
δ_2	Progression rate from I_{hs} to I_{hc}
τ	Progression rate from E_f to I_f
φ	Rate at which human looses immunity
σ_f	Contact Rate of the infected fly
ψ_1	Human recovery rate from E_h
ψ_2	Human recovery rate from I_{hs}
ψ_3	Human recovery rate from I_{hc}

3.3 BASIC PROPERTY OF THE MODEL

Eq. (3.1) is monitoring human and flies population. Therefore every parameter and state variable associated with the model are considered to be non-negetive for each $t > 0$.It is therefore instructive to show that all the state variables belonging to the model are non-negetive for all non-negetive initial condition before analysing the model.

Lemma 3.3.1. *Suppose* $(S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f)$ *is the solution of the Eq.* (3.1) *together with the initial condition,* $S_h(0) \geq 0, E_h(0) \geq 0, I_{hs}(0) \geq 0, I_{hc}(0) \geq 0, R_h(0) \geq 0, S_f(0) \geq 0, E_f(0) \geq 0, I_f(0) \geq 0$. *The closed set:*

$$\Omega = \left\{(S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f) \in \Re_+^8 \ni S_h + E_h + I_{hs} + I_{hc} + R_h = \frac{\Pi_h}{\mu_h}, S_f + E_f + I_f = \frac{\Pi_f}{\mu_f}\right\}$$

is positively-invariant and attractive with respect to the Eq. (3.1).

Proof. Adding the first five equations in Eq. (3.1), we get

$$\frac{dN_h}{dt} = \Pi_h - \mu_h N_h, \tag{3.2}$$

where

$$N_h = S_h + E_h + I_{hs} + I_{hc} + R_h.$$

In similar way, adding the last three equations in Eq. (3.1) we get

$$\frac{dN_f}{dt} = \Pi_f - \mu_f N_f, \tag{3.3}$$

where

$$N_f = S_f + E_f + I_f.$$

Since $\frac{dN_h}{dt} = \pi_h - \mu_h N_h$, it follows that $\frac{dN_h}{dt} \leq 0$ if $N_h(t) \geq \frac{\Pi_h}{\mu_h}$. It can be seen that the solution N_f of Eq. (3.3) approaches the equilibrium $\frac{\Pi_f}{\mu_f}$ as $\mu_f \to \infty$. By employing the concept presented in (Lakshmikantham et al 1989), we have, $N_h(t) \leq N_h(0)\, e^{-\mu_h t} + \frac{\Pi_h}{\mu_h}(1 - e^{-\mu_h t})$ particularly $N_h(t) \leq \frac{\Pi_h}{\mu_h}$ if $N_h(0) \leq \frac{\Pi_h}{\mu_h}$. This implies the set Ω is positively-invariant. Thust, if $N_h(t) > \frac{\Pi_h}{\mu_h}$ then the solution either enters Ω in finite time or $N_h(t)$ approaches $\frac{\Pi_h}{\mu_h}$. Hence all the solutions in $\Re_+^8$ are in Ω. This implies Ω is attracting. Consequently the model is considered to be mathematically well-posed and epidemiological sensible, as all the variables used throughout the model happen to be non-negative for every $t > 0$. Thus, it is adequate and enough to consider the dynamics of the Eq. (3.1) in Ω (Hethcote et al 2000). □

3.4 EXISTENCE OF THE EQUILIBRIA AND STABILITY ANALYSIS

In any research of epidemiological models there exist a threshold value usually known as basic reproduction number R_0 which is described as the average number of secondary infections generated by a single infected individuals during the epidemic in a completely susceptible population (Hethcote et al 2000,Anderson et al 1990,Kermack et al 1927).

3.4.1 The Disease-free equilibrium point

To obtain the disease-free equilibrium point (DFE) for the Eq. (3.1), we set the RHS of Eq. (3.1) to zero. Thus, ε_0=$\Big(S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f\Big) = \Big(S_h, 0, 0, 0, 0, S_f, 0, 0\Big) = \Big(\frac{\Pi_h}{\mu_h}, 0, 0, 0, 0, \frac{\Pi_f}{\mu_f}, 0, 0\Big)$. This means when there is no infection $E_h = I_{hs} = I_{hc} = E_f = I_f = 0$, the model has a unique disease-free equilibrium point

$$\varepsilon_0 = \left(\frac{\Pi_h}{\mu_h}, 0, 0, 0, 0, \frac{\Pi_f}{\mu_f}, 0, 0\right). \tag{3.4}$$

3.4.2 Local Stability of the Disease-free Equilibrium

The technique of next-generation operator as presented by (Diekmann et al 2010) is employed to determine a critical parameter called the Basic reproduction number R_0 which represent the average number of secondary cases produced by one infected agent throughout the duration of infectious period in a complete susceptible population. We used the recipe developed in (Van den Driessche and Wanmough 2000, Diekmann et al 1990). Now, we can rewrite Eq. (3.1) starting with the newly infectious classes.

$$\begin{aligned}
\frac{dE_h}{dt} &= \lambda_h S_h - \delta_1 E_h - \mu_h E_h - \psi E_h,\\
\frac{dI_{hs}}{dt} &= \delta_1 E_h - \delta_2 I_{hs} - \mu_h I_{hs} - \psi I_{hs},\\
\frac{dI_{hc}}{dt} &= \delta_2 I_{hs} - \psi I_{hc} - \mu_h I_{hc},\\
\frac{dE_f}{dt} &= \lambda_f S_f - \tau E_f - \mu_f E_f,\\
\frac{dI_f}{dt} &= \tau E_f - \mu_f I_f,\\
\frac{dS_h}{dt} &= \Pi_h - \lambda_h S_h + \varphi R_h - \mu_h S_h,\\
\frac{dS_f}{dt} &= \Pi_f - \lambda_f S_f - \mu_f S_f.
\end{aligned} \tag{3.5}$$

To investigate the linear stability of the DFE, we apply the technique of next generation matrix operator to Eq. (3.5) (Van den Driessche and Wanmough 2000,Diekmann et al 1990). The jacobian matrices f and v which are evaluated at DFE denoted by F the matrix of new infectious terms and V the matrix for the remaining transition terms, in relation to the model are given by:

$$f = \begin{bmatrix} \beta_h \sigma_f \frac{I_f}{N_h} S_h \\ 0 \\ 0 \\ \sigma_f \beta_f (\frac{I_{hs}+I_{hc}}{N_h}) S_f \\ 0 \end{bmatrix}, \; v = \begin{bmatrix} (\delta_1 + \mu_h + \psi) E_h \\ -\delta_1 E_h + (\delta_2 + \psi + \mu_h) I_{hs} \\ -\delta_2 I_{hs} + (\psi + \mu_h) I_{hc} \\ (\tau + \mu_f) E_f \\ -\tau E_f + \mu_f I_f \end{bmatrix}, \tag{3.6}$$

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \beta_h \sigma_f \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\sigma_f \beta_f \mu_h \pi_f}{\mu_f \pi_h} & \frac{\sigma_f \beta_f \mu_h \pi_f}{\mu_f \pi_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \; V = \begin{bmatrix} P_1 & 0 & 0 & 0 & 0 \\ -\delta_1 & P_2 & 0 & 0 & 0 \\ 0 & -\delta_2 & P_3 & 0 & 0 \\ 0 & 0 & 0 & P_4 & 0 \\ 0 & 0 & 0 & -\tau & \mu_f \end{bmatrix}, \tag{3.7}$$

$$V^{-1} = \begin{bmatrix} {P_1}^{-1} & 0 & 0 & 0 & 0 \\ \frac{\delta_1}{P_2 P_1} & {P_2}^{-1} & 0 & 0 & 0 \\ \frac{\delta_2 \delta_1}{P_2 P_1 P_3} & \frac{\delta_2}{P_2 P_3} & {P_3}^{-1} & 0 & 0 \\ 0 & 0 & 0 & {P_4}^{-1} & 0 \\ 0 & 0 & 0 & \frac{\tau}{P_4 \mu_f} & {\mu_f}^{-1} \end{bmatrix}, \tag{3.8}$$

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_h \sigma_f \tau}{P_4 \mu_f} & \frac{\beta_h \sigma_f}{\mu_f} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_f \sigma_f \mu_h \Pi_f \delta_1}{\mu_f \Pi_h P_1 P_2} + \frac{\beta_f \sigma_f \mu_h \Pi_f \delta_2 \delta_1}{\mu_f \Pi_h P_1 P_2 P_3} & \frac{\beta_f \sigma_f \mu_h \Pi_f}{\mu_f \Pi_h P_2} + \frac{\beta_f \sigma_f \mu_h \Pi_f \delta_2}{\mu_f \Pi_h P_2 P_3} & \frac{\beta_f \sigma_f \mu_h \Pi_f}{\mu_f \Pi_h P_3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \tag{3.9}$$

where

$$P_1 = \delta_1 + \mu_h + \psi, P_2 = \delta_2 + \mu_h + \psi,$$

$$P_3 = \mu_h + \psi, P_4 = \tau + \mu_f, P_5 = \mu_h + \varphi.$$

It can be seen that the basic reproduction number of the model is $R_0 = \rho(FV^{-1})$, ρ stands for the spectral radius of the matrix, FV^{-1}. And therefore the basic reproduction number is given by:

$$R_0 = \frac{\sqrt{P_2P_1P_3P_4\Pi_h\tau\,\Pi_f\beta_f\beta_h\delta_1\mu_h\,(P_3+\delta_2)}\sigma_f}{P_2P_1P_3P_4\Pi_h\mu_f}. \tag{3.10}$$

The following result is then established by applying theorem (2) from (Van den Driessche and Wanmough 2000) (i.e if the basic reproduction number R_0 is less than unity, it means the DFE is locally asymptotically stable and the trachoma eliminated can be possible from the population). We summarized the following:

Theorem 3.4.1. *The DFE of the trachoma is locally asymptotically stable "LAS" if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

3.4.3 Endemic Equilibrium Point

Suppose $\varepsilon^* = \left(S_h^*, E_h^*, I_{hs}^*, I_{hc}^*, R_h^*, S_f^*, E_f^*, I_f^*\right)$ is the steady state of $\varepsilon_0 = \left(S_h, E_h, I_{hs}, I_{hc}, R_h, S_f, E_f, I_f\right)$. The endemic equilibrium point of Eq. (3.1) in terms of the forces of infection, $\lambda_h^* = (\beta_h\sigma_f\frac{I_f^*}{N_h^*})$ and $\lambda_f^* = \sigma_f\beta_f(\frac{I_{hs}^*+I_{hc}^*}{N_h^*})$ is obtained by setting the Eq. (3.1) to zero and solve in terms of the forces of infection λ_h^* and λ_f^* as follows:

$$S_h^* = \frac{\Pi_h P_5P_3P_2P_1}{\left(\lambda_h^*+\mu_h\right)P_1P_2P_3P_5 - K\varphi\,\psi\,\lambda_h^*},$$

$$E_h^* = \frac{\lambda_h^*\Pi_h P_5P_3P_2}{\left(\lambda_h^*+\mu_h\right)P_1P_2P_3P_5 - K\phi\,\psi\,\lambda_h^*},$$

$$I_{hs}^* = \frac{\lambda_h^*\delta_1\Pi_h P_5P_3}{\left(\lambda_h^*+\mu_h\right)P_1P_2P_3P_5 - K\varphi\,\psi\,\lambda_h^*},$$

$$I_{hc}^* = \frac{\lambda_h^*\delta_2\delta_1\Pi_h P_5}{\left(\lambda_h^*+\mu_h\right)P_1P_2P_3P_5 - K\phi\,\psi\,\lambda_h^*},$$

$$R_h^* = \frac{\lambda_h^*\Pi_h\psi K}{\left(\lambda_h^*+\mu_h\right)P_1P_2P_3P_5 - K\phi\,\psi\,\lambda_h^*},$$

$$S_f^* = \frac{\Pi_f}{\left(\lambda_f^* + \mu_f\right)},$$
$$E_f^* = \frac{\lambda_f^* \Pi_f}{\left(\lambda_f^* + \mu_f\right) P_4},$$
$$I_f^* = \frac{\tau \lambda_f^* \Pi_f}{\mu_f \left(\lambda_f^* + \mu_f\right) P_4},$$

where

$$P_1 = \delta_1 + \mu_h + \psi, P_2 = \delta_2 + \mu_h + \psi, P_3 = \mu_h + \psi,$$

$$P_4 = \tau + \mu_f, P_5 = \mu_h + \varphi, K = (P_2 P_3 + P_3 \delta_1 + \delta_1 \delta_2).$$

By substituting putting the expression for I_f^* into λ_h^* and that of $(I_{hs}^* + I_{hc}^*)$ into λ_f^* , we obtain

$$\lambda_h^* = \frac{\beta_h \sigma_f \mu_h \tau \lambda_f \Pi_f}{\Pi_h \mu_f P_4 \left(\lambda_f + \mu_f\right)}, \tag{3.11}$$

and

$$\lambda_f^* = \frac{\beta_h \sigma_f \mu_h \lambda_h \delta_1 P_5 P_6}{(\lambda_h + \mu_h) P_1 P_2 P_3 P_5 - K \phi \psi \lambda_h}, \tag{3.12}$$

where

$$P_6 = P_3 + \delta_2.$$

Substituting Eq. 3.11 into Eq. 3.12, we can easily see that the equilibria of the model satisfy the following linear equation in terms of (λ_h^*) :

$$a_0(\lambda_h^*) + b_0 = 0, \tag{3.13}$$

where $a_0 = \mu_f P_4 \Pi_h P_6 \beta_h \delta_1 \mu_h \sigma_f P_5 + \mu_f^2 P_4 \Pi_h P_1 P_2 P_3 P_5 - \mu_f^2 P_4 \Pi_h \phi \psi K$ and $b_0 = \omega(R_0^2 - 1)$, $\omega = \frac{p_5 \mu_h}{\Pi_f P_6 \delta_1 \tau \mu_h \sigma_f^2 \beta_f^2}$. It is clear that a_0 is positive, and $b_0 > 0$ provided $R_0 > 1$ .Hence there exist an endemic equilibrium when $R_0 > 1$. To summarize the above results we have the following lemma:

Lemma 3.4.2. *The Eq.* (3.1) *usually has a disease-free equilibrium and a unique endemic equilibrium whenever* $R_0 > 1$.

