MATHEMATICAL MODEL OF CEREBROSPINAL MENINGITIS IN OBUASI MUNICIPALITY OF ASHANTI REGION, GHANA

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DECLARATION

I hereby declare that this submission is my own work towards the Master of Philosophy (MPhil.) and that, to the best of my knowledge, it contains no material previously published by another person nor material which has been accepted for the award of any other degree of the University, except where due acknowledgement has been made in the text.

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TO GOD BE THE GLORY.

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This work is dedicated to my late mother Alice Gyamfuaa, who worked assiduously to make me what I am now.

ABSTRACT

Meningococcal disease is a serious health threat in the human body which begins rapidly with a relative high case fatality rate particularly in the absence of treatment and thus is a priority when it comes to prevention. Delayed treatment can lead to death or invasive meningococcal disease which causes complications such as neurologic disorder, loss of limbs, hearing loss and paralysis even in survivors. In this work, therefore the main objective is to investigate the epidemiology of meningococcal meningitis in Obuasi municipality to develop a model for predict the spreading and combating of cerebrospinal meningitis within the municipality. Using SEIR model we employed the simple Susceptible-Exposed-Infectious and Recovered

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CHAPTER 1

INTRODUCTION

1.1 BACKGROUND INFORMATION

This chapter emphasis the background information of CSM which involve symptoms, risk factors and treatment. It also highlights statement of the problem objectives of the study, methodology, justification and organization of the study.

Cerebrospinal Meningitis (CSM) is a dangerous disease caused by Neisseria Meningitis (meningococcal) which colonizes the nasopharynx (the area of the upper throat that lies behind the nose). The disease spread when an individual come into contact with infected respiratory secretions, Mahon and Manuselis (2000).

According to Harrison (2000), humans are the sole hosts of Neisseria meningitides and it has been responsible for outbreak of the disease every year particularly in the northern part of Ghana. There are thirteen (13) different types' Sero groups of disease. Only 5 of them-A, B, C, Y and W135 are globally responsible for the disease. Currently Neisseria meningitides sero groups A and C are the most important clinically dealt with in the country. The bacteria enter the blood stream of an infected person then may invade and multiply in the cerebrospinal fluid. In most people, antibodies kill the bacteria to preventing them from causing the disease. However, it is possible to carry the meningococcal while it does not showing any symptoms of infection to the exposed class.

It usually occurs in epidemics, and symptoms are those of acute cerebral and spinal meningitis. There is also usually an eruption of erythematous, herpetic, or hemorrhagic

spots on the skin. The fulminating or malignant form is known as Waterhouse-Friderichsen syndrome.

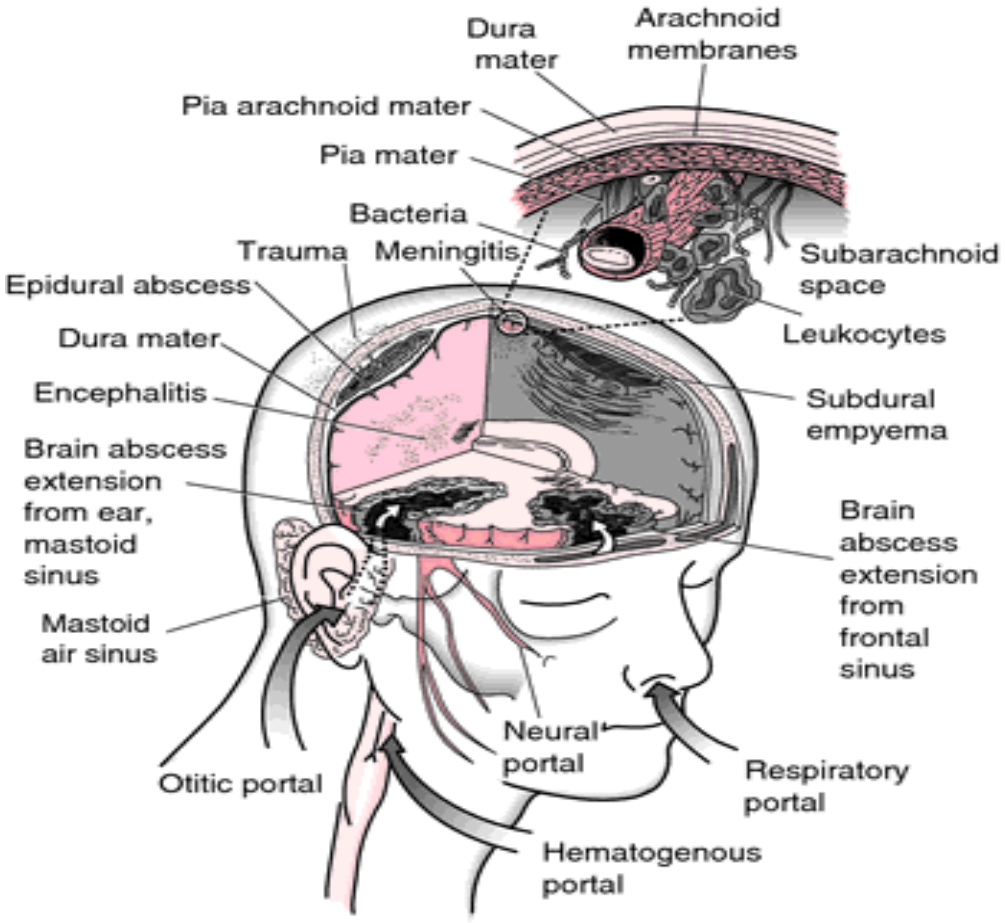


Figure 1.1 Portals of entry resulting in meningitis, meningoencephalitis, and intracranial mass lesions. From Mahon and Manuselis, 2000.

There are two types of meningitis thus, Bacterial and aseptic meningitis. Bacterial meningitis which is often severe than aseptic meningitis, affected both infants and the elderly. Prior to the use of bacteria antibiotics, about seventy (70) percent or more of bacterial meningitis cases are fatal in the northern Ghana; with the intervention of antibiotic treatment, the fatality rate has dropped to fifteen (15) percent or less. Bacterial meningitis is most common in the dry season and especially dusty areas like the mining

zones. The three bacteria that cause the cases of Meningitis are; Haemophilus influenzae, meningitidis Neisseria or Streptococcus pneumonia.

In Ghana an epidemic of CSM was reported for the first time in 1906. It started in the North-west of the Gold Coast and spread widely throughout the Southern area during the dry season (Horn, 1908). The disease spread rapidly westwards. The spread of the disease has been reported across West Africa for some time now especially in the French territories.

In Ghana, epidemics were subsequently reported in 1919/20, in 1939/40, in 1945, in 1949/50 (Waddy 1957), 1961, 1972/73 (Belcher et al. 1977), 1984 (A. Amankwa, personal communication) and in 1997/98 (Tikhomirov et al. 1997).

Figure 1.2: The African Meningitis Belt (Source: Moore 1992).

An extensive survey of published and unpublished records, obtained by personal visits to hospitals and ministries of health across West Africa, enabled Lapeyssonnie (1963) to produce the definitive report on CSM in West Africa during the first half of the 20th century: La méningite cérébrospinale en Afrique (Lapeyssonnie, 1963). He documented in detail the epidemiological features of CSM in Africa and drew attention to the fact that it is only in a restricted area of Africa that the infection behaves in such a characteristic and peculiar way. This led him to define the 'African meningitis belt', bounded to the North by the Sahara and to the south by areas of tropical rain forest.