3.4.4 Global Stability of the Endemic Equilibrium (ε^*)

Theorem 3.4.3. *When $R_0 > 1$ the endemic equilibrium (ε^*) is "GAS" globally asymptotically stable and not stable if $R_0 < 1$.*

Proof. Let us define a lyapunov function of the form

$$\begin{aligned} U =& c_1\left[S_h - S_h^* - S_h^* ln\left(\frac{S_h}{S_h^*}\right)\right] + c_1\left[E_h - E_h^* - E_h^* ln\left(\frac{E_h}{E_h^*}\right)\right] + c_2\left[I_{hs} - I_{hs}^* - I_{hs}^* ln\left(\frac{I_{hs}}{I_{hs}^*}\right)\right] \\ &+ c_3\left[I_{hc} - I_{hc}^* - I_{hc}^* ln\left(\frac{I_{hc}}{I_{hc}^*}\right)\right] + c_4\left[S_f - S_f^* - S_f^* ln\left(\frac{S_f}{S_f^*}\right)\right] + c_4\left[E_f - E_f^* - E_f^* ln\left(\frac{E_f}{E_f^*}\right)\right] \\ &+ c_5\left[I_f - I_f^* - I_f^* ln\left(\frac{I_f}{I_f^*}\right)\right], \end{aligned} \tag{3.14}$$

where c_1, c_2, c_3, c_4, c_5 are some determining constants. Then computing the derivative of U along the solutions of the Eq.3.15, we get

$$\begin{aligned} \dot{U} =& c_1\left(1 - \frac{S_h^*}{S_h}\right)\dot{S_h} + c_1\left(1 - \frac{E_h^*}{E_h}\right)\dot{E_h} + c_2\left(1 - \frac{I_{hs}^*}{I_{hs}}\right)\dot{I_{hs}} + c_3\left(1 - \frac{I_{hc}^*}{I_{hc}}\right)\dot{I_{hc}} + c_4\left(1 - \frac{S_f^*}{S_f}\right)\dot{S_f} \\ &+ c_4\left(1 - \frac{E_f^*}{E_f}\right)\dot{E_f} + c_5\left(1 - \frac{I_f^*}{I_f}\right)\dot{I_f}, \end{aligned} \tag{3.15}$$

from (3.15), we have that

$$\begin{aligned} c_1\left(1 - \frac{S_h^*}{S_h}\right)\dot{S_h} =& c_1\left(\lambda_h^* S_h^* + \mu_h S_h^* - \lambda_h S_h - \mu_h S_h\right) \\ =& c_1\left(1 - \frac{S_h^*}{S_h}\right)\left[\left(\lambda_h^* S_h^* - \lambda_h S_h\right) - \left(\mu_h S_h - \mu_h S_h^*\right)\right] \\ =& c_1\left[\lambda_h^* S_h^*\left(1 - \frac{S_h^*}{S_h}\right)\left(1 - \frac{\lambda_h S_h}{\lambda_h^* S_h^*}\right) - \frac{\mu_h}{S_h}\left(S_h - S_h^*\right)^2\right] \\ \leq& c_1\left[\lambda_h^* S_h^*\left(1 - \frac{S_h^*}{S_h}\right)\left(1 - \frac{\lambda_h S_h}{\lambda_h^* S_h^*}\right)\right] \end{aligned} \tag{3.16}$$

and

$$\begin{aligned} c_1\left(1 - \frac{E_h^*}{E_h}\right)\dot{E_h} =& c_1\left(1 - \frac{E_h^*}{E_h}\right)\left(\lambda_h S_h - \lambda_h^* S_h^* \frac{E_h}{E_h^*}\right) \\ =& c_1\lambda_h^* S_h^*\left(1 - \frac{E_h^*}{E_h}\right)\left(\frac{\lambda_h S_h}{\lambda_h^* S_h^*} - \frac{E_h}{E_h^*}\right) \end{aligned} \tag{3.17}$$

Likewise

$$\begin{aligned} c_2\left(1 - \frac{I_{hs}^*}{I_{hs}}\right)\dot{I_{hs}} =& c_2\left(1 - \frac{I_{hs}^*}{I_{hs}}\right)\left(\delta_1 E_h - \delta_1 E_h^* \frac{I_{hs}}{I_{hs}^*}\right) \\ =& c_2\delta_1 E_h^*\left(1 - \frac{I_{hs}^*}{I_{hs}}\right)\left(\frac{E_h}{E_h^*} - \frac{I_{hs}}{I_{hs}^*}\right) \end{aligned} \tag{3.18}$$

and

$$\begin{aligned} c_3\left(1-\frac{I_{hc}^*}{I_{hc}}\right)\dot{I_{hc}} =& c_3\left(1-\frac{I_{hc}^*}{I_{hc}}\right)\left(\delta_2 I_{hs}-\delta_2 I_{hs}^*\frac{I_{hc}}{I_{hc}^*}\right) \\ =& c_3\delta_2 I_{hs}^*\left(1-\frac{I_{hc}^*}{I_{hc}}\right)\left(\frac{I_{hs}}{I_{hs}^*}-\frac{I_{hc}}{I_{hc}^*}\right) \end{aligned} \tag{3.19}$$

In similar approach to the equations (3.16-3.19) above we get

$$\begin{aligned} c_4\left(1-\frac{S_f^*}{S_f}\right)\dot{S_f} =& c_1\left(\lambda_f^* S_f^* + \mu_f S_f^* - \lambda_f S_f - \mu_f S_f\right) \\ =& c_4\left(1-\frac{S_f^*}{S_f}\right)\left[(\lambda_f^* S_f^* - \lambda_f S_f) - (\mu_f S_f - \mu_f S_f^*)\right] \\ =& c_4\left[\lambda_f^* S_f^*\left(1-\frac{S_f^*}{S_f}\right)\left(1-\frac{\lambda_f S_f}{\lambda_f^* S_f^*}\right) - \frac{\mu_f}{S_f}\left(S_f - S_f^*\right)^2\right] \\ \leq& c_4\lambda_f^* S_f^*\left[\left(1-\frac{S_f^*}{S_f}\right)\left(1-\frac{\lambda_f S_f}{\lambda_f^* S_f^*}\right)\right] \end{aligned} \tag{3.20}$$

and

$$\begin{aligned} c_4\left(1-\frac{E_f^*}{E_f}\right)\dot{E_f} =& c_4\left(1-\frac{E_f^*}{E_f}\right)\left(\lambda_f S_f - \lambda_f^* S_f^*\frac{E_f}{E_f^*}\right) \\ =& c_4\lambda_f^* S_f^*\left(1-\frac{E_f^*}{E_f}\right)\left(\frac{\lambda_f S_f}{\lambda_f^* S_f^*}-\frac{E_f}{E_f^*}\right) \end{aligned} \tag{3.21}$$

also

$$\begin{aligned} c_5\left(1-\frac{I_f^*}{I_f}\right)\dot{I_f} =& c_5\left(1-\frac{I_f^*}{I_f}\right)\left(\tau E_f - \tau E_f^*\frac{I_f}{I_f^*}\right) \\ =& c_5\tau E_f^*\left(1-\frac{I_f^*}{I_f}\right)\left(\frac{E_f}{E_f^*}-\frac{I_f}{I_f^*}\right) \end{aligned} \tag{3.22}$$

From equations (3.16-3.22) we obtain

$$\begin{aligned} \dot{U} \leq& c_1\lambda_h^* S_h^*\left(1-\frac{S_h^*}{S_h}\right)\left(1-\frac{\lambda_h S_h}{\lambda_h^* S_h^*}\right) + c_1\lambda_h^* S_h^*\left(1-\frac{E_h^*}{E_h}\right)\left(\frac{\lambda_h S_h}{\lambda_h^* S_h^*}-\frac{E_h}{E_h^*}\right) \\ &+ c_2\delta_1 E_h^*\left(1-\frac{I_{hs}^*}{I_{hs}}\right)\left(\frac{E_h}{E_h^*}-\frac{I_{hs}}{I_{hs}^*}\right) + c_3\delta_2 E_h^*\left(1-\frac{I_{hc}^*}{I_{hc}}\right)\left(\frac{I_{hs}}{I_{hs}^*}-\frac{I_{hc}}{I_{hc}^*}\right) \\ &+ c_4\lambda_f^* S_f^*\left[\left(1-\frac{S_f^*}{S_f}\right)\left(1-\frac{\lambda_f S_f}{\lambda_f^* S_f^*}\right)\right] + c_4\lambda_f^* S_f^*\left(1-\frac{E_f^*}{E_f}\right)\left(\frac{\lambda_f S_f}{\lambda_f^* S_f^*}-\frac{E_f}{E_f^*}\right) \\ &+ c_5\tau E_f^*\left(1-\frac{I_f^*}{I_f}\right)\left(\frac{E_f}{E_f^*}-\frac{I_f}{I_f^*}\right) \\ =& c_1\lambda_h^* S_h^*\left[2-\frac{S_h^*}{S_h}-\frac{E_h}{E_h^*}-\frac{E_h^*\lambda_h S_h}{E_h\lambda_h^* S_h^*}+\frac{S_h^*\lambda_h S_h}{S_h\lambda_h^* S_h^*}\right] + c_2\delta_1 E_h^*\left[\frac{E_h}{E_h^*}-\frac{I_{hs}^* E_h}{I_{hs}E_h^*}-\frac{I_{hs}}{I_{hs}^*}+1\right] \\ &+ c_3\delta_2 I_{hs}^*\left[\frac{I_{hs}}{I_{hs}^*}-\frac{I_{hc}}{I_{hc}^*}-\frac{I_{hc}^* I_{hs}}{I_{hc}I_{hs}^*}+1\right] + c_4\lambda_f^* S_f^*\left[2-\frac{S_f^*}{S_f}-\frac{E_f}{E_f^*}-\frac{E_f^*\lambda_f S_f}{E_f\lambda_f^* S_f^*}+\frac{S_f^*\lambda_f S_f}{S_f\lambda_f^* S_f^*}\right] \\ &+ c_5\tau E_f^*\left[\frac{E_f}{E_f^*}-\frac{I_f}{I_f^*}-\frac{E_f I_f^*}{E_f^* I_f}+1\right] \end{aligned}$$

(3.23)

Considering the fact that $x - 1 > ln(x)$ whenever $x > 0$ and $x - 1 = ln(x)$ for $x = 1$. from (3.23) we obtained

$$\begin{aligned}\left[2 - \frac{S_h^*}{S_h} - \frac{E_h}{E_h^*} - \frac{E_h^*\lambda_h S_h}{E_h\lambda_h^* S_h^*} + \frac{S_h^*\lambda_h S_h}{S_h\lambda_h^* S_h^*}\right] &= -\left(1 - \frac{\lambda_h S_h}{\lambda_h^* S_h^*}\right)\left(1 - \frac{I_{hc}\lambda_h^* s_h^*}{I_{hc}^*\lambda_h S_h}\right) \\ &\quad + 3 - \frac{S_h^*}{S_h} - \frac{E_h}{E_h^*} - \frac{\lambda_h S_h}{\lambda_h^* S_h} - \frac{I_{hc}\lambda_h^* S_h^*}{I_{hc}^*\lambda_h S_h} + \frac{I_{hc}}{I_{hc}^*} \\ &\leq -\left(\frac{S_h^*}{S_h} - 1\right) - \left(\frac{I_{hc}\lambda_h^* S_h^*}{I_{hc}^*\lambda_h S_h} - 1\right) - \left(\frac{E_h^*\lambda_h S_h}{E_h\lambda_h^* S_h^*} - 1\right) - \frac{E_h}{E_h^*} + \frac{I_{hc}}{I_{hc}^*} \\ &= -ln\left[\frac{S_h^*}{S_h}\frac{I_{hc}\lambda_h^* s_h^*}{I_{hc}^*\lambda_h S_h}\frac{E_h^*\lambda_h S_h}{E_h\lambda_h^* S_h^*}\right] - \frac{E_h}{E_h^*} + \frac{I_{hc}}{I_{hc}^*} \\ &\leq \frac{I_{hc}}{I_{hc}^*} - ln(\frac{I_{hc}}{I_{hc}^*}) - \frac{E_h}{E_h^*} + ln(\frac{E_h}{E_h^*})\end{aligned} \tag{3.24}$$

and

$$\begin{aligned}\left[\frac{E_h}{E_h^*} - \frac{I_{hs}^* E_h}{I_{hs} E_h^*} - \frac{I_{hs}}{I_{hs}^*} + 1\right] &= -\left(\frac{I_{hs}^* E_h}{I_{hs} E_h^*} - 1\right) + \frac{E_h}{E_h^*} - \frac{I_{hs}}{I_{hs}^*} \leq -ln\left(\frac{I_{hs}^* E_h}{E_h^* I_{hs}}\right) + \frac{E_h}{E_h^*} - \frac{I_{hs}}{I_{hs}^*} \\ &= \left(\frac{E_h}{E_h^*} - ln(\frac{E_h}{E_h^*}) - \frac{I_{hs}}{I_{hs}^*} + ln(\frac{I_{hs}}{I_{hs}^*})\right)\end{aligned} \tag{3.25}$$

similarly

$$\begin{aligned}\left[\frac{I_{hs}}{I_{hs}^*} - \frac{I_{hc}}{I_{hc}^*} - \frac{I_{hc}^* I_{hs}}{I_{hc} I_{hs}^*} + 1\right] &= -\left(\frac{I_{hc}^* I_{hs}}{I_{hc} I_{hs}^*} - 1\right) + \frac{I_{hs}}{I_{hs}^*} - \frac{I_{hc}}{I_{hc}^*} \leq -ln\left(\frac{I_{hc}^* I_{hs}}{I_{hs}^* I_{hc}}\right) + \frac{I_{hs}}{I_{hs}^*} - \frac{I_{hc}}{I_{hc}^*} \\ &= \left(\frac{I_{hs}}{I_{hs}^*} - ln(\frac{I_{hs}}{I_{hs}^*}) - \frac{I_{hc}}{I_{hc}^*} + ln(\frac{I_{hc}}{I_{hc}^*})\right)\end{aligned} \tag{3.26}$$

we also compute

$$\begin{aligned}\left[2 - \frac{S_f^*}{S_f} - \frac{E_f}{E_f^*} - \frac{E_f^*\lambda_f S_f}{E_f\lambda_f^* S_f^*} + \frac{S_f^*\lambda_f S_f}{S_f\lambda_f^* S_f^*}\right] &= -\left(1 - \frac{\lambda_f S_f}{\lambda_f^* S_f^*}\right)\left(1 - \frac{I_f\lambda_f^* s_f^*}{I_f^*\lambda_f S_f}\right) \\ &\quad + 3 - \frac{S_f^*}{S_f} - \frac{E_f}{E_f^*} - \frac{\lambda_f S_f}{\lambda_f^* S_f} - \frac{I_f\lambda_f^* S_f^*}{I_f^*\lambda_f S_f} + \frac{I_f}{I_f^*} \\ &\leq -\left(\frac{S_f^*}{S_f} - 1\right) - \left(\frac{I_f\lambda_f^* S_f^*}{I_f^*\lambda_f S_f} - 1\right) - \left(\frac{E_f^*\lambda_f S_f}{E_f\lambda_f^* S_h^*} - 1\right) - \frac{E_h}{E_h^*} + \frac{I_{hc}}{I_{hc}^*} \\ &= -ln\left[\frac{S_f^*}{S_f}\frac{I_f\lambda_f^* s_f^*}{I_f^*\lambda_f S_f}\frac{E_f^*\lambda_f S_f}{E_f\lambda_f^* S_f^*}\right] - \frac{E_f}{E_f^*} + \frac{I_f}{I_f^*} \\ &\leq \frac{I_f}{I_f^*} - ln(\frac{I_f}{I_f^*}) - \frac{E_f}{E_f^*} + ln(\frac{E_f}{E_f^*})\end{aligned} \tag{3.27}$$

and

$$\left[\frac{E_f}{E_f^*}-\frac{I_f^*E_f}{I_fE_f^*}-\frac{I_f}{I_f^*}+1\right]=-\left(\frac{I_f^*E_f}{I_fE_f^*}-1\right)+\frac{E_f}{E_f^*}-\frac{I_f}{I_f^*}\leq -ln\left(\frac{I_f^*E_f}{E_f^*I_f}\right)+\frac{E_f}{E_f^*}-\frac{I_f}{I_f^*}$$
$$=\left(\frac{E_f}{E_f^*}-ln(\frac{E_f}{E_f^*})-\frac{I_f}{I_f^*}+ln(\frac{I_f}{I_f^*})\right) \tag{3.28}$$

Putting equations (3.24-3.28) into (3.23) we've

$$\begin{aligned}\dot{U}\leq\ & c_1\lambda_h^*S_h^*\left[\frac{I_{hc}}{I_{hc}^*}-ln(\frac{I_{hc}}{I_{hc}^*})-\frac{E_h}{E_h^*}+ln(\frac{E_h}{E_h^*})\right]+c_2\delta_1E_h^*\left[\frac{E_h}{E_h^*}-ln(\frac{E_h}{E_h^*})-\frac{I_{hs}}{I_{hs}^*}+ln(\frac{I_{hs}}{I_{hs}^*})\right]\\ &+c_3\delta_2I_{hs}^*\left[\frac{I_{hs}}{I_{hs}^*}-ln(\frac{I_{hs}}{I_{hs}^*})-\frac{I_{hc}}{I_{hc}^*}+ln(\frac{I_{hc}}{I_{hc}^*})\right]+c_4\lambda_f^*S_f^*\left[\frac{I_f}{I_f^*}-ln(\frac{I_f}{I_f^*})-\frac{E_f}{E_f^*}+ln(\frac{E_f}{E_f^*})\right]\\ &+c_5\tau E_f^*\left[\frac{E_f}{E_f^*}-ln(\frac{E_f}{E_f^*})-\frac{I_f}{I_f^*}+ln(\frac{I_f}{I_f^*})\right]\end{aligned} \tag{3.29}$$

By taking the constants $C_1=\delta_2I_{hs}^*$, $C_2=\frac{\delta_2\lambda_h^*S_h^*I_{hs}^*}{\delta_1E_h^*}$, $C_3=\lambda_h^*S_h^*$, $C_4=\tau E_f^*$, and $C_5=\lambda_f^*S_f^*$ and further simplifying (3.29) we can simply obtain $\dot{U}\leq 0$.while strictly $\dot{U}=0$ is true only when $S_h=S_h^*, E_h=E_h^*, I_{hs}=I_{hs}^*, I_{hc}=I_{hc}^*$ and $S_f=S_f^*, E_f=E_f^*, I_f=I_f^*$. then the only invariant set of the model (3.1) is the endemic equilibrium point ε^*. Hence Applying Lasalle invarinace principle (Lasalle et al 1976), the endemic equilibrium ε^* of the trachoma model is globally asymptotically stable "GAS". □

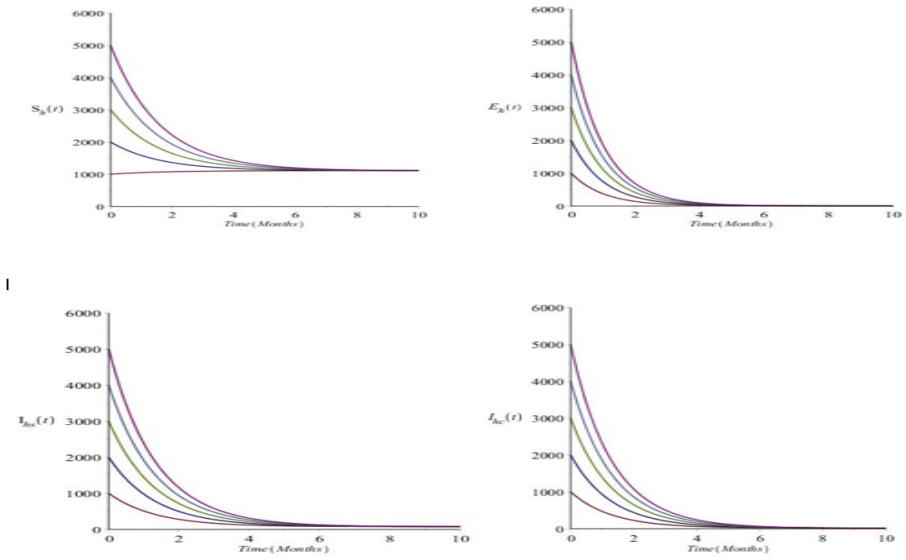


Figure 3.2. The time series plot of model (3.1)
considering various initial conditions(represented by different colours). Taking the parameter values as presented in table (3.1) so that $R_0 < 1$; $S_h(t)$ the number of susceptible human, $E_h(t)$ the number of exposed human with no clinical symptom of trachoma; $I_{hs}(t)$ group 1 of infected human with active trachoma(either TF/TI or trachomatous scare), $I_{hc}(t)$ Group two of the infected human with the severe symptoms(Trachomatous trichiasis and incomplete corneal opacity).