Various mining activities augment the spread of diseases. Activities of surface mining in and around Obuasi, pollutes the air with a lot of dust which causing upper respiratory tract infections. Secondly, disposal of mining waste such as cyanide in rivers exposes the inhabitants to the risk of meningitis.

Li et al., (1999) studied SEIR epidemiological models with general nonlinear incidence in a constant population under the restriction that the rate of loss of immunity is either sufficiently small or sufficiently large, global asymptotic stability of the endemic equilibrium when it is unique is proven using a geometric approach to global stability for nonlinear autonomous systems. For the special case of bilinear incidence, this global result completes determination of a sharp threshold for the classical SEIR model under the above restriction.

Several authors have studied the compartmental modeling on the epidemics Michael Y. Li et at., (1999) studied global dynamics of a SEIR model with varying total population size. The authors reported that transmission rate and recovery rate were the dominant parameters in the spread of epidemics. Li and Zou (2009), considered a generalization

made by Kermack and Mckendric (1927) their work assumed that an infectious disease has a fixed latent period in a population.

An important factor in compartmental model is to study the activities and effects of the CSM in each compartment. The incidence of CSM has been increasing due to global warming and surface mining activities at Obuasi and its environs. It is important to understand the essential activities in the spread of the disease and develop effective, strategies for its prevention and control such as Climatic changes. This will develop gradually over several decades. Some of them, such as heat waves, which have a direct effect on human health causing heat illness such as CSM and rashes. But many of these changes will have mainly indirect effects by changing natural ecosystems thereby affecting such aspects as food production, vector-borne diseases, and a number of other infectious and non-infectious diseases.

In this thesis we shall put forward the epidemiology of cerebrospinal meningitis (CSM) and its control using a mathematical model. The model is used to predict the spread of CSM as well as impart of the vaccination interventions to control the outbreak.

1.1.1 SYMPTOMS OF CSM

Acute meningitis leads to illness developed over one or two days, but it can also rapidly progress in a matter of hours. Not everyone with meningitis will have the same symptoms, but any of the following are possible: Severe Headache, High temperature, vomiting, sensitivity to bright lights, neck stiffness, joint pains, and drowsiness or confusion.

The magnitude of disease during meningococcal epidemics makes it easy to recognize in retrospect. Since intervals between epidemics vary greatly, even in a given area, the occurrence of an epidemic is not predictable anywhere in the world.

Although there are several potential risk factors for meningococcal epidemics, not one factor is sufficient to explain why an epidemic is occurs or where one is likely to occur next. For this reason, early warning systems for meningococcal epidemics are based on information on the occurrence of meningococcal disease, rather than on changes in the prevalence.

Information from the field investigation of a suspected meningococcal outbreak should be used to calculate attack rates within a region for specific age groups (e.g. <5 years old, 5-14, 15-29, 30-44, etc.). Identifying the age groups with the highest rates of disease can help guide vaccination plans. Focusing vaccination campaigns on geographical areas and age groups with the highest rates of disease can permit rapid coverage of the highest-risk populations and save resources.

Particularly children and adults, there may be a rash of ting, red – purple spots or bruises caused by breeding under the skin. This can occur anywhere on the body. Most infected experience several illness without sudden onset and stiff neck.

The risk of getting bacterial meningitis in all age group is about 2.4 cases per 100,000 populations per year. However, the less risk group of the disease, meningococcal meningitis, is highest among children 2 to 8 years old.

If meningitis is diagnosed early and promptly attended to the majority of the people make a complete recovery. In some cases it can be fatal or a person may be left with a permanent disability, such as deafness, blindness, amputation or brain damage (resulting in mental retardation or paralysis even with prompt treatment). If earlier detection, proper treatment and medication is not administered.

Figure 1.2 depicts gangrene that has resulted from infection by Neisseria meningitides, this instance in the form of `meningococcemia:

Figure 1.3: Four month old female with gangrene of knee due to meningococcemia. Courtesy of CDC/Mr. Gust

[http://phil.cdc.gov/Phil/home.asp] Accessed 02/11/07

1.1.2 RISK FACTORS FOR CEREBROSPINAL MENINGITIS EPIDEMIC

Several factors that enhance the spread of cerebrospinal meningitis in the sub region, the risk factors for invasive disease and for outbreaks are not completely understood. Combinations of conditions (environment, host and organism) are necessary for an epidemic to occur. These include: immunological susceptibility of the population (perhaps due to loss of herd immunity to the prevalent strain), special climatic conditions (dry season, dust storm), low socioeconomic status and transmission of a virulent strain stench from mining areas. Acute respiratory tract infections may also contribute to the development of meningococcal disease epidemics. The organism causing the disease is transmitted through direct contact via respiratory droplets from an infected person to an uninfected person in a population

In considering the environmental factors, climate plays an important role in the seasonal upsurge of meningococcal disease. In sub-Saharan Africa the spread of infection may be enhanced by drought and dust storm; meningococcal epidemics generally stop with the onset of the rainy season. Low absolute humidity and dust may enhance meningococcal invasion by damaging the mucosal barrier directly or by inhibiting mucosal immune defenses. Unfavorable climatic conditions may lead to the crowding of people in poorly ventilated dwellings, where spread of virulent meningococcal is optimal.

With regards to demographic factors, travel and migration facilitate the circulation of virulent strains inside a country or from country to country. The gathering of susceptible people is an important risk factor for outbreaks, as exemplified in military communities; boarding schools and conventions where human interactions are possible for outbreaks are likely to occurred, particularly children. Large population movements, such as a pilgrimage, also play a major role in the spread of diseases. The outbreak, which occurred in Mecca in 1987, at the end of the pilgrimage period, caused more cases

among pilgrims than among the Saudi native population. In many countries, returning pilgrims caused the occurrence of cases of meningococcal meningitis in their immediate communities. Other large population displacements, example, those in refugees camps, may pose similar risks.

Moreover socioeconomic play a major role in the spreading of cerebrospinal meningitis especially Northern part of Ghana where poor living conditions and overcrowded housing are linked with the other factors contribute the to higher incidence of meningococcal disease.

Concurrent infections, thus upper respiratory tract infections may contribute to some meningococcal outbreaks. The association between acute respiratory infections and meningococcal disease has been found both in temperate and tropical climates. Mining activities and disposal of its waste cannot be failed. To notice in terms an outbreak of epidemic, discharging of waste into the streams and explosion of poisonous chemical into the air contribute the spread of meningitis.

1.1.3 TREATMENT OF CEREBROSPINAL MENINGITS

Delayed treatment can lead to death or invasive meningococcal disease which causes complications such as neurologic disorders, loss of limbs, hearing loss and paralysis even in survivors.

Treatment should be well systematized if best results are to be obtained. The disease requires constant nursing and medical attention, and poor results are inevitable if treatments are given irregularly and without a definite plan of procedure; or if nearly constant attention is not given to the patient, especially after administration of prescribe medicine. It is uncommon for a patient to collapse shortly after administration of serum (medicine). There appear to be true manifestations of collapse and usually occur in those

patients who resist treatment rather violently. In many instance prompt injection of epinephrine together with the use of artificial respiration and oxygen were lives saving procedure.

For severe cases three treatments course were given the first twenty four hours, twelve for the next two days or possibly three days and one daily until there was a define change for the better symptom and the fluid became clear.

The initial treatment approach to the patient with suspected acute bacterial meningitis depends on early recognition of the meningitis syndrome, rapid diagnostic evaluation, and emergent antimicrobial and adjunctive therapy. Lippincott Williams and Wilkins, (2001).

Personal hygiene plays an important role in the prevention of CSM, thus persons should cover their noses and mouths when sneezing or coughing and discard used tissues promptly. Wash hands thoroughly following exposure to respiratory secretions, including handling of soiled tissues and handkerchiefs. Persons should not share straws, cups, glasses, water bottles used during sports or recreation, eating utensils, cigarettes, etc. Eating and drinking utensils should not be shared and should be used by others only after they have been washed. Discouraging persons from kissing an infant, toddler or child on the mouth also can help prevent the spread of bacterial meningitis like CSM.

Preventing viral and bacterial meningitis also requires proper hand washing to remove fecal contamination after toileting, changing diapers, assisting toddlers with toileting and so forth.

All persons should avoid smoking and exposure to secondhand smoke, which are risk factors for meningococcal disease. Practice good hygiene by washing hands and covering coughs and sneezes. Proper ventilation should be observed in our homes.

Infected person should be handled with care to avoid transmission of the bacteria. Secondhand clothing should be well wash and disinfect before used. To protect against meningococcal and childhood pneumococcal infections, have your child vaccinated promptly, starting at 2 months of age; parents should not wait until the child is ready to enter school before injection for immunization. For adolescents, a single dose of meningococcal conjugate vaccine (MCV) is recommended, starting at age 11-12 years. MCV prevents infection with four strains of meningococcal bacteria, including two of three major strains in the Northern part of Ghana

According to Branford (2010), bacterial meningitis is treated with antibiotics intravenous. Corticosteroid drugs, such as dexamethasone may also be used to reduce inflammation in the brain. A person who closes contact with someone who has been diagnosed with meningitis may also need antibiotics, proper ventilation should be provided in our homes. Personal hygiene should also be observed to avoid the spreading of the disease. Symptoms relating to CSM should quickly be reported to hospital for early treatment.

1.2 STATEMENT OF THE PROBLEM

In spite of vaccination strategies, the incidence of CSM has become a serious global concern especially those living the tropical areas and the dusty areas (mining zones).

CSM data reported by the municipal health management team (MHMT) (2007-2010) stated that CSM causes brain damage emotional and psychological problems which results in low productivity.

Another problem is that the symptoms can be difficult to distinguish from other less serious infections that could result in worsening the situation. Although most people recover from the disease, some are left deaf or blind. Unfortunately, a colossal amount of

money is spent in treatment of the disease annually. These problems lead to wasting of much time at the hospitals and above all causing fears and panic at the social centers in the municipality. As a result of this, it calls for health professionals in the municipality to be alert in their discharge of duties which also increases work load.

1.3 OBJECTIVE OF THE STUDY

The objectives of this thesis are to:

(i) Investigate the epidemiology of meningococcal meningitis in Obuasi municipality.

(ii) Develop a model to predict the spread of CSM in Obuasi using Susceptible, Exposed, Infected and Recovery (SEIR).

(iii) Investigate the impact of vaccination programmes on the control of the disease (CSM) and determine the proportion of the resident in Obuasi that should be vaccinated in order to bring the spread of the disease under control.

1.4 METHODOLOGY

The Simple Susceptible-Exposed-Infective-Recovered

1.5 JUSTIFICATION

(i) The thesis seeks to predict effectiveness of intervention measures instituted to check the spread of CSM disease in Obuasi municipality and assess how helpful those policies instituted could be used to prevent an outbreak to become endemic

(ii) The outcome of the thesis could help to improve upon the welfare of the people, as people recover from the sickness and are able to earn their livelihood and improve their lot

(iii) This thesis would assist the stake holders in the municipality to make future planning in terms of combating an outbreak of infections and area of research

1.6 ORGANIZATION OF THE STUDY

In Chapter 1, we considered the background, problem statement and objective of the study. The methodology and justification were also considered.

In Chapter 2 we shall put forward pertinent related literature on CSM and SEIR. These include various publications, journals and seminars.

Chapter 3 presents the methodology of the study; relevant mathematical tools are given in each segment of the work.

Chapter 4 is devoted for collecting and analysis of data.

Chapter 5, the final chapter presents the discussion of results, conclusions and suggestions for further studies.

CHAPTER 2

LITERATURE REVIEW

2.1 INTRODUCTION

Epidemic meningitis has posed a major health problem since it was first recognized in 1805 (Vieusseux 1806). Epidemics have recurred approximately every 10 years in this century within the African “meningitis belt” of sub-Saharan Africa (Lapeyssonnie 1963, Caugant 1998, Achtman 1990) and until the mid-1980’s within China (Wang *et al.* 1992). Epidemics in other geographical areas are more sporadic and in recent decades, only few industrialized cities have suffered from large epidemics (Caugant 1998), with the notable of mining and unhygienic human activities. In view of that, Obuasi which is the major mining town and densely populated in the country was considered for our research.

In this chapter the spread of communicable diseases such as meningitis have been gainfully studied with mathematical model SEIR. Several researches have attempted to model epidemiology of cerebrospinal meningitis in Obuasi municipality as far the mining is concern. However the compartmental model has not been considered to study the spread of epidemic outbreaks in Obuasi Municipality in spite of the refuse engulf the municipality.

Compartmental model is often used to describe transportation of material in biological systems. A compartmental model contains a number of compartments, each containing a well defined material and compartments exchange material with each other following certain rules.

Disease can occur from one compartment to another, it can be added from the outside through a source, or it can be removed through a drain or a sink. Generally, this material

(disease) represents the amount of something that we wish to account for (disease). To account for the material the model must follow some conservation law. In the model, the compartments will be developed based on conservation of energy which state that energy cannot be created nor destroyed but can changed from one form to another. Since no energy is lost during the transfer, we can calculate the increase and decrease of energy from one compartment to the other.

Most compartment models (as the one shown in Figure 3.1 and 3.2) have more than one compartment and equations, for such models are obtained by following the similar procedure as described by conservational law of energy for each compartment.

According to Kernack and McKendrick (1927) an epidemic, which acts on a short temporal scale, may be described as a sudden outbreak of a disease that infects a substantial portion of the population in a region before it disappears. Epidemics usually leave many members untouched.

Often these attacks recur with intervals of several years between outbreaks, possibly diminishing in severity as populations develop some immunity. Throughout history, epidemics have had major effects on the course of events.

One of the early triumphs of mathematical epidemiology was the formulation of a simple model that predicted behavior very similar to this behavior, observed in countless epidemics. The Kernack and McKendrick (1927), model is a compartmental based on relatively simple assumptions on the rates of flow between different classes of members of the population.

Another important distinction is between epidemics and endemic situations. An epidemic acts on a short time scale and may be described as a sudden outbreak of a disease that infects a substantial portion of the population in a region before it disappears. Epidemics

usually leave many members untouched. In an endemic situation, a disease becomes established in a population and remains for a long time.