3.5 PARAMETER ESTIMATION

This section discusses how to fit the parameters in the proposed Trachoma model using real-life trachoma cases from Northern Nigeria. The epidemic cases were reported between May and July of 2013, (Caleb et al 2017). At the time when this research is prepared, The population of Northern Nigeria is estimated to be $N_h(0) = 90.3$M for initial conditions, with an initial exposed population of $E_h(0) = 300000$ and Using the relation $N_h(0) = S_h(0) + E_h(0) + I_{hs}(0) + I_{hc}(0) + R_h$, we can estimate the rest of the initial values for the state variables $N_f(0) = S_f(0) + E_f(0) + I_f(0)$. In this case, $S_h(0) = 6140400$, $I_{hs}(0) = 410$, $I_{hc}(0) = 270$, and $R_h(0) = 150$ were obtained, with $S_f(0) = 341000$ and $I_f(0) = 3000$. There are 14 biological parameters that have been predicted using the least-square fitting method, coming up with the best fit of the Trachoma model's solution to actual epidemic cases.

The best values of the biological parameters are obtained by reducing the average absolute relative error between the actual Trachoma cases and the model solution. With a value of $9.8748e-02$, the objective function produces a relatively small error. Figure 3.3 depicts actual Trachoma cases as solid circles, while the best-fitting curve of the model is depicted as a solid line. Table 3.2 lists the biological parameters used in the model, along with their best approximate values obtained using the least-squares method. For the real Trachoma cases in Northern Nigeria from May to July 2013, these parameters finally provided the value of the basic reproduction number equal to $R_0 = 1.66$.

Table 3.2. Baseline values of the parameters used in the trachoma model (3.1)

Parameter	Value	Units/Remarks	Sources
$N_h(0)$	90.3*million*	Constant	(Central Intelligence 2016)
$N_f(0)$	11 million	Constant	(Animal Diversity 2016)
$S_h(0)$	$0.68 \times N(0)$	Constant	Assumed
$S_f(0)$	$0.31 \times N(0)$	Constant	Assumed
β_f	0.07258	day^{-1}	Assumed
τ	2.213	day^{-1}	(Omondi et al2016)
β_h	0.08353	day^{-1}	Estimated
φ	0.501	day^{-1}	(Pinsent et al 2018)
Π_h	24.9991	day^{-1}	Fitted
Π_f	1.5×10^6	day^{-1}	Fitted
μ_h	0.0014	day^{-1}	Fitted
μ_f	1.354	day^{-1}	(Animal Diversity 2016)
δ_1	0.01212	day^{-1}	Fitted
δ_2	0.012003	day^{-1}	Fitted
σ_f	0.2903	day^{-1}	Fitted
ψ_1	0.3010	day^{-1}	Fitted
ψ_2	0.1121	day^{-1}	Fitted
ψ_3	0.1428	day^{-1}	Fitted

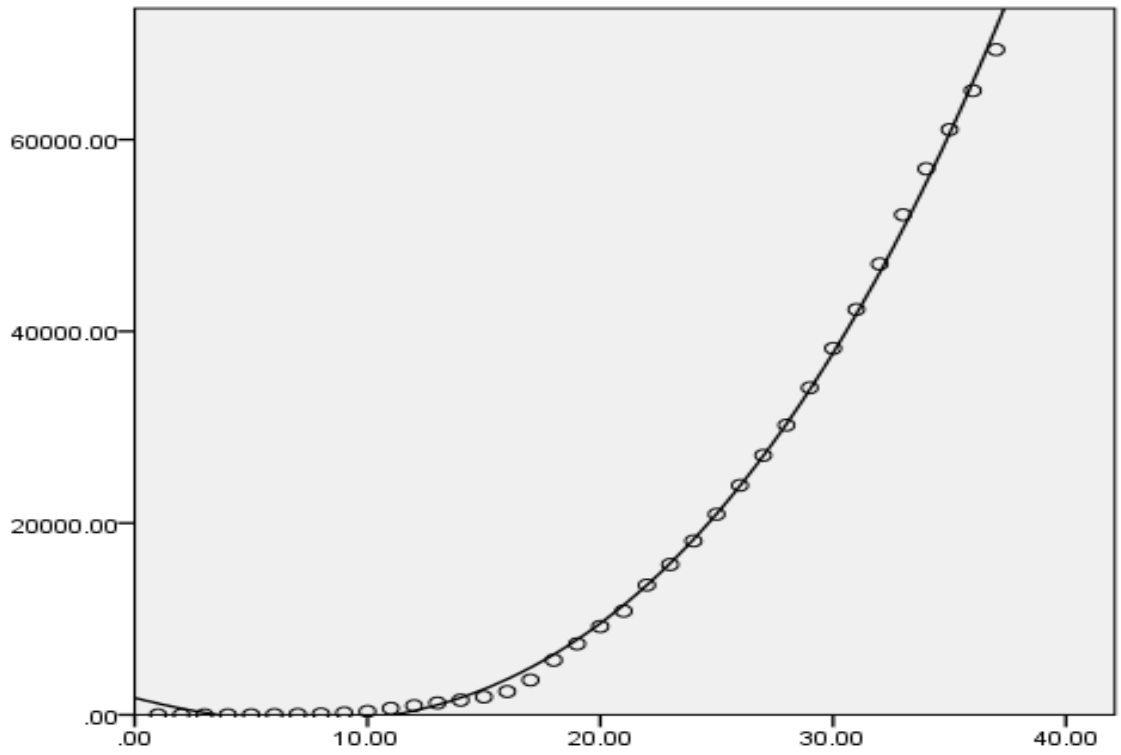


Figure 3.3. Data fitting for the real Trachoma cases in Northern Nigeria.

3.6 THE NORMALIZED SENSITIVITY ANALYSIS

In this section we employed the method of local sensitivity analysis to highlight the sensitivity of the basic reproduction number R_0 to some key associated parameters for a trachoma model earlier developed and rigorously analysed in sections above. The basic reproduction number is obtained and describe as a parameter dependent output of the model and the severity indicator of the (chlamydia trachomatis/ocular chlamydia) of which lowering the number below a critical figure (i.e less than unity) is considered as the Major way of curtailing and aborting the spread of the infectious trachoma in a population, and saving individuals from corneal opacifications or even perpetual and irreversible blindness. In this direction, investigating the monotonicity between the model parameters and the basic reproduction number became crucially interested and motivated. The basic properties of the model have been demonstrated earlier in section (3.3), detailing the boundedness, positivity and well-posedness of the solutions. Our main interest here is to understand the sensitivity and impressionability of the basic reproduction number in relation to the parameters used throughout the model.The computation of the expression of basic reproduction number is carried out using the technique of next generation matrix, see(3.6) above, and its denoted by

$$R_0^T = \frac{\sqrt{\Pi_h\left(\mu_h+\psi_3\right)\left(\delta_2+\mu_h+\psi_2\right)\left(\delta_1+\mu_h+\psi_1\right)\left(\mu_h+\tau\right)\tau\,\Pi_f\beta_f\beta_h\delta_1\mu_h\left(\mu_h+\psi_3+\delta_2\right)}\sigma_f}{\Pi_h\left(\mu_h+\psi_3\right)\left(\delta_2+\mu_h+\psi_2\right)\left(\delta_1+\mu_h+\psi_1\right)\left(\mu_h+\tau\right)\mu_f}$$

We will now proceed to computation of the local sensitivity indices of R_0^T proportional the parameters in connection the trachoma model (3.1). Here the series of input parameters relative to R_0^T is

$$\rho = \left\{\Pi_h, \mu_h, \psi_3, \delta_2, \psi_2, \delta_1, \psi_1, \tau, \Pi_f, \beta_f, \beta_h, \sigma_f, \mu_f\right\} \tag{3.30}$$

We then Scrutinize the significance on the normalized forward sensitivity or (Elasticity index) of R_0^T due to parameter discrepancy. Usually if a system is of various parameters, changes in parameters may not equally change the results due to variation in the sensitivity of the parameters, some have high sensitivity while others are slightly sensitive and some are neutrally sensitive (having zero relative sensitivity).The optimization of the output is achieved by determining the sensitivity status of each parameter T(amas et al 1990, Gambhir et al 2013). We denote by $\Gamma_\omega^{R_0^T}$ the normalized local sensitivity index of the output R_0^T with respect to a parameter

(ω), where $\omega \in \rho$, and is defined as

$$\dot{\Upsilon}_{\omega} = \Gamma_{\omega}^{R_0^T} = \frac{\omega}{R_0^T} \times \frac{\partial R_0^T}{\partial \omega} = \frac{\partial ln(R_0^T)}{\partial ln(\omega)} \tag{3.31}$$

$$\Gamma_{\omega}^{R_0^T} = \frac{\partial ln(R_0^T)}{\partial ln(\omega)} \tag{3.32}$$

(Griensven et al 2006, Xiaobo et al 2008, Zhike et al 2011). Using the above definition we compute the following indices for the output R_0^T with respect to every parameter presented in (3.30).

$$\Gamma_{\sigma_f}^{R_0^T} = \frac{\sigma_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \sigma_f} = 1$$

$$\Gamma_{\beta_h}^{R_0^T} = \frac{\beta_h}{R_0^T} \times \frac{\partial R_0^T}{\partial \beta_h} = \frac{1}{2}$$

$$\Gamma_{\beta_f}^{R_0^T} = \frac{\beta_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \beta_f} = \frac{1}{2}$$

$$\Gamma_{\Pi_h}^{R_0^T} = \frac{\Pi_h}{R_0^T} \times \frac{\partial R_0^T}{\partial \Pi_h} = -\frac{1}{2}$$

$$\Gamma_{\Pi_f}^{R_0^T} = \frac{\Pi_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \Pi_f} = \frac{1}{2}$$

$$\Gamma_{\tau}^{R_0^T} = \frac{\tau}{R_0^T} \times \frac{\partial R_0^T}{\partial \tau} = \frac{1}{2}\frac{\mu_h}{\mu_h + \tau}$$

$$\Gamma_{\delta_1}^{R_0^T} = \frac{\delta_1}{R_0^T} \times \frac{\partial R_0^T}{\partial \delta_1} = \frac{1}{2}\frac{\mu_h + \psi_1}{\delta_1 + \mu_h + \psi_1}$$

$$\Gamma_{\delta_2}^{R_0^T} = \frac{\delta_2}{R_0^T} \times \frac{\partial R_0^T}{\partial \delta_2} = \frac{1}{2}\frac{(\psi_2 - \psi_3)\delta_2}{(\delta_2 + \mu_h + \psi_2)(\mu_h + \psi_3 + \delta_2 +)}$$

$$\Gamma_{\psi_1}^{R_0^T} = \frac{\psi_1}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_1} = -\frac{1}{2}\frac{\psi_1}{(\delta_1 + \mu_h + \psi_1)}$$

$$\Gamma_{\psi_2}^{R_0^T} = \frac{\psi_2}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_2} = -\frac{1}{2}\frac{\psi_2}{(\delta_2 + \mu_h + \psi_2)}$$

$$\Gamma_{\psi_3}^{R_0^T} = \frac{\psi_3}{R_0^T} \times \frac{\partial R_0^T}{\partial \psi_3} = -\frac{1}{2}\frac{(\delta_2\psi_3)}{(\mu_h + \psi_3)(\mu_h + \psi_3 + \delta_2)}$$

$$\Gamma_{\mu_f}^{R_0^T} = \frac{\mu_f}{R_0^T} \times \frac{\partial R_0^T}{\partial \mu_f} = -1$$

$$\Gamma_{\sigma_f}^{R_0^T} = 1,\ \Gamma_{\beta_h}^{R_0^T} = \frac{1}{2},\ \Gamma_{\beta_f}^{R_0^T} = \frac{1}{2},\ \Gamma_{\Pi_h}^{R_0^T} = -\frac{1}{2},\ \Gamma_{\Pi_f}^{R_0^T} = \frac{1}{2},\ \Gamma_{\tau}^{R_0^T} = \frac{1}{2}\frac{\mu_h}{\mu_h + \tau},\ \Gamma_{\delta_1}^{R_0^T} = \frac{1}{2}\frac{\mu_h + \psi_1}{\delta_1 + \mu_h + \psi_1},$$

$$\Gamma_{\delta_2}^{R_0^T} = \frac{1}{2}\frac{(\psi_2 - \psi_3)\delta_2}{(\delta_2 + \mu_h + \psi_2)(\mu_h + \psi_3 + \delta_2 +)},\ \Gamma_{\psi_1}^{R_0^T} = -\frac{1}{2}\frac{\psi_1}{(\delta_1 + \mu_h + \psi_1)},\ \Gamma_{\psi_2}^{R_0^T} = -\frac{1}{2}\frac{\psi_2}{(\delta_2 + \mu_h + \psi_2)},$$

$$\Gamma^{R_0^T}_{\psi_3} = -\frac{1}{2}\frac{(\delta_2\psi_3)}{(\mu_h+\psi_3)(\mu_h+\psi_3+\delta_2)} \qquad , \Gamma^{R_0^T}_{\mu_f} = -1$$

$$\Gamma^{R_0^T}_{\mu_h} = \frac{1}{2}\frac{\left(A\psi_3 - B{\mu_h}^2\right){\delta_2}^2 + 2\left(A\psi_3 - B{\mu_h}^2\right)(D)\,\delta_2 + (\mu_h+\psi_3)^2\left(A\psi_2 - B{\mu_h}^2\right)}{(\mu_h+\psi_3)\,(\delta_2+\mu_h+\psi_2)\,(\delta_1+\mu_h+\psi_1)\,(\mu_h+\tau)\,(\mu_h+\psi_3+\delta_2)}$$

where; $A = \tau\delta_1 + \tau\psi_1 - {\mu_h}^2$, $B = \tau + \delta_1 + 2\mu_h + \psi_1$, $D = \frac{1}{2}\psi_3 + \mu_h + \frac{1}{2}\psi_2$

Remark. We can see from the above expressions, the local sensitivity indices reveal the following facts:

$(i)\Gamma^{R_0^T}_{\sigma_f} = 1$

$(ii)0 < \Gamma^{R_0^T}_{\beta_h}, \Gamma^{R_0^T}_{\beta_f}, \Gamma^{R_0^T}_{\pi_f}, \Gamma^{R_0^T}_{\delta_1}, \Gamma^{R_0^T}_{\tau} < 1$

$(iii)\Gamma^{R_0^T}_{\beta_h} = \Gamma^{R_0^T}_{\beta_f} = \Gamma^{R_0^T}_{\pi_f} = \frac{1}{2}$

$(iv) -1 < \Gamma^{R_0^T}_{\pi_h} < \Gamma^{R_0^T}_{\mu_h} < \Gamma^{R_0^T}_{\psi_1} < \Gamma^{R_0^T}_{\psi_2} < \Gamma^{R_0^T}_{\psi_3} < \Gamma^{R_0^T}_{\delta_2} < 0$

$(v)\left|\Gamma^{R_0^T}_{\sigma_f}\right| = \left|\Gamma^{R_0^T}_{\mu_f}\right|, \left|\Gamma^{R_0^T}_{\beta_h}\right| = \left|\Gamma^{R_0^T}_{\beta_f}\right| = \left|\Gamma^{R_0^T}_{\pi_f}\right| = \left|\Gamma^{R_0^T}_{\pi_h}\right|$

$(vi)\left|\Gamma^{R_0^T}_{\tau}\right| < \left|\Gamma^{R_0^T}_{\psi_1}\right|.$

On the basis of the elasticity analysis above, we can easily see that whenever we increase σ_f by 5% while fixing the remaining parameters, this will consequently attract a 5% increase in R_0^T, which apparently shows a strong connection between the basic reproduction number(R_0^T and the individual parameters. Similarly, it reveals that, increasing the transmission rates, β_f, β_h, and flies recruitment rate Π_f each by 10% and keeping the remaining parameters unchanged will equally produce a 5% increment in the basic reproduction number and this will boost the trachoma prevalence in the population. Likewise, raising the values of the parameters τ and δ_1 by 1.0% (one percent) will correspondingly gain less than 1% increment in the basic reproduction number R_0^T. Meanwhile, raising the values of the parameters μ_f and Π_h by 10% each will consequently attract a corresponding 10% and 5% decrements in R_0^T respectively. In a similar manner, whenever we introduce an increments in each of δ_1, ψ_1, ψ_2, ψ_3 and μ_h by a 1.0%, it will provide us a consequential decrease in the basic reproduction number with less than 1.0%. Therefore, later are the most sensitive parameters that should be targeted to attain the goal of Global Trachoma Elimination as a Public Health Problem, as targeted by the World Health Organization (WHO).