In models for epidemics, one usually ignores demographic effects (births and deaths not due to disease) because of the short time scale. The justification for ignoring demographic effects is that the demographic time scale is normally much longer than the disease time scale, and may be neglected. Endemic situations, on the other hand, may endure for years, and it is necessary to include demographic effects in modeling them Dietz (1982).

According to Arino et al., (2007), in the mathematical modeling of disease transmission, as in most other areas of mathematical modeling, there is always a trade-off between simple models, which omit most details and are designed only to highlight general qualitative behavior, and detailed models, usually designed for specific situations including short-term quantitative predictions. Detailed models are generally difficult or impossible to solve analytically and hence their usefulness for theoretical purposes is limited, although their strategic value may be high.

In their example, very simple models for epidemics predict that an epidemic will die out after some time, leaving a part of the population untouched by disease, and this is also true of models that include control measures. This qualitative principle is not by itself very helpful in suggesting what control measures would be most effective in a given situation, but it implies that a detailed model describing the situation as accurately as possible might be useful for public health professionals.

Such a model might have many equations and in practice could only be solved approximately by numerical simulations. This has become feasible in recent years because of the developments in high-speed computing.

David and Jon (2010) stated that, the patterns by which epidemics spread through groups of people is determined not just by the properties of the pathogen carrying it — including its contagiousness, the length of its infectious period, and its severity — but also by network structures within the population it is affecting. The social network within a population — recording who knows whom — determines a lot about how the disease is likely to spread from one person to another. But more generally, the opportunities for a disease to spread are given by a contact network: there is a node for each person and an edge if two people come into contact with each other in a way that makes it possible for the disease to spread from one to the other. This suggests that accurately compartmental modeling is crucial to understanding the spread of an epidemic.

Some of the more recent papers on the mathematical modeling of CSM have included environmental effect Li et al., (2002). They derived a model where humans move through multiple susceptible-Exposed-Infectious-Recovered (SEIR) stages. They introduced dependence of the parameters for the epidemic population sub-model on an environmental parameter (temperature) and calculate the dependence of the reproductive number, for the full epidemic model, on this environmental parameter however the study do not decided when does the calculated dependent reproductive number exceeds unity and the result.

Elif et al., (2010), in this paper, the authors introduced a fractional order SEIR epidemic model with vertical transmission, where the death rate of the population is in density dependent, that is, dependent on the population size. It is also assumed that there exists an infection related death rate. The authors showed the existence of nonnegative solutions of the model, and also a detailed stability analysis of disease free and positive fixed points was also discussed.

The model for CSM is an extension of the equations introduced by Ngwa and Shu (2000), Humans being follows an SEIRS-like pattern and CSM follow SEIR pattern with only one immune class for humans. Humans move from the susceptible to the exposed class at some probability when they come into contact of the infectious person and then to the infectious class. However infectious people can then recover with the aid of vaccination gain immunity; either return to the susceptible class, or move to the recovered class.

Anderson et al., (1986) studied an epidemic such as mumps in United Kingdom. The work was done on virus transmission, herd immunity and the potential impact of immunization. On their findings children are mostly affected by the disease. However vaccination of susceptible population does not confer permanent immunity, but, regards to SEIR model effective vaccination permanent immunity would be attained for a longer period before the vaccination will lose it effectiveness.

Mortality from meningococcal disease was determined during an epidemic in a rural area of Gambia with few medical resources, but where a system of registration of births and deaths had been established before the introduction of a primary health care programme. Thirty three (33) deaths were recorded among one hundred and twenty-seven (127) patients, a case mortality rate of 26%. 84% of deaths occurred within the first 24 hours of illness and many patients died before they could reach any source of treatment because of its treat. Previous studies, based on regional statistics or on hospital series, may have underestimated mortality from epidemic meningitis in Africa. Mortality from this infection will be reduced only if treatment can be made readily accessible to patients early in the course of their illness. Belcher et al., (1977).

Hethcote, (2000), introduced SEIR model to describe the spread of epidemics. According to his studies, the dynamics of the disease depends on infection rate, the removal rate. There is an outbreak of the disease if the reproductive number is exceeds unity. The disease dies out in the susceptible population if the reproductive number is less than one (unity). Moreover, an outbreak of the disease is likely to ensure, if the density of susceptible is high and the removal rate of infection is low. The solution of number of removals depend on infection rate, removal rate, initial number of susceptible and population size. On the other hand the solution of the removal class cannot be used to estimate removals if the outbreak results in large population.

Girard et al. (2006), attest that bacterial meningitis should always be viewed as a medical emergency since it is potentially fatal and treatment must be initiated as quickly as possible in trying to reduce the number of carriers in the community a range of drugs available currently including penicillin G. ampicillin, chloramphenicol and ceftriaxone. In spite of all these drugs the aim was not achieved as the factors influencing the spread of the disease was over sighted. Our thesis unearths types of pathogens that cause CSM and the mode of transmission, which will make the combating to become easily and effective.

WHO (2000), considered, detecting meningococcal meningitis epidemics in highly epidemic Africa Countries as rejected and stigmatize the patients with CSM. Contrary to the report of Hom (1908) it is possible to carry the meningococcal and be infectious while not show any symptoms of infection. Symptoms of CSM were also described by Jacger, M.D. (2000) as suddenly seized with high and rapid rising fever. Sever vomiting which so often accompanies injury to the brain also occurs. Moreover disturbance of

sight and speech, palsy and involuntary urine and dejections complete the picture of malady which eventually ends in death.

Liu z et al., (2003), with the view on general networks with both homogeneous and heterogeneous components and later buttress their opinion that epidemic models have been widely used in different forms for studying epidemiological processes such as the spread of CSM and other communicable diseases. These indications show that our model is useful not only for the study of CSM bacterial, but also of other infectious diseases which have quite different natural features and histories.

Bernoulli, (1760) developed a mathematical model of the impact of vaccination against CSM in, and since then there has been an increasing number of mathematical epidemiology published. Using mathematical models, we gain a better understanding of the dynamics of the infection and some of the underlying features of infections that may not be easily observed, for example the carriage state of CSM bacteria, where people can be infectious but shows no outward signs of infection(exposed). These people could play a key role in aid the control of an epidemic, yet in practice it is hard to identify the role they have in the epidemic process-hence there is a need for models that allow us to take these aspects into consideration to better implement control measures.

Diekmann and Heesterbeek, (2000) a predictive threshold is often found through the study of the eigenvalues of the Jacobian at the disease-free equilibrium. This is a simple, widely used method for ordinary differential equation systems. Using this method, a parameter is derived from the condition that all of the eigenvalues of the Jacobian have a negative real part. This can easily be done using the characteristic polynomial and the Routh–Hurwitz stability conditions.

The Jacobian method clearly allows us to derive a parameter that reflects the stability of the disease-free equilibrium. The parameter obtained in this way, however, may or may not reflect the biologically meaningful value of

transmissions by trying to practice proper sanitation, ventilation and avoidance of overcrowding especial those infected.

Anderson et al., (1983) attest on vaccination against epidemics that, simple and deterministic mathematical models were used to examine impart that vaccination policies have on age-specific incidence of CSM and other tropical diseases.

Four specific antigens related to serogroups A, C, Y and W135 are currently available. They are distributed in freeze-dried form, injectable by IM route, either as bivalent AC vaccine, or quadrivalent A, C, Y, W135 vaccine, containing 50 µg of each antigen.