It is now quite enough to position our selves to use the generated coefficients of the normalized local sensitivity indices to highlight the relative influences of each of the parameters on R_0^T.

Table 3.3. Normalized Local Sensitivity Indices of the reproduction number for the trachoma model with parameters presented in table 1.

Parameter	**Elasticity Indices Expressions**	**Values of the Elasticity index**
σ_f	1	1.0000
β_h	$\frac{1}{2}$	0.5000
β_f	$\frac{1}{2}$	0.5000
Π_h	$-\frac{1}{2}$	-0.5000
μ_f	-1	-1.0000
Π_f	$\frac{1}{2}$	0.5000
τ	$\frac{1}{2}\frac{\mu_h}{\mu_h+\tau}$	0.1109
δ_1	$\frac{1}{2}\frac{\mu_h+\psi_1}{\delta_1+\mu_h+\psi_1}$	0.4641
δ_2	$-\frac{1}{2}\frac{(\psi_2-\psi_3)\delta_2}{(\delta_2+\mu_h+\psi_2)(\mu_h+\psi_3+\delta_2+)}$	-0.0001
ψ_1	$-\frac{1}{2}\frac{\psi_1}{(\delta_1+\mu_h+\psi_1)}$	-0.14970
ψ_2	$-\frac{1}{2}\frac{\psi_2}{(\delta_2+\mu_h+\psi_2)}$	-0.0658
ψ_3	$-\frac{1}{2}\frac{(\delta_2\psi_3)}{(\mu_h+\psi_3)(\mu_h+\psi_3+\delta_2)}$	-0.0034
μ_h	$\frac{1}{2}\frac{\left(A\psi_3-B{\mu_h}^2\right){\delta_2}^2+2\left(A\psi_3-B{\mu_h}^2\right)(D)\delta_2+(\mu_h+\psi_3)^2\left(A\psi_2-B{\mu_h}^2\right)}{(\mu_h+\psi_3)(\delta_2+\mu_h+\psi_2)(\delta_1+\mu_h+\psi_1)(\mu_h+\tau)(\mu_h+\psi_3+\delta_2)}$	-0.35499

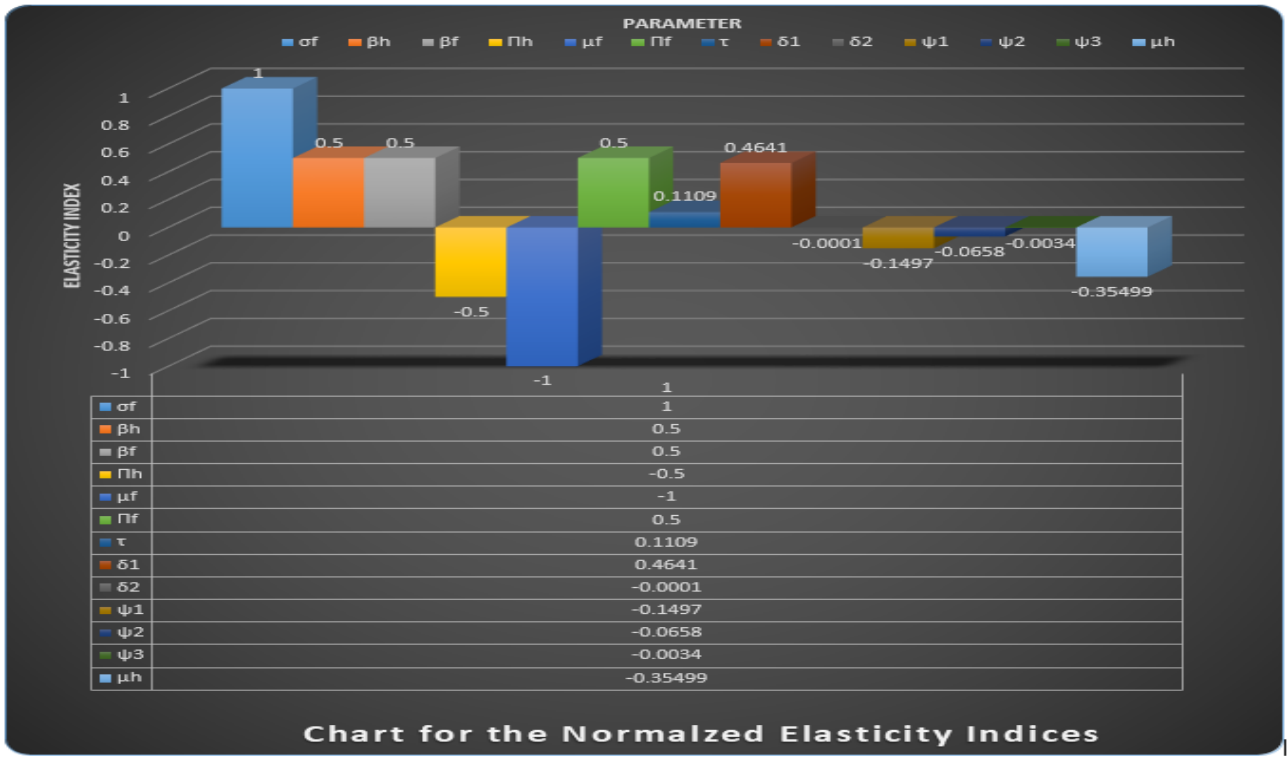


Figure 3.4. The local elasticity indices of R_o with respect to every parameter of the model (3.1) as presented in table 3.3

From figure 3.4 we can interpret the elasticity indices recorded in the chart based on the values presented earlier in table 3.3 It reveals that the most sensitive and influential parameter in changing the size of the basic reproduction number R_0^T is σ_f (the contact rate of the eye-seeking flies or the musca sovens) whose index value is +1.0000 as earlier explained in the computations of the elasticity expressions. Thus the discrepancies with this parameter will give much more variation in the basic reproduction number R_0^T and consequently, the general model output. Particularly, whenever σ_f is variably increase(respectively decrease) by 20%, then the basic reproduction number R_0^T will equally increase(respectively decrease) by 20%. Followed by the parameters (β_h, β_f, and Π_f), the rate of transmission of the chlamydia trachomatis from vector to host, the rate of transmission of chlamydia trachomatis from the eye-seeking flies to human and the rate at which flies are recruited into the vector population respectively. The approximate elasticity sensitivity index is +0.5000, therefore, changing any of them by 20% will correspondingly attract a 10% change in R_0^T. Another set of influential parameters that shows more sensitivity to R_0^T are μ_f, δ_1,and τ, the natural death rate of flies that facilitate the spread of Trachoma with elasticity index (-1.0000), the progression rate at which the exposed individuals proceed to show the clinical symptoms of active trachoma(Trachomatous follicular/Intense TF/TI or even begin to develop scars on the tarsal conjunctiva (TS)) with the index of about (0.4641), and τ the corresponding progression rate at which the exposed eye-seeking flies proceed to infective stage with elasticity index (0.1109) respectively. Among these three, μ_f is the appropriate parameter to concentrate on, so as to achieve (Global Elimination of Blinding Trachoma as a public Health Problem) possibly by the year 2030. This is because reducing the population of flies that transmit the disease through proper environmental sanitation and or insecticide spraying by 20% will certainly bring about a 20% decrease in the size of basic reproduction number (R_0^T) and this will consequently results in curtailing the prevalence of the trachoma in the targeted population by a significant percentage.

The remaining parameters together with their respective indices in a hierarchical curtailing significance include; δ_2, the progression rate of an infected individual from active trachoma stage (i.e Trachomatous Folicular/Intense TF/TI) to the severe stage (i.e Trachomatous Trachiasis or Corneal opacity), ψ_3, the recovery rate from severe

stage of trachoma(TT) through surgery (the S-component of SAFE strategy), ψ_2, the recovery rate of individual from early stages of trachoma(TF/TI or TS) which is achieved by Antibiotic Administration(Azithromycin or Tropical tetracyclin) the A-component of SAFE strategy, ψ_1, the recovery rate of exposed individuals who are yet to show any symptom of trachoma(F-component of SAFE strategy), μ_h, the death rate of human population, and finally, Π_h, the rate at which individuals are recruited into the human population.

Clearly, if a proper concentration is made on the most sensitive parameters for possible adjustment, it will cut down the size of the basic reproduction number (R_0^T), and hence the Global Elimination of Blinding Trachoma as a Public Health Problem will be made achievable as targeted by WHO by the year 2030. Precisely, there should be a means of minimizing the rate of contact between the infected flies and susceptible human(σ_f) as low as possible, the transmission of trachoma from flies to human and vice-vasa should also be terminated. Also the means by which the flies are being recruited should be blocked through proper personal and environmental hygiene; and amplifying the death rate of the eye-seeking flies(the musca-sovens) to curtail or truncate the spread of trachoma in a susceptible population requires a consideration.

CHAPTER 4

NUMERICAL SIMULATIONS AND DISCURSION OF RESULTS

4.1 NUMERICAL SIMULATIONS

Since the basic reproductive number R_0 is the most important quantity to comprehend the extent for the spread of an epidemic. It can be seen in figure (4.7 - 4.10) that displayed the behaviour of the trachoma model when $R_0 > 1$, and the behaviour of the trachoma model when $R_0 < 1$. It shows that the effective use of WHO adopted control scheme (SAFE) plays a significant role toward achieving the WHO targeted Trachoma eradication as challenge in the public health. R_0 has also been investigated by varying different kinds of biological parameters of the proposed Trachoma model. Using mesh plot and the parameter values in Table 3.2, we obtained some numerical results. The result as depicted in Figures 4.11 and 4.12, shows a significant increase with the variation in the transmission rates (β_h,β_f) and that of vector contact rate σ_f while in Figures (4.13 - 4.14), R_0 decreases/increases with the decreasing/increasing value of progression rate from exposed flies to infected flies τ, progression rates from class of exposed individuals to active trachoma class (δ_1), from active trachoma to severe infection class (δ_2) and that of vector contact rate σ_f, and Figures (4.15 - 4.16, shows R_0 increases with decrease of natural death rates of both human and flies populations (μ_h, μ_f), while in figures (4.17 - 4.18) R_0 decreases/increases with the decreasing/increasing value progression rates from class of exposed individuals to active trachoma class (δ_1), from active trachoma to severe infection class (δ_2) and that of vector contact rate σ_f.

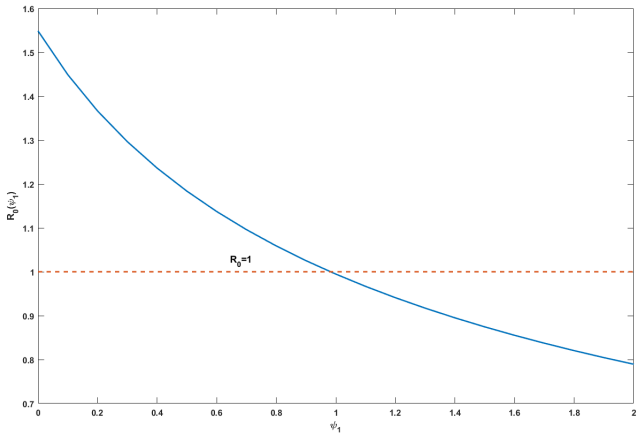


Figure 4.1. Plot for R_0^T against parameter ψ_1, with ψ_1 from 0.01 – 2.00 and other parameters remained unchanged

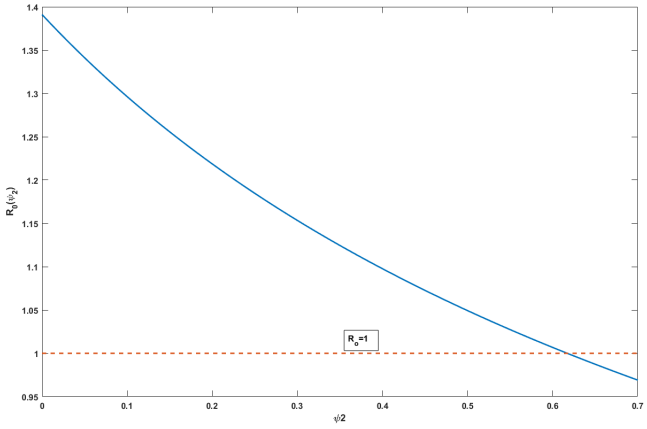


Figure 4.2. Plot for R_0^T against parameter ψ_2, with ψ_2 from 0.100– 0.700 and other parameters remained unchanged

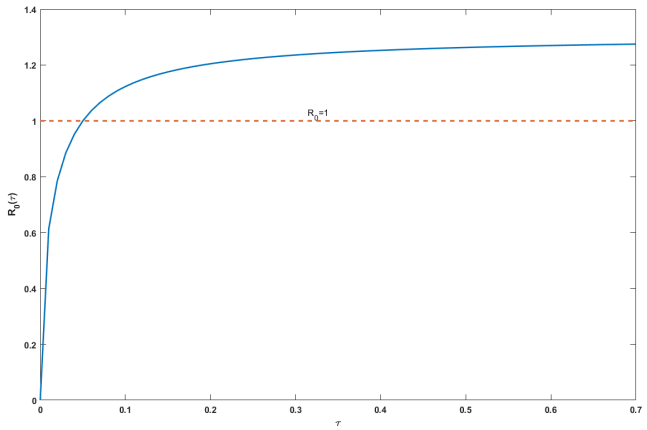


Figure 4.3. plot for R_0^T against parameter τ, with τ from $0.1-0.70$ and other parameters remained unchanged

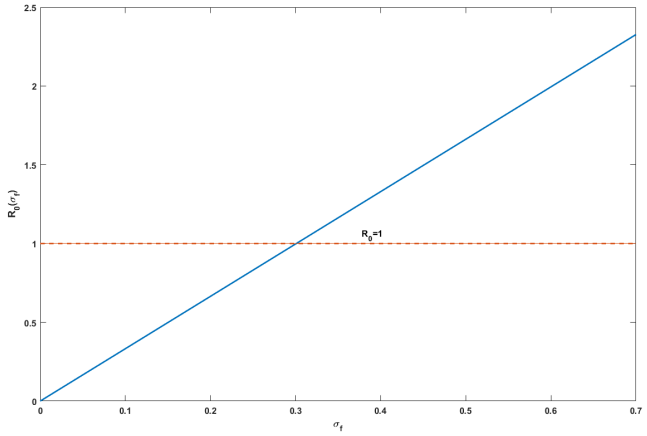


Figure 4.4. Plot for R_0^T against parameter σ_f, with σ_f from $0.100 - 0.700$ and other parameters remained unchanged