Meningococcal A, C, Y, W135 vaccines are based on capsular polysaccharide antigens and induce a T cell-independent immune response which is age dependent. In adults and in children above 4 years of age, a single dose induces a rapid rise of antibodies and protection within 10 days in over 85% of recipients. Protection lasts for at least one year and often several years longer. The response is poorer in infants and young children.

The group A vaccine is more immunogenic than some other polysaccharide vaccines and it can induce an appreciable antibody concentration even in three-month-old infants. These polysaccharide vaccines are generally very well tolerated but may induce some mild adverse reactions (local pain and swelling, fever and malaise) in 10-20% of recipients, for the two-three days following the vaccination.

Gotschlich et al., (1969) described the development of highly immunogenic serogroup A and C meningococcal vaccines based on purified meningococcal capsular polysaccharides. The following year it was reported that a serogroup C meningococcal polysaccharide vaccine gave a high degree of protection against serogroup C meningococcal meningitis in American military recruits (Artenstein et al., 1970) and a

serogroup A polysaccharide vaccine was soon shown to be equally effective in preventing serogroup A meningococcal disease in Egypt (Wahdan et al., 1973), the Sudan (Erwa et al., 1973), and Upper Volta (Ettori et al., 1977). Subsequently, serogroup A + C meningococcal polysaccharide vaccines have been used extensively in Africa where they have been shown to be very effective at bringing epidemics rapidly under control (Greenwood, 1999). However, meningococcal polysaccharide vaccines are poorly immunogenic in young children and do not induce long-lasting, T cell-dependent immunological memory (Reingold et al., 1985). Furthermore, meningococcal polysaccharide vaccines do not seem to reduce the prevalence or incidence of nasopharingeal carriage of serogroup A or C meningococcal, as was found in studies conducted in Nigeria (Blakebrough et al., 1983) and The Gambia (Hassan-King et al., 1988).

CHAPTER 3

METHODOLOGY

3.1 INTRODUCTION

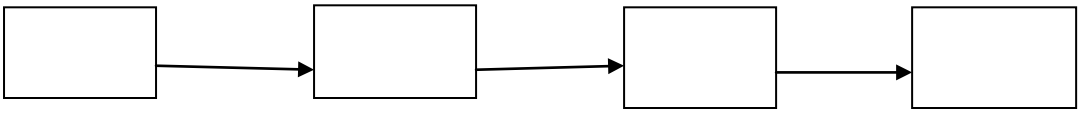
In this chapter a mathematical model that governs the epidemiology of

polluting the water bodies and vegetation. In spite of several measures that have been instituted in Obuasi i.e. environmental and social concerns raised to enhance sanitation and combating epidemics by the mining companies, the activities of illegal and surface mining do not make its achieve the desire objective thus eradicate the epidemics of CSM.

The issue of corporate review of environmental performance was undertaken in Obuasi in 2005, its primary concern was on sanitation by the mining companies. It looked particularly at the discharging of mining liquid waste into the water bodies and ensuring rehabilitation of an area for safe keeping of other solid waste. There is quarterly mass spraying and vaccination exercise in Obuasi to prevent malaria and combating epidemics for every year. We have included the recorded trend of spread of cerebrospinal meningitis for the past four years, with data from the Municipal Health Meningitis Team (MHMT), (2011).

3.2 DESCRIPTION FOR

infection), in most of the infections, there is a significant period of time during which the individual have been infected but not yet infectious. During this latent period, the individual is in the exposed



3.3 ASSUMPTIONS OF THE MODEL

The disease spreads in a closed environment; that is there is no emigration or immigration assume that; assume that (birth and death are equal in the population)

reproductive number below the level which would allow an epidemic to start. In Ghana in particular, the CSM vaccine is administered to the densely populated area and more especially in dusty, mining areas and hot temperature regions.

Anderson and May, (1981) described mathematical model to reduce tropical communicable diseases such as CSM, mumps, chicken pox etc, they gave their view about the importance of vaccination as guarantees for life and vaccinated babies are considered as recovered. The section of residents in Obuasi who were not vaccinated during the vaccination period (at birth and mass vaccination) is exposed to infection.

The rate at which the susceptible comes in contact with the disease depends solely on contact rates thus new infection is proportional to the susceptible

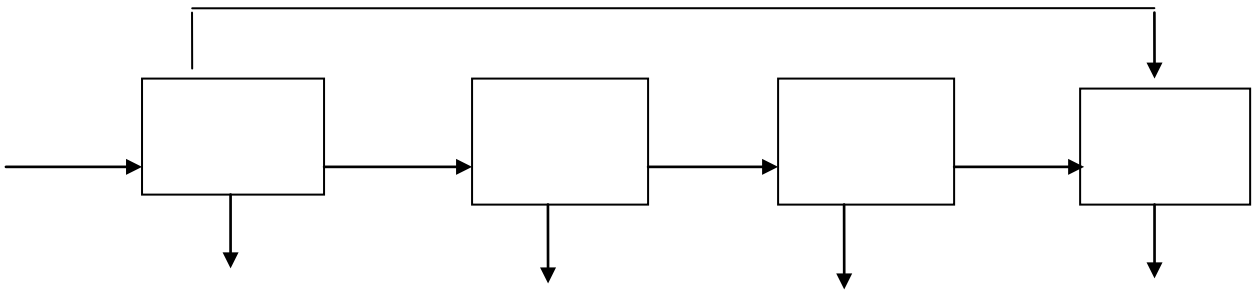


Table 3.1 Model parameters and its interpretations

Parameters	Description	Estimates	Source

expressing equation (3.11) – (3.14) as a proportion of the population we divide through by the total population to obtain,

risk of an epidemic or pandemic in emerging infectious disease. For example, the estimation of

susceptible–exposed– infectious–recovered

3.9 EFFECTIVE REPRODUCTIVE NUMBER (E_R)

Effective Reproductive Number, denoted

found are disease-free(where

=

CHAPTER 4

MODEL ANALYSIS AND DISCUSSION OF RESULTS

4.1 INTRODUCTION

This chapter deals with the analysis of the model and the discussion of the results obtained. We use MatLab to run our simulations. For our systems of nonlinear differential equations, we used the ordinary differential equations (45) which is a fourth order variable Runge-Kutta method.

Sensitivity analysis is performed on the parameter values to determine the effect of these values on the rate of spread of CSM

4.2 DISCUSSION OF RESULTS

In our thesis, we used standard SEIR differential equation model to predict the spread of CSM in Obuasi municipal of Ghana. We discussed the existence and stability of the disease free and disease endemic equilibrium of the model and performed sensitivity analysis of the parameters. We also considered Herd immunity and Control vaccination Number

From Figure 4.1 we noted that, during the initial days, there is a very sharp drop in the population of susceptible individuals, while other graphs (exposed and infectious) particularly increased. The sharp rise and fall after the 50^{th} to 200^{th} days in the population of exposed could be attributed to complacency on the part of some individuals or may be due to oscillations in the system independent of external factors. However in Figure 4.3, there is a gradual rise from day 100^{th} which happens as a result of people not taking a serious look of the disease that ends the increment of the population of infectives.

We find out that people tend to relax after the initial shock of the disease threat. However, the recovered proportion took almost half of the year to increase their number as a result of populace negligence of the disease CSM. With the intervention of vaccination programs the recovered population increase each of the graph also we notes that the increasing could be attributed to the fact that vaccination exercise was effective and continue to rise in the various graphs, so as people continue to receive vaccination, infection is controlled.

Furthermore, the simulation in figure 4.1-4.8 indicate that the recovered population increase as the initial proportion of infectives reduces with the aid of education and sensitization of the population on the effects and prevention of CSM vaccination intervention programmes,

The perturbation analysis of the disease free equilibrium revealed that when the reproductive number

model are

(ii) Increase in initial proportion of susceptible, exposed and infectives in the population.

To depict the dynamics of the compartments during the outbreak, we assume the initial proportions of susceptible, exposed, infectives and the recovered on the table below.

Table 4.1. Initial values of SEIR

Susceptible (s)	Exposed (e)	infective (i)	Recovered (r)
100	48	20	0
98	20	15	0

Graphs depicting the dynamics of the compartments during the outbreak

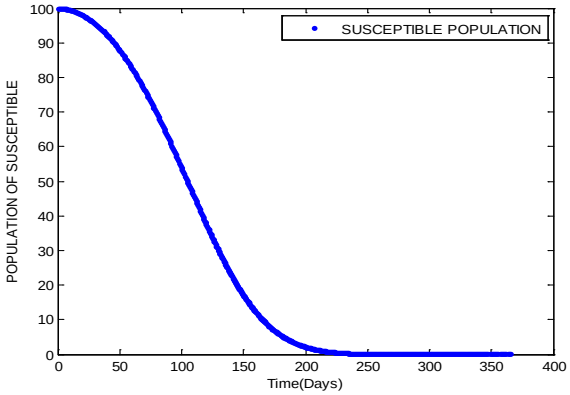


Figure 4. 1 Evolution of Susceptible population per year in Obuasi

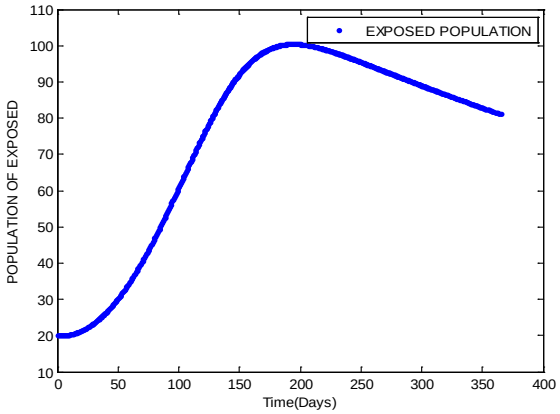


Figure 4. 2 Evolution of Exposed population per year in Obuasi

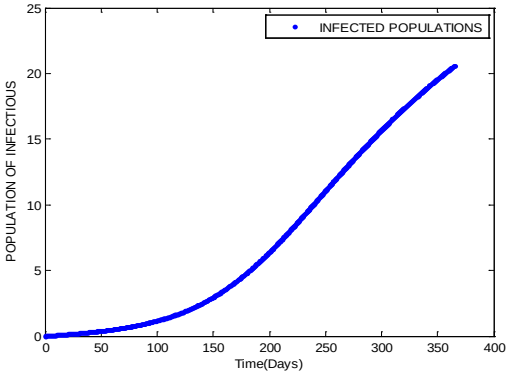


Figure 4. 3 Evolution of Infectious population per year in Obuasi

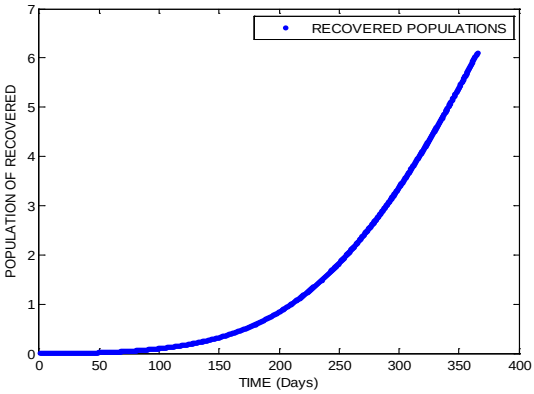


Figure 4. 4 Evolution of Recovered population per year in Obuasi

4.4 GRAPHS OF SEIR WITH VACCINATION

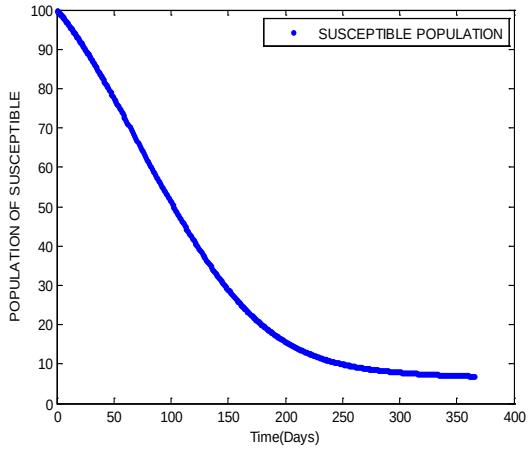


Figure 4. 5 Evolution of Susceptible population per year in Obuasi

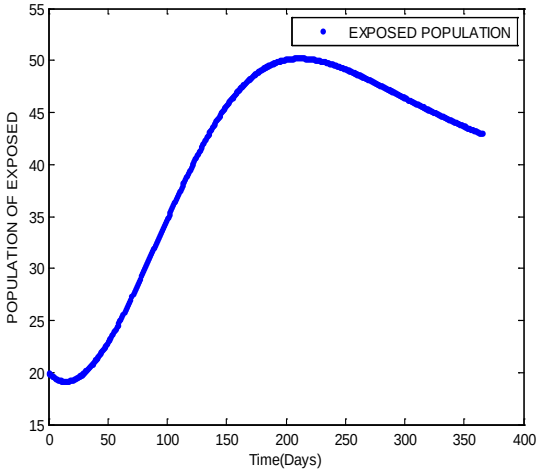


Figure 4. 6 Evolution of Exposed population per year in Obuasi

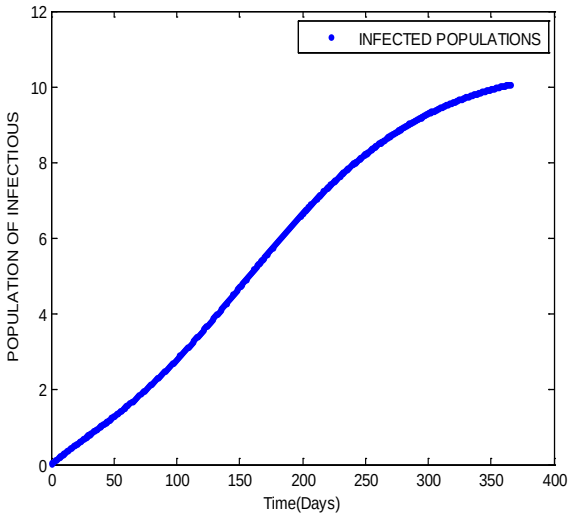


Figure 4. 7 Evolution of Infected population per year in Obuasi

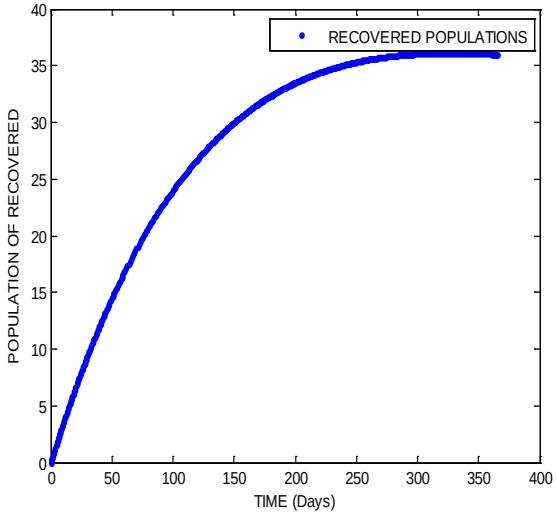


Figure 4. 8 Evolution of Recovered population per year in Obuasi

4.5 GRAPHS OF REDUCED PARAMETER OF SEIR WITH VACCINATION

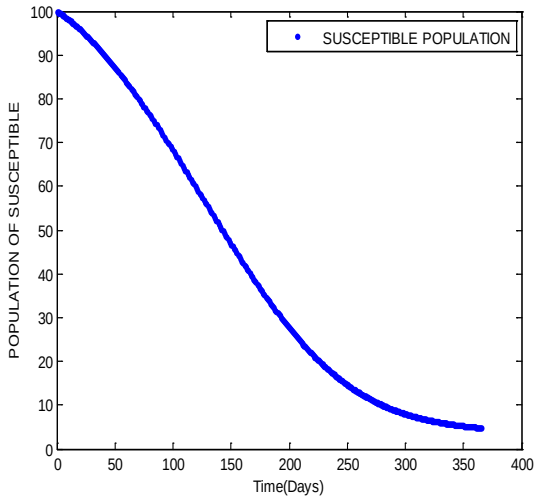


Figure 4. 9 Evolution of Susceptible population per year in Obuasi

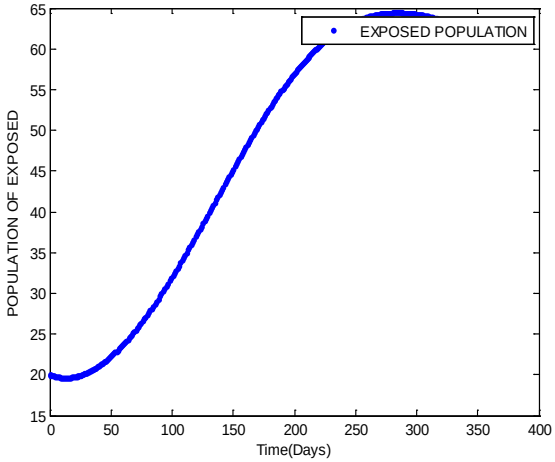


Figure 4. 10 Evolution of Exposed population per year in Obuasi