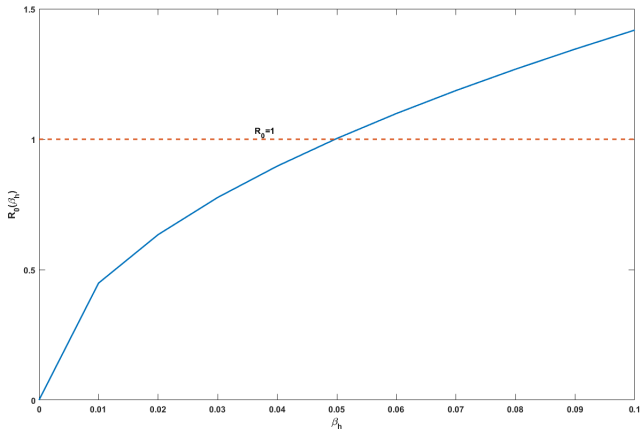


Figure 4.5. Plot for R_0^T against parameter β_h, with β_h from 0.01 – 0.10 and other parameters remained unchanged

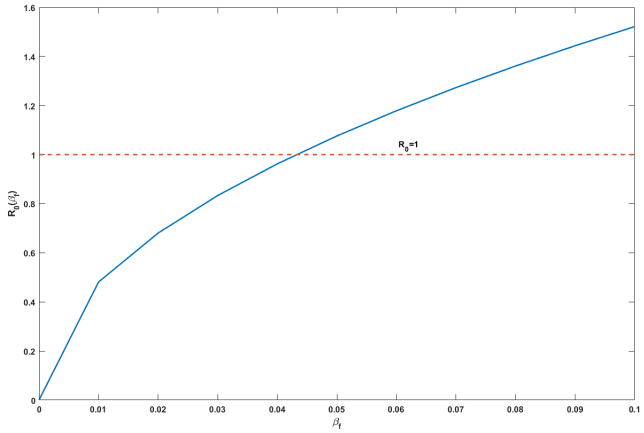


Figure 4.6. Plot for R_0^T against parameter β_f, with β_f from 0.01 – 0.10 and other parameters remained unchanged

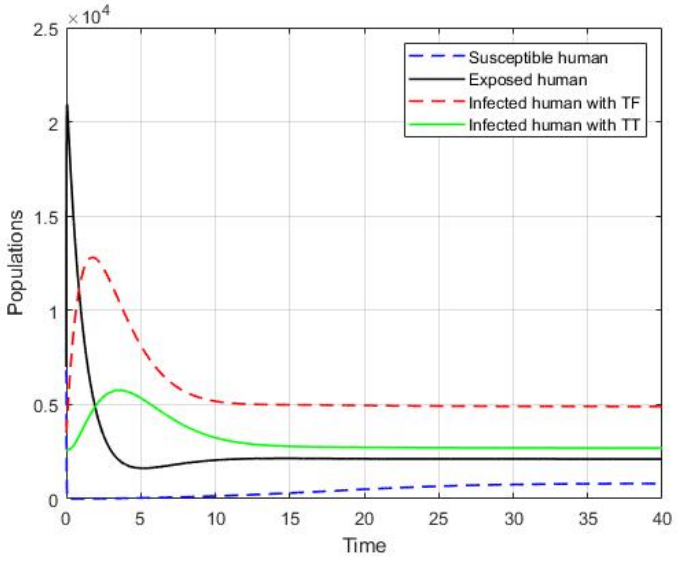


Figure 4.7. Effect of SAFE control strategy on human population. (4.7) displays the behaviour of the trachoma model without control when $R_0 > 1$

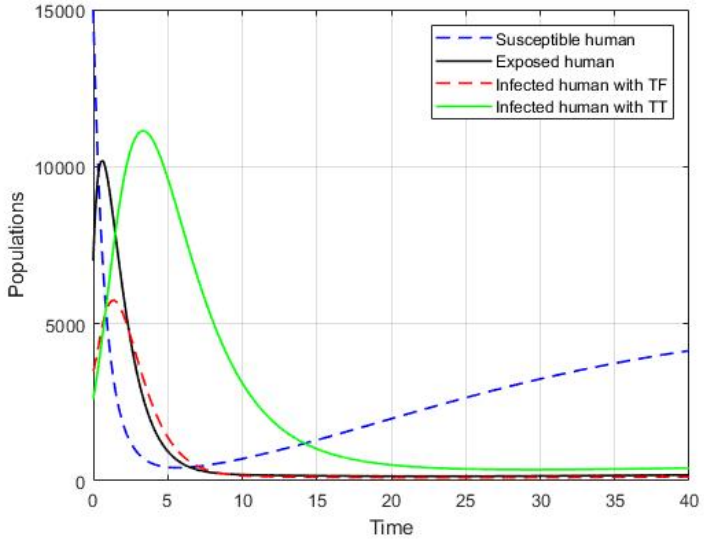


Figure 4.8. Effect of SAFE control strategy on human population. (4.8) displays the behaviour of the trachoma model when $R_0 < 1$

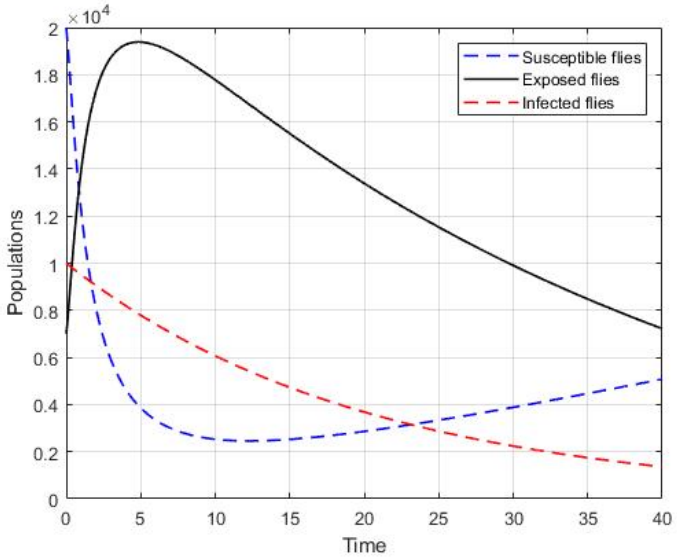


Figure 4.9. Effect of SAFE control strategy on flies population. (4.9) displays the behaviour of the trachoma model when $R_0 > 1$

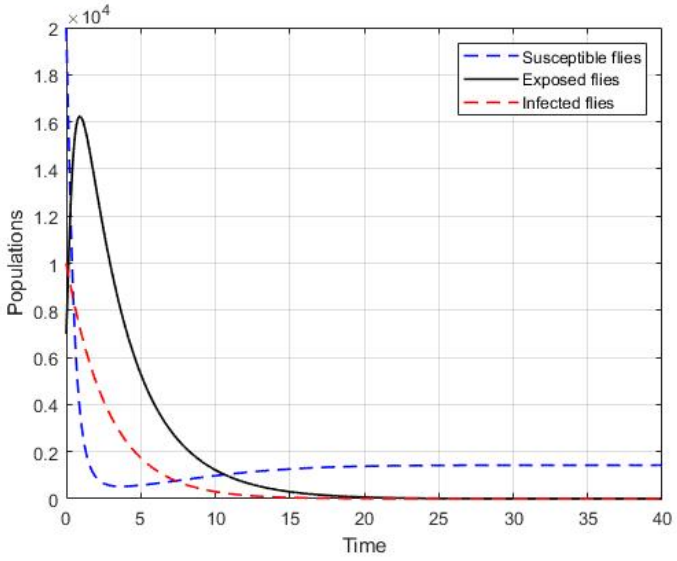


Figure 4.10. Effect of SAFE control strategy on flies population. (4.10) displays the behaviour of the trachoma model when $R_0 < 1$

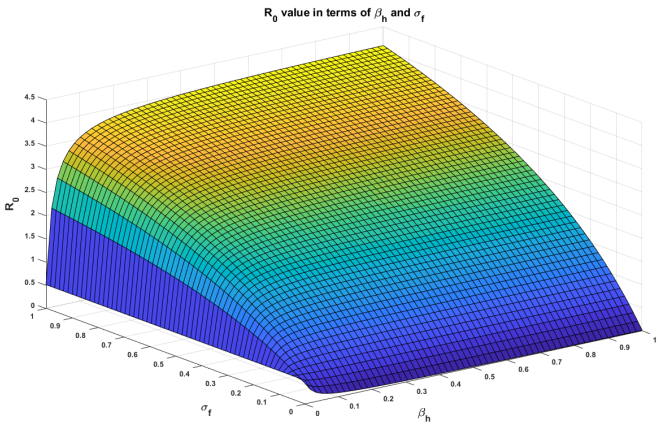


Figure 4.11. Profile of reproductive number with variation in the contact rate of the disease vector σ_f, and that of human transmission rates (β_h,)

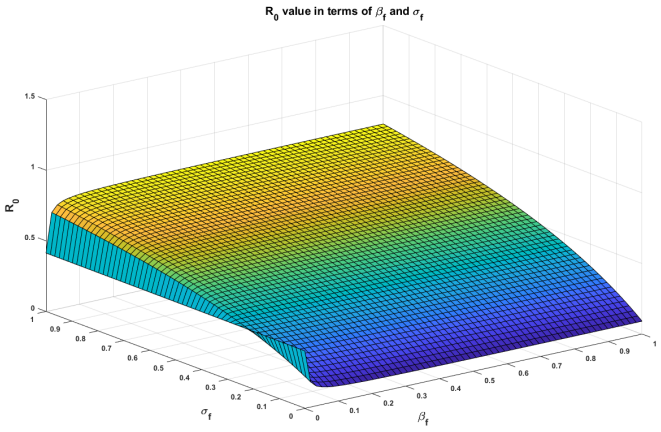


Figure 4.12. Profile of reproductive number with variation in the contact rate of the disease vector σ_f, and that of vector transmission rates (β_f)

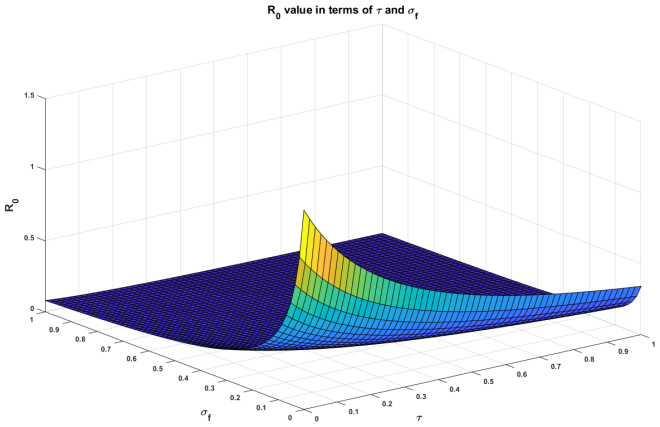


Figure 4.13. Profile of reproductive number with variation in the contact rate of the disease vector σ_f, and that of vector progression rate from exposed to infectious flies τ,

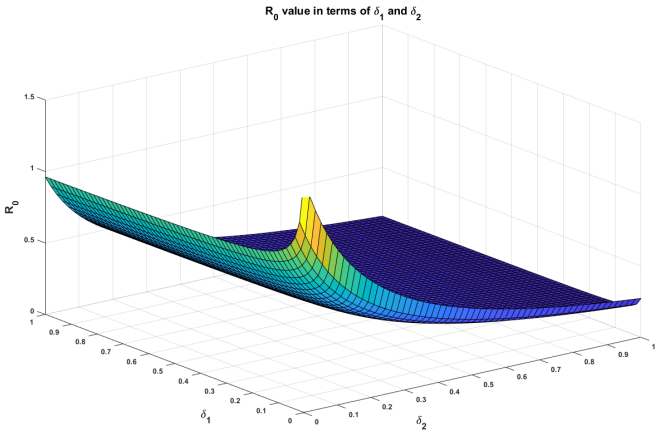


Figure 4.14. ofile of reproductive number with variation in human transmission rates from exposed individual class to early stage of trachoma (δ_1) to severe stage (δ_2).

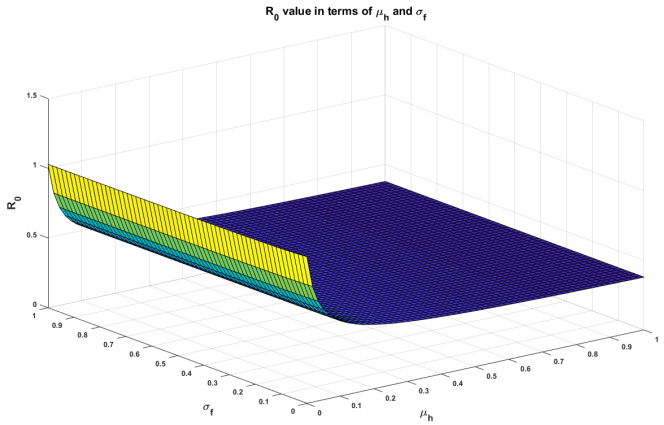


Figure 4.15. Profile of reproductive number with variation in contact rate of the disease vector σ_f, and that of the natural death rate of human μ_h,

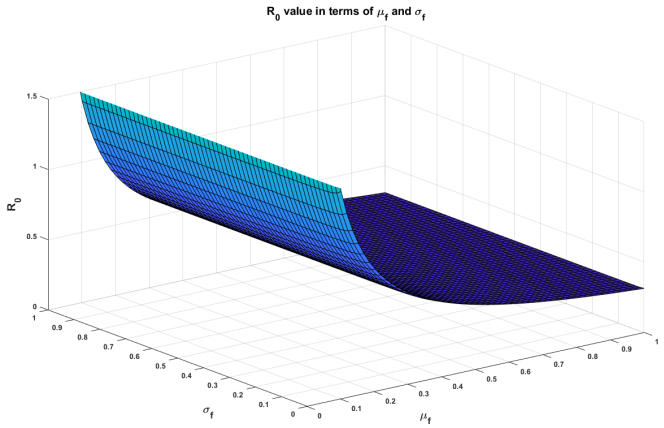


Figure 4.16. Profile of reproductive number with variation in contact rate of the disease vector σ_f, and the natural death rate of the eye-seeking fly μ_f.

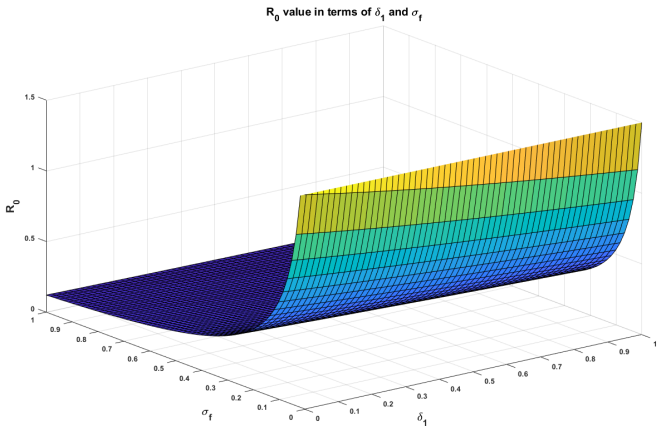


Figure 4.17. Profile of reproductive number with variation in the contact rate of the disease vector σ_f, and the human transmission rates from exposed individual class to early stage of trachoma (δ_1

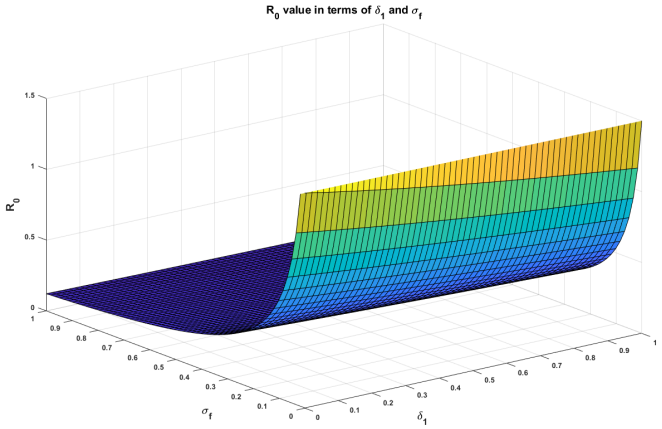


Figure 4.18. Profile of reproductive number with variation in the contact rate of the disease vector σ_f, and the human transmission rates from exposed individual to severe stage of trachoma (δ_2)

4.2 DISCURSION OF RESULTS

we have developed and rigorously analyzed a compartmental model of (Kermack-Mckenderick) type to monitor the transmission dynamics of chlamydia trachomatis in a multi-stains structured format. It is confirmed that the local asymptotic stability of the disease-free equilibrium and the global asymptotic stability of the unique endemic equilibrium is guaranteed whenever the computed basic reproduction number R_0 is below unity and R_0 greater than one respectively. We have also estimated the model parameters and fit the model with the use of field data cases from Northern Nigeria using least-square fitting method.