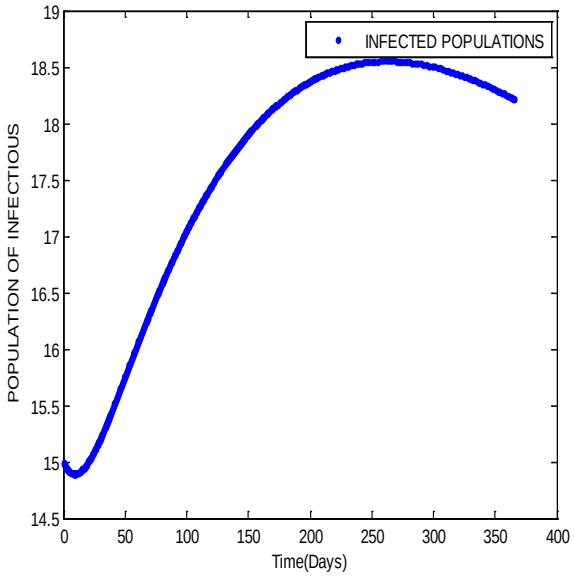


Figure 4. 11 Evolution of Infectious population per year in Obuasi

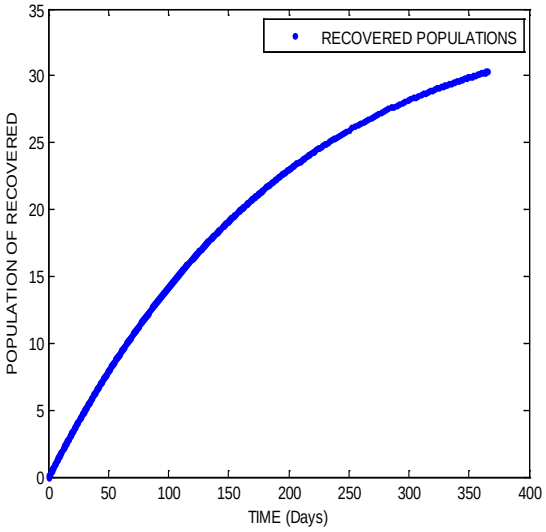


Figure 4. 12 Evolution of Recovered population per year in Obuasi

From figure 4.1, shows the initial proportional of susceptible is 100% thus, the outbreak of CSM declines gradually from the apex (100%) level to asymptotic within 0-365 days. Figure 4.2 begins with 20% of the population and rises to 65%. The exposed cannot start from all the populace due to different immune system among other members. Figure 4.3 shows infectious begins at a gradual rate from 0^{th} day until day 50^{th} where 18.5% of the population were infected until the end of the year, then the population later reduced at the end of 365 days. Recovered starts increasing from day 0^{th}, but was not effective until day 100^{th}. Considering figure 4.5 to 4.9 which depict the graphs of SEIR with vaccination has similar features as figure 4.1-4.4 as far as susceptibility and exposure are concerned figure 4.6 which starts from 20% and begin to rise up to 50% within the period of 365 days of the population but with the aid of vaccination interventions it could not proceed the rising but decline. Infectives in figure 4.6 started at day 0^{th} with exponential increasing to 10% of the population it started reducing the number of infectives because the populace were enlighten to the symptoms and how to combat the disease.

In spite, of reduction on parameters which retard the rate of spreading meningococcal meningitis the graphs were similar to that of vaccination. However, each proportion of the susceptible, exposed in infectious and recovered proportion of the population attains different peak values time.

4.6 FORCE OF INFECTION

From equation (3.1) the force of infection which is

This implies that, the number of infection rate in the mist of CSM patient among susceptible is

That is less than 8% of the population in Obuasi would be susceptible for

From the numbers generated by the analysis on the table, stake holders can predict the covered areas that will allow them to measure the effectiveness of the policies and decide if occurrence of the disease is increasing, decreasing, or stable.

4.12 THE HERD IMMUNITY THRESHOLD (

This implies that the presence of a person infected with CSM in Obuasi will eventually result in an outbreak of the disease. We will later consider the effect of the changes in the parameter values of the stability of the equilibrium its phase portrait is shown below.