Moreover, we displayed the significance of the model parameters in changing the size of the basic reproduction number R_0^T which can be seen in figure 3.3. it is confirmed from computed elasticity indices that the most sensitive parameter to basic reproduction number is σ_f (vector contact rate), followed by rates of transmission (β_h and β_f) then the rest follows. The elasticity expressions of the model parameters and their respective indices shown in section 3.6. reveal that those parameters whose indices are other than 1 change with the change in some of the parameters associated with their computed elasticity expression. However, investigating how manifested is the variation when a particular parameter is adjusted either (increase or decreased) is becoming of interest, starting with ψ_1 and leaving other parameters unchanged, a plot against ψ_1 of R_0^T is shown in figure 4.1., similar figure 4.2 shows the relationship between the basic reproduction number R_0^T against the parameter ψ_2, and figures (4.3 and 4.4) displayed the sensitivity of the parameters τ and σ_f to R_0^T respectively, while figures (4.5 and 4.6) shows the sensitivity of human transmission rates β_h and β_f to R_0^T respectively. From our graphs in figures (4.1- 4.2) above, we can see that while keeping the parameters unchanged if not for μ_h which is adjusted from 0.0014 - 0.03, whenever the non clinical control ψ_1 (i.e the E and F components of SAFE strategy) is adjusted from its normal 0.10 to 2.00 the model associated basic reproduction number R_0^T decreases from its endemic value of 1.6 to a disease-free state 0.78. This is true because, improving environmental sanitation and adequate supply of pure water give rise to regular facial cleanliness which plays a significant role in reducing the level exposures to trachoma in the society. In similar way the graphs show the remaining parameters ($\psi_2, \tau, \sigma_f, \beta_h$ and β_f) displaying their respective influence in changing the

basic reproduction number R_0^T, see figures (4.3 -4.6). Furthermore, figure (4.7 - 4.10) display the behaviour of the trachoma model when $R_0 > 1$, and when $R_0 < 1$. It is revealed that the effective use of WHO adopted control scheme (SAFE) plays a significant role toward achieving the WHO targeted Trachoma eradication as challenge in the public health in the next 10years. We have also obtained some simulation results with the aid of mesh plots for the reproductive number R_0 as a function of two different biological parameters as presented in figures(4.11 - 4.18).
Furthermore, it should also be emphasized that the present study will be strengthened in future research work by analyzing and investigating the modern fractional operators and optimal control system. The control strategies are indeed a significant step in drastically curtailing the unknown characteristics and other features of this epidemic.

CHAPTER 5
CONCLUSIONS AND RECOMMENDATIONS

5.1 CONCLUSIONS

we have developed and rigorously analyzed a compartmental model of (Kermack-Mckenderick) type to monitor the transmission dynamics of chlamydia trachomatis in a multi-stains structured format. It is confirmed that the local asymptotic stability of the disease-free equilibrium and the global asymptotic stability of the unique endemic equilibrium is guaranteed whenever the computed basic reproduction number R_0 is below unity and R_0 greater than one respectively. We have also estimated the model parameters and fit the model with the use of field data cases from Northern Nigeria using least-square fitting method.

Moreover, we displayed the significance of the model parameters in changing the size of the basic reproduction number R_0^T which can be seen in figure 2. it is confirmed from computed elasticity indices that the most sensitive parameter to basic reproduction number is σ_f (vector contact rate), followed by rates of transmission (β_h and β_f) then the rest follows. The elasticity expressions of the model parameters and their respective indices shown in section 3.6. reveal that those parameters whose indices are other than 1 change with the change in some of the parameters associated with their computed elasticity expression. However, investigating how manifested is the variation when a particular parameter is adjusted either (increase or decreased) is becoming of interest, starting with ψ_1 and leaving other parameters unchanged, a plot against ψ_1 of R_0^T is shown in figure(4.1), similar figure (4.2) shows the relationship between the basic reproduction number R_0^T against the parameter ψ_2, and figures (4.3 and 4.4). displayed the sensitivity of the parameters τ and σ_f to R_0^T respectively, while figures (4.4 and 4.6) shows the sensitivity of the trachoma transmission rates β_h and β_f to R_0^T respectively. From our graphs in figure (4.1-4.2) we can see that while keeping the parameters unchanged if not for μ_h which is adjusted from 0.0014 - 0.03, whenever the non clinical control ψ_1 (i.e the E and F components of SAFE strategy) is adjusted from its normal 0.10 to 2.00 the model associated basic reproduction number R_0^T decreases from its endemic value of 1.6 to a disease-free state 0.78. This is true because, improving environmental sanitation and adequate supply of pure water give

rise to regular facial cleanliness which plays a significant role in reducing the level exposures to trachoma in the society. In similar way the graphs show the remaining parameters ($\psi_2, \tau, \sigma_f, \beta_h$ and β_f) displaying their respective influence in changing the basic reproduction number R_0^T, see figures (4.2 4.6). Furthermore, figure (4.7- 4.10) displays the behaviour of the trachoma model when $R_0 > 1$, and when $R_0 < 1$. It is revealed that the effective use of WHO adopted control scheme (SAFE) plays a significant role toward achieving the WHO targeted Trachoma eradication as challenge in the public health in the next 10years. We have also obtained some simulation results with the aid of mesh plots for the reproductive number R_0 as a function of two different biological parameters see figures (4.11-4.18).

5.2 RECOMMENDATIONS

Furthermore, it should also be emphasized that the present study will be strengthened in future research work by analyzing and investigating the modern fractional operators and optimal control system. The control strategies are indeed a significant step in drastically curtailing the unknown characteristics and other features of this epidemic.

REFERENCES

Al-Rifai KM (1988) Trachoma through history. International Ophthalmology 12, 9–14.

Allen, L. J. S. (2017). A primer on stochastic epidemic models: Formulation, numerical simulation, and analysis. Infectious Disease Modelling, 2(2), 128–142. https://doi.org/10.1016/j.idm.2017.03.001

Ameen, I., Baleanu, D., Ali, H. M. (2020). An efficient algorithm for solving the fractional optimal control of SIRV epidemic model with a combination of vaccination and treatment. Chaos, Solitons and Fractals, 137, 109892. https://doi.org/10.1016/j.chaos.2020.109892

Andreasen AA, Burton MJ, Holland MJ et al. (2008) Chlamydia trachomatis ompA variants in trachoma: what do they tell us? PLoS Neglected Tropical Diseases 2, e306.

Animal Diversity Web, Onchocerca volvulus, http://animaldiversity.org/accounts/Onchocerca volvulus/, accessed Feb 2016.

Astle WF, Wiafe B, Ingram AD, Mwanga M Glassco CB (2006) Trachoma control in Southern Zambia-an international team project employing the SAFE strategy. Ophthalmic Epidemiology13, 227–236.

Bailey R, Osmond C, Mabey DC, Whittle HC Ward ME (1989) Analysis of the household distribution of trachoma in a Gambian village using a Monte Carlo simulation procedure. International Journal of Epidemiology 18, 944–951.

Bailey RL, Arullendran P, Whittle HC Mabey DC (1993) Randomised controlled trial of single-dose azithromycin in treatment of trachoma. Lancet 342, 453–456.

Bailey, R., Duong, T., Carpenter, R., Whittle, H., Mabey, D., 1999. The duration of hu-man ocular Chlamydia trachomatis infection is age dependent. Epidemiol. Infect. 123, 479e486.

Bailey R, Duong T, Carpenter R, Whittle H Mabey D (1999). The duration of human ocular Chlamydia trachomatis infection is age dependent. Epidemiology and Infection 123, 479–486.

Blake IM, Burton MJ, Bailey RL et al. (2009) Estimating household and community transmission of ocular chlamydia trachomatis. PLoS Neglected Tropical Diseases 3, e401.

Blake IM, Burton MJ, Solomon AW, West SK, Basa´n˜ez M-G, et al. (2010) Targeting Antibiotics to Households for Trachoma Control. PLoS Negl. Trop. Dis 4(11): e862. doi:10.1371/journal.pntd.0000862

Bowman RJ, Jatta B, Faal H, Bailey R, Foster A Johnson GJ (2000a) Long-term follow-up of lid surgery for trichiasis in the Gambia: surgical success and patient perceptions. Eye (London, England) 14, 864–868.

Bowman RJ, Sillah A, Van Dehn C et al. (2000b) Operational comparison of single-dose azithromycin and topical tetracycline for trachoma. Investigative Ophthalmology Visual Science 41, 4074–4079.

Bowman RJ, Jatta B, Cham B et al. (2001) Natural history of trachomatous scarring in The Gambia: results of a 12-year longitudinal follow-up. Ophthalmology 108, 2219–2224.

Bowman RJ, Faal H, MyattMet al. (2002b) Longitudinal study of trachomatous trichiasis in the Gambia. The British Journal of Ophthalmology 86, 339–343.

Brauer, F. (2017). Mathematical epidemiology: Past, present, and future. Infectious Disease Modelling, 2(2), 113–127. https://doi.org/10.1016/j.idm.2017.02.001 Britton, T. (2010). Stochastic epidemic models: A survey. Mathematical Biosciences, 225(1), 24–35. https://doi.org/10.1016/j.mbs.2010.01.006

Brauer, F., and Castillo-Chavez, C. (2012). Mathematical Models in Population Biology and Epidemiology (pp. 134-146). New York, NY: Dordrecht Heidlberg London.

Brunham RC, Laga M, Simonsen JN et al. (1990) The prevalence of Chlamydia trachomatis infection among mothers of children with trachoma. American Journal of Epidemiology 132, 946– 952.

Burton MJ Mabey DC (2009).The global burden of trachoma: a review. PLoS Neglected Tropical Diseases 3, e460.

Burton MJ, Holland MJ, Faal N et al. (2003).Which members of a community need antibiotics to control trachoma? Conjunctival Chlamydia trachomatis infection load in Gambian villages. Investigative Ophthalmology Visual Science 44, 4215–4222.

Burton MJ, Bowman RJ, Faal H et al. (2005a) Long term outcome of trichiasis surgery in the Gambia. The British Journal of Ophthalmology 89, 575–579.

Burton MJ, Bowman RJ, Faal H et al. (2006) The long-term natural history of trachomatous trichiasis in the Gambia. Investigative Ophthalmology Visual Science 47, 847–852.

Burton, M.J., Mabey, D.C.W., 2009. The global burden of trachoma: a review. PLoS Negl. Trop. Dis. 3, e460.

Caleb Mpyet, Nasiru Muhammad, Mohammed Dantani Adamu, Habila Muazu, Murtala Muhammad Umar, Musa Goyol, Hadi Bala Yahaya, Uwazoeke Onyebuchi, Chris Ogoshi, Tijjani Hussaini, Sunday Isiyaku, Adamani William, Rebecca M. Flueckiger, Brian K. Chu, Rebecca Willis, Alexandre L. Pavluck, Nicholas Olobio, Sophie Phelan, Colin Macleod, Anthony W. Solomon and for the Global Trachoma Mapping Project (2017) Prevalence of Trachoma in Kano State, Nigeria: Results of 44 Local Government Area-Level Surveys, Ophthalmic Epidemiology, 24:3, 195-203, DOI: 10.1080/09286586.2016.1265657

Central Intelligence Agency: The World Fact Book, The World of a Nation: The Center of Intelligence,

Dawson CR, Daghfous T, Messadi M, Hoshiwara I Schachter J (1976) Severe endemic trachoma in Tunisia. The British Journal of Ophthalmology 60, 245–252.

Dawson CM, Daghfous R, Juster R Schachter J (1990) What Clinical Signs are Critical in Evaluating the Impact of Intervention in Trachoma? Chlamydia Infections. Proceedings of the Seventh International Symposium on Human Chlamydial Infections Cambridge University Press, London.

Dawson CR, Schachter J, Sallam S, Sheta A, Rubinstein RA Washton H (1997) A comparison of oral azithromycin with topical oxytetracycline / polymyxin for the treatment of trachoma in children. Clinical Infectious Diseases: an official publication of the Infectious Diseases Society of America 24, 363–368.

Diekmann, O., Heesterbeek, J. A. P., Metz, J. A. J. (1990). On the definition and the computation of the basic reproduction ratio R0 in models for infectious diseases in heterogeneous populations. Journal of Mathematical Biology, 28(4), 365–382. https://doi.org/10.1007/BF00178324

Driesseche Pvd, Watmough J. Reproduction numbers and sub-threshold endermic equilibria for compartmental models of diseases transmission. Math. Bioci 2002;180:29-48.

Diekmann. O., A. Heesterbeek, JAP, Robert MG.the construction of next-generation matrices for compartmental epidemic models. J R Soc interface 2010;7:873-85.

Dolin PJ, Faal H, Johnson GJ et al. (1997) Reduction of trachoma in a sub-Saharan village in absence of a disease control programme. Lancet 349, 1511–1512.

Dolin PJ, Faal H, Johnson GJ, Ajewole J, Mohamed AA Lee PS (1998) Trachoma in The Gambia. The British Journal of Ophthalmology 82, 930–933.

Emerson PM, Lindsay SW, Walraven GE et al. (1999) Effect of fly control on trachoma and diarrhoea. Lancet 353, 1401–1403.

Emerson PM, Lindsay SW, Alexander N et al. (2004) Role of flies and provision of latrines in trachoma control: cluster-randomised controlled trial. Lancet 363, 1093–1098.

Frick KD, Basilion EV, Hanson CL Colchero MA (2003a) Estimating the burden and economic impact of trachomatous visual loss. Ophthalmic Epidemiology 10, 121–132.

Frick KD, Hanson CL Jacobson GA (2003b) Global burden of trachoma and economics of the disease. The American Journal of Tropical Medicine Hygiene 69, 1–10.

Gambhir, M., Basáñez, M.-G., Burton, M. J., Solomon, A. W., Bailey, R. L., Holland, M. J., … Grassly, N. C. (2009). The Development of an Age-Structured Model for Trachoma Transmission Dynamics, Pathogenesis and Control. PLoS Neglected Tropical Diseases, 3(6), e462.

Gebre T, Ayele B, Zerihun M, Genet A, Stoller NE, Zhou Z, House JI, Yu SN, Ray KJ, Emerson PM, et al. Comparison of annual versus twice-yearly mass azithromycin treatment for hyperendemic trachoma in Ethiopia: a cluster-randomised trial. Lancet. 2011 doi:10.1016/S0140-6736(11)61515-8. * This community-randomized trial in 24 subdistricts in northern Ethiopia demonstrated rapid elimination of ocular infection in biannual treatment groups. However, after 42 months, prevalence was similar to annual treatment groups.

Gower EW, Solomon AW, Burton MJ et al. (2006) Chlamydial positivity of nasal discharge at baseline is associated with ocular chlamydial positivity 2 months following azithromycin treatment. Investigative Ophthalmology Visual Science 47, 4767– 4771.

Grassly NC, Ward ME, Ferris S, Mabey DC Bailey RL (2008) The natural history of trachoma infection and disease in a gambian cohort with frequent follow-up. PLoS Neglected Tropical Diseases 2, e341.

Halder, A., Bhattacharya, R. (2011). Model validation: A probabilistic formulation. IEEE Conference on Decision and Control, 1692–1697. Harding-Esch EM, Edwards T, Sillah A et al. (2008) Risk factors for active trachoma in The Gambia. Transactions of the Royal Society of Tropical Medicine Hygiene 102, 1255–1262.

Hardy, A., Magnello, M. E. (2002). Statistical methods in epidemiology: Karl Pearson, Ronald Ross, Major Greenwood and Austin Bradford Hill, 1900-1945. International Journal of Public Health, 47(2), 80–89. https://doi.org/10.1142/9789813148963$_0$018.$HaylorHR(2008)Trahcoma$: $ABlindingScourgefromtheBronzeAgetotheTwenty$ – $FirstCentury.HaddingtonPress, Melbourne.HirschbergJ(1982)TheHistoryofOphthalmolog$

Antiquity.Wayenborg, Verlag.

Heffernan, J. M., Smith, R. J., Wahl, L. M. (2005). Perspectives on the basic reproductive ratio. Journal of the Royal Society Interface, 2(4), 281–293. Hethcote, H. W. (2000). The Mathematics of Infectious Diseases. SIAM Review, 42(4), 599–653. Hoechsmann A, Metcalfe N, Kanjaloti S et al. (2001) Reduction of trachoma in the absence of antibiotic treatment: evidence from a population-based survey in Malawi. Ophthalmic Epidemiology 8, 145–153.

Hethcote, H.W. The mathematics of infectious diseases. SIAM Rev., 42,pp. 599-653. 2000.

Hotez, P.J., Alvarado, M., Basanez, M.G., Bolliger, I., Bourne, R., Boussinesq, M., Brooker, S.J., Brown, A.S., Buckle, G., Budke, C.M., Carabin, H., Coffeng, L.E., Fevre, E.M., Furst, T., Halasa, Y.A., Jasrasaria, R., Johns, N.E., Keiser, J., King, C.H., Lozano, R., Murdoch, M.E., O'Hanlon, S., Pion, S.D., Pullan, R.L., Ramaiah, K.D., Roberts, T., Shepard, D.S., Smith, J.L., Stolk, W.A., Undurraga, E.A., Utzinger, J., Wang, M., Murray, C.J., Naghavi, M., 2014. The global burden of disease study 2010: interpretation and implications for the neglected tropical diseases. PLoS Negl. Trop. Dis. 8, e2865.

Hu VH, Harding-Esch EM, Burton MJ, Bailey RL, Kadimpeul J, Mabey DCW. Epidemiology and control of trachoma: systematic review. Trop. Med. Int. Health. 2010; 15:673–691. [PubMed: 20374566]

Hu VH, Massae P, Weiss HA, Cree IA, Courtright P, Mabey DCW, Bailey RL, Burton MJ. In vivo confocal microscopy of trachoma in relation to normal tarsal conjunctiva. Ophthalmology. 2011; 118:747–754. * This study proposes a grading system to characterize conjunctival scarring and inflammation, representing the first time confocal microscopy has been applied to trachoma. [PubMed: 21055819]

Hu VH, Weiss HA, Massae P, Courtright P, Makupa W, Mabey DCW, Bailey RL, Burton MJ. In vivo confocal microscopy in scarring trachoma. Ophthalmology. 2011; 118:2138–2146. [PubMed: 21920608]

Huang, S. Z. (2008). A new SEIR epidemic model with applications to the theory of eradication and control of diseases, and to the calculation of R0. Mathematical Biosciences, 215(1), 84–104. https://doi.org/10.1016/j.mbs.2008.06.005

World Health Organization (WHO), 2012. Global WHO alliance for the elimination of blinding trachoma by 2020. Wkly. Epidemiol. Rec. 87, 161e168.

Wright, H.R., Taylor, H.R., 2005. Clinical examination and laboratory tests for estimation of trachoma prevalence in a remote setting: what are they really telling us? Lancet Infect. Dis. 5, 313e320.

Jones BR (1975) The prevention of blindness from trachoma. Transactions of the Ophthalmological Societies of the United Kingdom 95, 16–33.

Keeling, M. J., Rohani, P. (2008). Modeling Infectious Diseases in Humans and Animals. Princeton University Press.

Keenan JD, Lakew T, Alemayehu W, Melese M, House JI, Acharya NR, Porco TC, Gaynor BD, Lietman TM. Slow Resolution of Clinically Active Trachoma Following Successful Mass Antibiotic Treatments. Arch Ophthalmol-Chic. 2011; 129:512–513.

Keenan JD, Ayele B, Gebre T, Moncada J, Stoller NE, Zhou Z, Porco TC, McCulloch CE, Gaynor BD, Emerson PM, et al. Ribosomal RNA Evidence of Ocular Chlamydia trachomatis Infection Following 3 Annual Mass Azithromycin Distributions in Communities With Highly Prevalent Trachoma. Clin. Infect. Dis. 2012; 54:253–256. [PubMed: 22095569]

Katz J, Zeger SL Tielsch JM (1988) Village and household clustering of xerophthalmia and trachoma. International Journal of Epidemiology 17, 865–869.

King JD, Ngondi J, Gatpan G, Lopidia B, Becknell S Emerson PM (2008) The burden of trachoma in ayod county of southern Sudan. PLoS Neglected Tropical Diseases 2, e299.

Kuper H, Solomon AW, Buchan J, Zondervan M, Foster A Mabey D (2003) A critical review of the SAFE strategy for the prevention of blinding trachoma. Lancet Infectious Diseases 3, 372–381.

Lakshmikantham, V., Leela, S., Martynyuk, A. A. Stability Analysis of Nonlinear Systems. Mareel Dekker, Inc., New York and Basel, pp.155-170. 2000.

Maccallan AF (1931) The epidemiology of trachoma. The British Journal of Ophthalmology 15, 369–411.

Malaty R, Zaki S, Said ME, Vastine DW, DawsonDWSchachter J (1981) Extraocular infections in children in areas with endemic trachoma. The Journal of Infectious Diseases 143, 853.

Mariotti SP, Pascolini D Rose-Nussbaumer J (2009) Trachoma: global magnitude of a preventable cause of blindness. The British Journal of Ophthalmology 93, 563–568.

Martcheva, M. (2013). An Introduction to Mathematical Epidemiology. Springer Science+Business Media LLC.

Melese M, Chidambaram JD, Alemayehu W et al. (2004b) Feasibility of eliminating ocular Chlamydia trachomatis with repeat mass antibiotic treatments. The Journal of the American Medical Association 292, 721–725.

Meraf A. Wolle and Sheila K. West (2019) Ocular Chlamydia trachomatis infection: elimination with mass drug administration, Expert Review of Anti-infective Therapy, 17:3, 189-200, DOI: 10.1080/14787210.2019.1577136

Michel C-EC, Roper KG, Divena MA, Lee HH, Taylor HR. Correlation of clinical trachoma and infection in Aboriginal communities. PLoS Negl Trop Dis. 2011; 5:e986. [PubMed: 21423648]

Miller K, Pakpour N, Yi E et al. (2004a) Pesky trachoma suspect finally caught. The British Journal of Ophthalmology 88, 750– 751. Miller

Munoz B, Bobo L, Mkocha H, Lynch M, Hsieh YH and West S (1999) Incidence of trichiasis in a cohort of women with and without scarring. International Journal of Epidemiology 28, 1167–1171.

Munoz B, Stare D, Mkocha H, Gaydos C, Quinn T, West SK. Can clinical signs of trachoma be used after multiple rounds of mass antibiotic treatment to indicate infection? Invest. Ophthalmol. Vis. Sci. 2011; 52:8806–8810. [PubMed: 22025578].

Murray, C.J., Vos, T., Lozano, R., Naghavi, M., Flaxman, A.D., Michaud, C., Ezzati, M., Shibuya, K., Salomon, J.A., Abdalla, S., Aboyans, V., Abraham, J., Ackerman, I.,

Aggarwal, R., Ahn, S.Y., Ali, M.K., Alvarado, M., Anderson, H.R., Anderson, L.M., Andrews, K.G., Atkinson, C., Baddour, L.M., Bahalim, A.N., Barker-Collo, S., Barrero, L.H., Bartels, D.H., Basanez, M.G., Baxter, A., Bell, M.L., Benjamin, E.J., Bennett, D., Bernabé, E., Bhalla, K., Bhandari, B., Bikbov, B., Bin Abdulhak, A., Birbeck, G., Black, J.A., Blencowe, H., Blore, J.D., Blyth, F., Bolliger, I., Bonaventure, A., Boufous, S., Bourne, R., Boussinesq, M., Braithwaite, T., Brayne, C., Bridgett, L., Brooker, S., Brooks, P., Brugha, T.S., Bryan-Hancock, C., Bucello, C., Buchbinder, R., Buckle, G., Budke, C.M., Burch, M., Burney, P., Burstein, R., Calabria, B., Campbell, B., Canter, C.E., Carabin, H., Carapetis, J., Carmona, L., Cella, C., Charlson, F., Chen, H., Cheng, A.T., Chou, D., Chugh, S.S., Coffeng, L.E., Colan, S.D., Colquhoun, S., Colson, K.E., Condon, J., Connor, M.D., Cooper, L.T., Corriere, M., Cortinovis, M., de Vaccaro, K.C., Couser, W., Cowie, B.C., Criqui, M.H., Cross, M., Dabhadkar, K.C., Dahiya, M., Dahodwala, N., Damsere-Derry, J., Danaei, G., Davis, A., De Leo, D., Degenhardt, L., Dellavalle, R., Delossantos, A., Denenberg, J., Derrett, S., Des Jarlais, D.C., Dharmaratne, S.D., Dherani, M., Diaz-Torne, C., Dolk, H., Dorsey, E.R., Driscoll, T., Duber, H., Ebel, B., Edmond, K., Elbaz, A., Ali, S.E., Erskine, H., Erwin, P.J., Espindola, P., Ewoigbokhan, S.E., Farzadfar, F., Feigin, V., Felson, D.T., Ferrari, A., Ferri, C.P., Fevre, E.M., Finucane, M.M., Flaxman, S., Flood, L., Foreman, K., Forouzanfar, M.H., Fowkes, F.G., Fransen, M., Freeman, M.K., Gabbe, B.J., Gabriel, S.E., Gakidou, E., Ganatra, H.A., Garcia, B., Gaspari, F., Gillum, R.F., Gmel, G., Gonzalez-Medina, D., Gosselin, R., Grainger, R., Grant, B., Groeger, J., Guillemin, F., Gunnell, D., Gupta, R., Haagsma, J., Hagan, H., Halasa, Y.A., Hall, W., Haring, D., Haro, J.M., Harrison, J.E., Havmoeller, R., Hay, R.J., Higashi, H., Hill, C., Hoen, B., Hoffman, H., Hotez, P.J., Hoy, D., Huang, J.J., Ibeanusi, S.E., Jacobsen, K.H., James, S.L., Jarvis, D., Jasrasaria, R., Jayaraman, S., Johns, N., Jonas, J.B., Karthikeyan, G., Kassebaum, N., Kawakami, N., Keren, A., Khoo, J.P., King, C.H., Knowlton, L.M., Kobusingye, O., Koranteng, A., Krishnamurthi, R., Laden, F., Lalloo, R., Laslett, L.L., Lathlean, T., Leasher, J.L., Lee, Y.Y., Leigh, J., Levinson, D., Lim, S.S., Limb, E., Lin, J.K., Lipnick, M., Lipshultz, S.E., Liu, W., Loane, M., Ohno, S.L., Lyons, R., Mabweijano, J., MacIntyre, M.F., Malekzadeh, R., Mallinger, L., Manivannan, S., Marcenes, W., March, L., Margolis, D.J., Marks, G.B., Marks, R., Matsumori, A., Matzopoulos, R.,

Mayosi, B.M., McAnulty, J.H., McDermott, M.M., McGill, N., McGrath, J., Medina-Mora, M.E., Meltzer, M., Mensah, G.A., Merriman, T.R., Meyer, A.C., Miglioli, V., Miller, M., Miller, T.R., Mitchell, P.B., Mock, C., Mocumbi, A.O., Moffitt, T.E., Mokdad, A.A., Monasta, L., Montico, M., Moradi-Lakeh, M., Moran, A., Morawska, L., Mori, R., Murdoch, M.E., Mwaniki, M.K., Naidoo, K., Nair, M.N., Naldi, L., Narayan, K.M., Nelson, P.K., Nelson, R.G., Nevitt, M.C., Newton, C.R., Nolte, S., Norman, P., Norman, R., O'Donnell, M., O'Hanlon, S., Olives, C., Omer, S.B., Ortblad, K., Osborne, R., Ozgediz, D., Page, A., Pahari, B., Pandian, J.D., Rivero, A.P., Patten, S.B., Pearce, N., Padilla, R.P., Perez-Ruiz, F., Perico, N., Pesudovs, K., Phillips, D., Phillips, M.R., Pierce, K., Pion, S., Polanczyk, G.V., Polinder, S., Pope 3rd, C.A., Popova, S., Porrini, E., Pourmalek, F., Prince, M., Pullan, R.L., Ramaiah, K.D., Ranganathan, D., Razavi, H., Regan, M., Rehm, J.T., Rein, D.B., Remuzzi, G., Richardson, K., Rivara, F.P., Roberts, T., Robinson, C., De Leon, F.R., Ronfani, L., Room, R., Rosenfeld, L.C., Rushton, L., Sacco, R.L., Saha, S., Sampson, U., Sanchez-Riera, L., Sanman, E., Schwebel, D.C., Scott, J.G., Segui-Gomez, M., Shahraz, S., Shepard, D.S., Shin, H., Shivakoti, R., Singh, D., Singh, G.M., Singh, J.A., Singleton, J., Sleet, D.A., Sliwa, K., Smith, E., Smith, J.L., Stapelberg, N.J., Steer, A., Steiner, T., Stolk, W.A., Stovner, L.J., Sudfeld, C., Syed, S., Tamburlini, G., Tavakkoli, M., Taylor, H.R., Taylor, J.A., Taylor, W.J., Thomas, B., Thomson, W.M., Thurston, G.D., Tleyjeh, I.M., Tonelli, M., Towbin, J.A., Truelsen, T., Tsilimbaris, M.K., Ubeda, C., Undurraga, E.A., van der Werf, M.J., van Os, J., Vavilala, M.S., Venketasubramanian, N., Wang, M., Wang, W., Watt, K., Weatherall, D.J., Weinstock, M.A., Weintraub, R., Weisskopf, M.G., Weissman, M.M., White, R.A., Whiteford, H., Wiebe, N., Wiersma, S.T., Wilkinson, J.D., Williams, H.C., Williams, S.R., Witt, E., Wolfe, F., Woolf, A.D., Wulf, S., Yeh, P.H., Zaidi, A.K., Zheng, Z.J., Zonies, D., Lopez, A.D., Al Mazroa, M.A., Memish, Z.A., 2012. Disability-adjusted life years (DALYs) for 291 diseases and injuries in 21 regions, 1990e2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 380, 2197e2223.

Negrel AD Mariotti SP (1999) Trachoma rapid assessment: rationale and basic principles. Community Eye Health 12, 51–53.

Ngondi J, Ole-Sempele F, Onsarigo A et al. (2006a) Blinding trachoma in postconflict

southern Sudan. PLoS Medicine 3, e478.

Ngondi J, Ole-Sempele F, Onsarigo A et al. (2006b) Prevalence and causes of blindness and low vision in southern Sudan. PLoS Medicine 3, e477.

Ngondi J, Reacher M, Matthews F et al. (2007) The epidemiology of low vision and blindness associated with trichiasis in southern Sudan. BMC Ophthalmology 7, 12.

Ngondi J, Matthews F, Reacher M, Baba S, Brayne C Emerson P (2008) Associations between active trachoma and community intervention with antibiotics, facial cleanliness, and environmental improvement (A,F,E). PLoS Neglectd Tropical Diseases 2, e229.

Ngondi J, Gebre T, Shargie EB et al. (2009a) Evaluation of Three Years of the SAFE Strategy (Surgery, Antibiotics, Facial cleanliness and Environmental Improvement) for Trachoma Control in Five Districts of Ethiopia Hyperendemic for Trachoma. Transactions of the Royal Society of Tropical Medicine Hygiene.

Pinsent A, Hollingsworth TD (2018) Optimising sampling regimes and data collection to inform surveillance for trachoma control. PLoS Negl Trop Dis 12(10): e0006531. https://doi.org/10.1371/journal.pntd.0006531.

Polack S, Brooker S, Kuper H, Mariotti S, Mabey D Foster A (2005) Mapping the global distribution of trachoma. Bulletin of the World Health Organization 83, 913–919.

Rabiu M, Alhassan M Ejere H (2007) Environmental sanitary interventions for preventing active trachoma. Cochrane Database of Systematic Reviews CD004003.

Rao, F. (2014). Dynamics Analysis of a Stochastic SIR Epidemic Model. Abstract and Applied Analysis, 2014, 1–9.

Reacher MH, Munoz B, Alghassany A, Daar AS, Elbualy M Taylor HR (1992) A controlled trial of surgery for trachomatous trichiasis of the upper lid. Archives of Ophthalmology 110, 667–674.

Resnikoff S, Pascolini D, Etya'ale D et al. (2004) Global data on visual impairment in the year 2002. Bulletin of the World Health Organization 82, 844–851.

R.M. Anderson, R.M.May, Infectious diseases of Humans: Dynamics and Control, Oxford University Press,Oxford, 1990.

Roddam, A. W. (2001). Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation. International Journal of Epidemiology, 30(1), 186. https://doi.org/10.1093/ije/30.1.186

Schachter J, West SK, Mabey D et al. (1999) Azithromycin in control of trachoma. Lancet 354, 630–635.