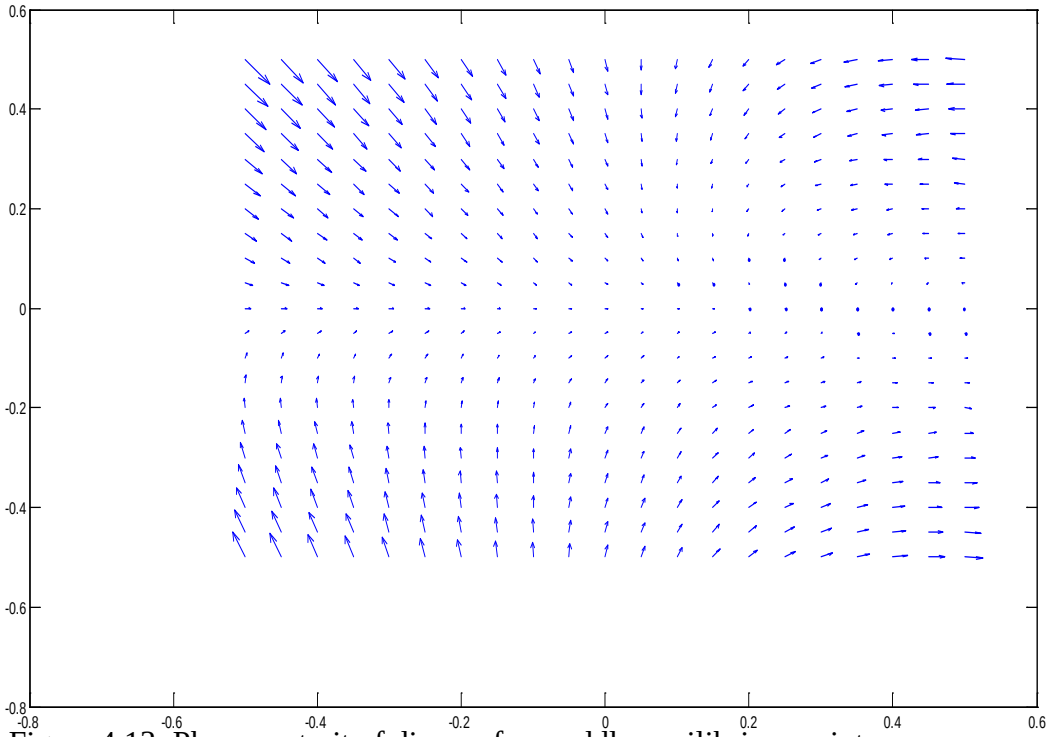


Figure 4.13: Phase portrait of disease free saddle equilibrium point

4.14 STABILITY OF THE ENDEMIC EQUILIBRIUM.

At the point in time where all the compartments of the population coexist is called an endemic period. The presence of an infected person is a problem in the epidemiology of infectious disease. We study the behavior of this endemic equilibrium point

The characteristic equation obtained from the above Jacobian matrix is of the form,

4.16 SENSITIVITY ANALYSIS OF AN ENDEMIC EQULIBRIUM STATE WITH INCREASE PARAMETERS.

The parameters of table (3.1) has been increased as follows,

4.17 SENSITIVITY ANALYSIS OF AN ENDEMIC EQULIBRIUM STATE WITH DECREASE PARAMETERS.

The parameters of table (3.1) have been increased as follows,

CHAPTER 5

CONCLUSION AND RECOMMENDATIONS

5.1. INTRODUCTION

In this chapter we deal with the conclude and give necessary recommendation for further studies about the results obtained from chapter four

5.2. CONCLUSIONS

The derivation and analysis of the modified

About 50% of the susceptible population should be immune or covered during vaccination in order not to have an epidemic during an outbreak.

The simulation result of the study confirmed the transmission rate and recovery rate as the dominant parameters in the spread of the disease in Obuasi municipality

5.3. RECOMMENDATIONS

Further research work is recommended particularly for non-constant and heterogeneous population and also on vaccination.

Vaccination programmes should be intensify by the ministry of health and should target at vaccinating about 50% of the susceptible population in order to fully bring the disease under control where an outbreak is considered epidemic.

Proper sanitation should be ensured, especially there should be proper and safe keeping of the mining liquid and gaseous waste. Mining activities should be operated far from the Obuasi municipality. Miners should observe safety rules that check an outbreak of epidemic.

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APPENDIX

MATHLAB CODES

M-FILE FOR CSM WITHOUT VACCINATION

```
function dy=model(t,y,beta,gamma,epislon)
dy=zeros(4,1);
dy(1)= -beta*y(1)*y(3);
dy(2)= beta*y(1)*y(3)-epislon*y(2);
dy(3)= nu*y(2)-gamma*y(3);
dy(4)= gamma*y(3);
```

SCRIPTS FOR CALLING M-FILE WITHOUT VACCINATION

```
gamma=0.04;

beta=0.02;

epislon=0.001

options = odeset('RelTol',1e-9,'AbsTol',1e-9); [t,y] = ode45(@seir,[0 365],[1.00
0.09 0.03 0.00],options, beta,gamma,epislon);
figure(1)
plot(T,Y(:,1),'.')
legend('SUSCEPTIBLE POPULATION')
xlabel('Time(Days)');ylabel('POPULATION OF SUSCEPTIBLE');
figure(2)
plot(T,Y(:,2),'.')
legend('EXPOSED POPULATION')
```

```
xlabel('Time(Days)');ylabel('POPULATION OF EXPOSED ');
figure(3)
plot(T,Y(:,3),'.')
legend('INFECTED POPULATIONS')
xlabel('Time(Days)');ylabel('POPULATION OF INFECTIOUS');
figure(4)
plot(T,Y(:,4),'.')
legend('RECOVERED POPULATIONS')
xlabel('TIME (Days)');ylabel('POPULATION OF RECOVERED');
```

M-FILE FOR CSM WITH VACCINATION

```
function dy=model(t,y,beta,gamma,epsilon,alpha, ,mu,)
dy=zeros(4,1);
dy(1)=beta-y(1)*(alpha)*y(3)-(mu-rho);
dy(2)=alpha*y(1)*y(3)-y(2)*(mu+epsilon);
dy(3)=epsilon*y(2)-y(3)*(gamma+mu);
dy(4)=gamma*y(3)-mu*y(4)+rho*y(1);
```

SCRIPTS FOR CALLING M-FILE WITH VACCINATION

```
rho = 0.00977;
alpha =0.0275;
mu =0.02293;
epsilon = 0.004916;
beta = 0.030417;
```

```
gamma =0.02293;

epislon =0.021429;

options = odeset('RelTol',1e-9,'AbsTol',1e-9); [t,y] = ode45(@seir,[0 365],[1.00

0.09 0.03 0.00], options, beta, gamma, nu, delta, kappa, alpha, mu, epsilon);

figure(1)

plot(t,y(:,1),'.')

figure(1)

plot(T,Y(:,1),'.')

legend('SUSCEPTIBLE POPULATION')

xlabel('Time(Days)');ylabel('POPULATION OF SUSCEPTIBLE');

figure(2)

plot(T,Y(:,2),'.')

legend('EXPOSED POPULATION')

xlabel('Time(Days)');ylabel('POPULATION OF EXPOSED ');

figure(3)

plot(T,Y(:,3),'.')

legend('INFECTED POPULATIONS')

xlabel('Time(Days)');ylabel('POPULATION OF INFECTIOUS');

figure(4)

plot(T,Y(:,4),'.')

legend('RECOVERED POPULATIONS')

xlabel('TIME (Days)');ylabel('POPULATION OF RECOVERED');
```