See CW, Alemayehu W, Melese M, Zhou Z, Porco TC, Shiboski S, Gaynor BD, Eng J, Keenan JD, Lietman TM. How reliable are tests for trachoma?–a latent class approach. Invest. Ophthalmol. Vis. Sci. 2011; 52:6133–6137. [PubMed: 21685340]

ShattockAJ,GambhirM,Taylor HR, Cowling CS,Kaldor JM,Wilson DP(2015).Controlof Trachoma in Australia:AModel Based Evaluation of Current Interventions.PLoSNeglTropDis9(4): e0003474.doi:10.1371/journal.pntd.0003474

Stare D, Harding-Esch E, Munoz B, Bailey R, Mabey D, Holland M, Gaydos C, West S, Group OBOPS. Design and Baseline Data of a Randomized Trial to Evaluate Coverage and Frequency of Mass Treatment with Azithromycin: The Partnership for Rapid Elimination of Trachoma (PRET) in Tanzania and The Gambia. Ophthalmic Epidemiol. 18:20–19. [PubMed: 21275593]

Sokat, K. Y., Edlund, S., Clarkson, K., Kaufman, J. (2019). Comparing direct and indirect transmission in a simple model of veterinary disease. Mathematics, 7(11), 1–14.

Solomon AW, Holland MJ, Burton MJ et al. (2003) Strategies for control of trachoma: observational study with quantitative PCR. Lancet 362, 198–204.

Solomon AW, Foster A, Mabey DCW. Clinical examination versus Chlamydia trachomatis assays to guide antibiotic use in trachoma control programmes. Lancet Infect Dis. 2006; 6:5–6. author reply 7–8. [PubMed: 16377526]

Tabbara KF, Abu-El-Asrar A, Al-Omar O, Choudhury AH Al-Faisal Z (1996) Single-dose azithromycin in the treatment of trachoma. A randomized, controlled study. Ophthalmology 103, 842–846.

Taylor HR, Velasco FM Sommer A (1985) The ecology of trachoma: an epidemiological study in southern Mexico. Bulletin of the World Health Organization 63, 559–567.

Taylor, H.R., Burton, M.J., Haddad, D., West, S., Wright, H., 2014. Trachoma. Lancet 384, 2142e2152.

Tellis B, Keeffe JETaylorHR(2007) Surveillance report for activetrachoma, 2006: National Trachoma Surveillance and Reporting Unit. Communicable Diseases Intelligence 31, 366–374.

Thylefors B, Dawson CR, Jones BR, West SK Taylor HR (1987) A simple system for the assessment of trachoma and its complications. Bulletin of the World Health Organization 65, 477–483.

Thylefors B, Negrel AD, Pararajasegaram R Dadzie KY (1995) Global data on blindness. Bulletin of the World Health Organization 73, 115–121.

Van den Driessche P., Wanmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartimental models of disease transmition. Math. Biosci,180,29-48. 2000.

Wang SP, Grayston JT. Pannus with experimental trachoma and inclusion conjunctivitis agent infection of Taiwan monkeys. Am. J. Ophthalmol. 1967; 63(Suppl):1133–45. [PubMed: 4960878]

Wang, X. (2015). An SIRS Epidemic Model with Vital Dynamics and a Ratio-Dependent Saturation Incidence Rate. Discrete Dynamics in Nature and Society, 2015(1). https://doi.org/10.1155/2015/720682

West SK (1999) Azithromycin for control of trachoma. Community Eye Health 12, 55–56.

West SK, Munoz B, Turner VM, Mmbaga BB and Taylor HR (1991b) The epidemiology of trachoma in central Tanzania. International Journal of Epidemiology 20, 1088–1092.

West S, Munoz B, Bobo L et al. (1993) Nonocular Chlamydia infection and risk of ocular reinfection after mass treatment in a trachoma hyperendemic area. Investigative Ophthalmology and Visual Science 34, 3194–3198.

West SK, Munoz B, Mkocha H, Hsieh YH and Lynch MC (2001) Progression of active trachoma to scarring in a cohort of Tanzanian children. Ophthalmic Epidemiology 8, 137–144.

West S, Munoz B, Mkocha H, Holland M, Aguirre A, Solomon A, Foster A, Bailey R, Mabey D. Infection with Chlamydia trachomatis after mass treatment of a trachoma hyperendemic community in Tanzania: a longitudinal study. Lancet. 2005; 366:1296–1300. [PubMed: 16214600]

West SK, Munoz B, Mkocha H, Gaydos CA, Quinn TC. Number of Years of Annual Mass Treatment With Azithromycin Needed to Control Trachoma in Hyper-endemic Communities in Tanzania. J. Infect. Dis. 2011; 204:268–273. * This study examined communities after multiple years of MDA in a previously hyperendemic areas (average prevalence 51 percent in children under 5). A regression model predicts that ¿7 years of annual treatment is needed to achieve control of hyperendemic communities (infection ¡5 percent). Further examination of antibiotic coverage and its relation to the reduction of infection can provide insight into effective treatment strategies. [PubMed: 21673038]

West SK, Stare D, Mkocha H, Munoz B, Gaydos C, Quinn TC. Do infants increase the risk of re-emergent infection in households after mass drug administration for trachoma? Invest. Ophthalmol. Vis. Sci. 2011; 52:6040–6042. [PubMed: 21680866]

WHO (2006) Trachoma Control – A Guide for Programme Managers. World Health Organization, Geneva.

WHO (2008) The Global Burden of Disease: 2004 Update. World

WHO. (2018). The top 10 causes of death, Fact Sheet. https://www.who.int/news-room/fact-sheets/detail/the-top-10-causes-of-death

Wilkinson, R. R., Ball, F. G., and Sharkey, K. J. (2016). The deterministic Kermack–McKendrick model bounds the general stochastic epidemic. Journal of Applied Probability, 53(4), 1031–1040.

Wolle MA, Munoz B, Mkocha H West SK (2009) Age, sex, and cohort effects in a longitudinal study of trachomatous scarring. Investigative Ophthalmology and Visual

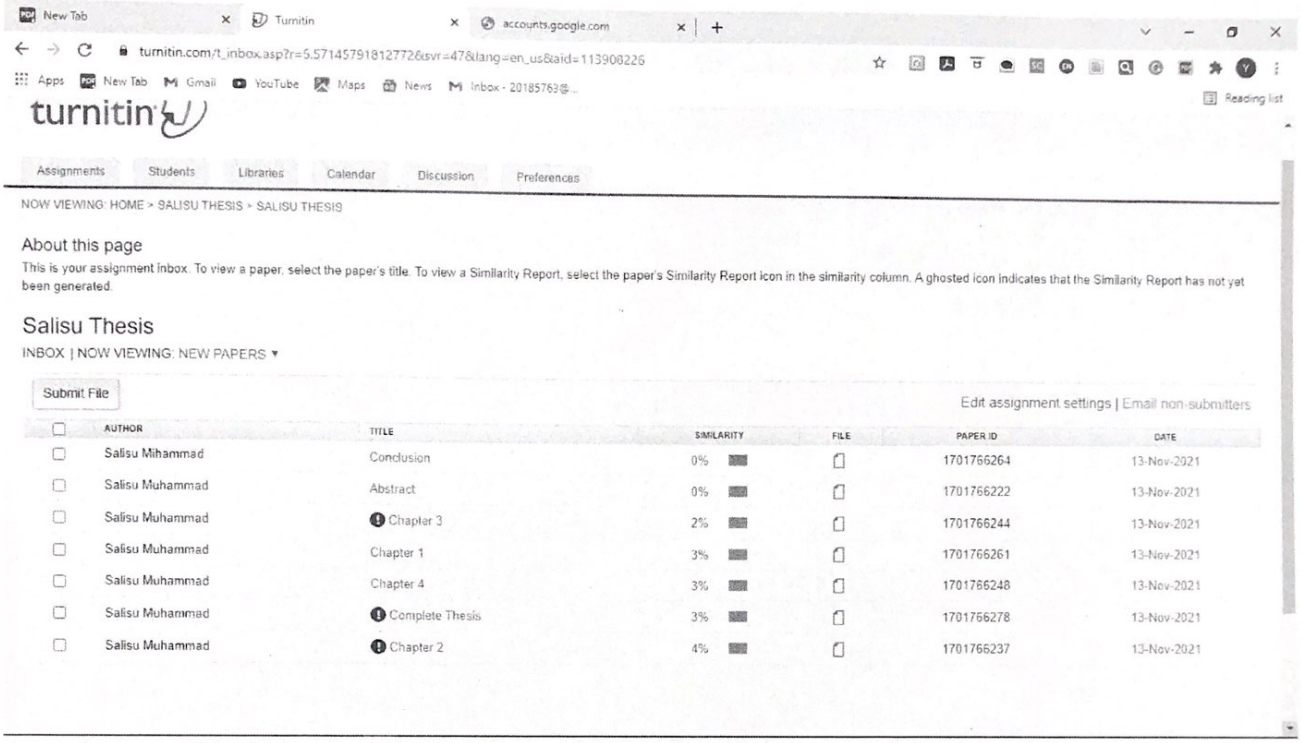
Science 50, 592–596.

W.O. Kermack, A.G. Mackendrick, A contribution to the mathematical theory of epidemic, Proc. R. Soc. Lond. Ser. A 115 (1927) 700–721.

Yang JL, Schachter J, Moncada J, Habte D, Zerihun M, House JI, Zhou Z, Hong KC, Maxey K, Gaynor BD, et al. Comparison of an rRNA-based and DNA-based nucleic acid amplification test for the detection of Chlamydia trachomatis in trachoma. Br J Ophthalmol. 2007; 91:293–295. [PubMed: 17050583]

Zack R, Mkocha H, Zack E, Munoz B West SK (2008) Issues in defining and measuring facial cleanliness for national trachoma control programs. Transactions of the Royal Society of Tropical Medicine and Hygiene 102, 426–431.

Zhang, T. (2015). Permanence and extinction in a nonautonomous discrete SIRVS epidemic model with vaccination. Applied Mathematics and Computation, 271, 716–729.



Prof. Dr. Kuren Hamad

13.11.21

CURRICULUM VITAE

MALAMAWA AREA BUNGUDU.
BUNGUDU LOCAL GOVERNMENT,
ZAMFARA STATE, NIGERIA.
08066326719, 07011448978
shadowzainab@yahoo.com

PERSONAL PROFILE:

NAME:	Salisu Muhammad Muhammad
PLACE OF BIRTH:	Bungudu
DATE OF BIRTH:	22 June. 1984
LOCAL GOVERNMENT:	Bungudu
STATE OF ORIGIN:	Zamfara
NATIONALITY:	Nigerian
MARITAL STATUS:	Married
TELEPHONE:	08066326719

EDUCATIONAL QUALFICATIONS WITH DATE(S):

- *PhD Computational mathematics* *2018-date*
- *M. Sc. Computational Mathematics* *2011-2014*
- B.Sc. Mathematics 2004-2008
- Certificate in Microsoft office Tools 2010
- Senior secondary school certificate 1997-2003
- Primary school certificate 1991-1997
- NYSC 2009-2010

SCHOOLS ATTENDED WITH DATE(S):

- Near East University Cyprus 2018-date
- Usmanu Danfodiyo University Sokoto 2011-2014
- Usmanu Danfodiyo University Sokoto 2004-2008
- Flying High Computers Sokoto 2009/2010
- Govt. Science Secondary School Gusau 1997-2003
- Nahuche One Model Primary school 1991-1997

WORKING EXPERIENCES:

- Federal University Gusau, Mathematics Department, Assistant Lecturer 2015-2018
- Federal University Gusau, Mathematics Department, Lecturer II 2018-2020
- Federal University Gusau, Mathematics Department, Lecturer II 2020-date
- Federal University Gusau, Mathematics Department, Examination officer 2016-2018
- Interim President, Bungudu Amalgamated Student Association (BASA) 2017-2019
- Chairman Strategic planning committee, Department of Mathematical Science Federal University Gusau
  2017-2018
- Chairman Accreditation committee , Department of Mathematical Science Federal University Gusau 2017- 2018
- Secretary Examination committee, Department of Mathematical Science Federal University Gusau
  2016-2018
- Federal University Gusau, Part time Lecturer Mathematics Department, School of Pre sciences, 2014-date
- Zamfara College of Art and Science (ZACAS) Gusau, Lecturer Mathematics Department 2012 – 2015
- Zamfara College of Art and Science (ZACAS) Gusau, Secretary Examination Malpractice Committee, 2014
- Mathematics Teacher Nagarta College Sokoto, 2010
- Ondo State Ministry of Education, Planning Office (NYSC) 2009
- Zamfara state Universal Basic Education Board Gusau, Education Officer II 2008 -2011
- Zamfara state Universal Basic Education Board Gusau, Assistant Headmaster, Nahuche One Model Pri. Sch. 2007-2008

- Zamfara state Universal Basic Education Board Gusau, class room teacher, Nahuche I Model primary School 2005-2007

- Interim President, Bungudu Amalgamated Student Association (BASA) 2017-2019
- Chairman Strategic planning committee, Department of Mathematical Science Federal University Gusau 2017- 2018
- Chairman Accreditation committee , Department of Mathematical Science Federal University Gusau 2017- 2018
- Secretary Examination committee, Department of Mathematical Science Federal University Gusa 2016-2018
- Teaching /Supervision /Professional Practice Load, please indicate season. 2017…. /2018…..

S/No	Course	Course Code	Number of Unit	Points Scored	Total Scores
1	**Abstract Algebra I**	**MTH 301**	**3**		
2	**Theory of Partial Differential Equations**	**MTH 402**	**3**		
3	**Abstract Algebra II**	**MTH 406**	**3**		
4	**Seminnar**	**MTH 400**			
5	**Project**	**MTH 410**	**6**		
	Set And Logic	**MTH 203**			

-

RESEARCH AND PUBLICATIONS

SM Muhammad, E Hincal, B Kaymakamzade, N Gokbulut (2021)

- Sensitivity analysis on the SEIR-SEI model for the dynamics of blinding trachoma, AIP Conference Proceedings 2325 (1), 020014

- SM Muhammad, E Hincal(2021)
- Mathematical Modelling for the Transmission Dynamics of blinding Trachoma Preprints

- E Hincal, B Kaymakamzade, UT Mustapha, SM Muhammad, N Gokbulut (2021) Mathematical modelling of HIV infection with the effect of horizontal and vertical transmissions
  AIP Conference Proceedings 2325 (1), 020058

- **Salisu M. Muhammad** and U. A. Ali (2014) Availability Determination of Series-Parallel system Using Markovian Birth-Death Process*. A Paper Presented at the National Conference on the Impact of Basic and Applied Science on Resources Acquisition and Management, Organized by Sokoto State University in Conjunction With the ALGEBRA GROUP of Usmanu Dan fodiyo University Sokoto March, 2014.*

- **Salisu M. Muhammad** and U. A. Ali (2015) Availability Determination of complex repairable system Using Markovian Birth-Death Process*. International Journal of science for global sustainability, vol. 1, no. 1 2015, pp 43-56...*

- **Salisu M. Muhammad,** jibril Lawal and Nasiru NaALLAH (2019). Busy Period Analysis of a complex Repairable System Using Markov process, a paper presented at the 56th annual National Conference of the Mathematical association of Nigeria (MAN) Held at Rivers State University, Port Harcourt from 1st-6th September 2019.

- Jibril Lawal **and Salisu M. Muhammad (2019),** Modelling and Analysis of the Prolonged Sitting. A conference paper presented at the 56th annual National Conference of the Mathematical association of Nigeria (MAN) Held at Rivers State University, Port Harcourt from 1st-6th September 2019.

- **Salisu M. Muhammad,** Muhammad L. Jabaka, Laminu Idris and Aliyu U. Moyi (2020) Modelling and Economic Analysis of a Simple Repairable System with Constant Failure and repairs**.** *aaaAccepted for publication in the International Journal of science for global sustainability, vol. 6, no. 3*

- **Salisu M Muhammad and** *Lawal A. Anka* (2020) Optimally Reducing Exposure to Mercury Poisons in Dareta Villages of Anka Local Government Area of Zamfara State, Nigeria Submitted for publication to *world journal of research and reviews*.

HOBBIES:

Reading/Research, Charity and tourism

REFEREES:

- **Alh. Hassan Attahiru Bungudu,**

  Emir of Bungudu, Zamfara state,

  08037041809,
- **Alh. Garba Dan Azumi Kaura,**

  Director Inspectorate Service ministry of Education,

  Gusau, Zamfara State.
- **Prof. Uba Ahmad Ali,**

  Department of Mathematics,

  Usman Danfodiyo University Sokoto,

  08067426921.