A MATHEMATICAL MODEL FOR MEASLES TRANSMISSION DYNAMICS IN
LUWEERO DISTRICT OF UGANDA

BY

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APRIL 2019
DECLARATION

I Kiyimba Quraish declare that this research report is my original work. It has not been submitted to any other University or higher institution for any award.

Signature ................................................ Date 1/4/2019

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APPROVAL

I hereby certify that this work entitled A Mathematical Model for Measles Transmission Dynamics in Luweero District of Uganda has been submitted with the approval of my university supervisor.

Signature .................................. Date: 1st April 2019

MADAM TALIGOOLA DEBORAH

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DEDICATION

To my beloved dad Mr. Kiyimba Yahaya, mum (Ms Nassimbwa Jane) for their financial support and encouragement towards my education. May the lord bless you.
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I thank the lord almighty for keeping, protecting and giving me sound mind and endurance during the course of my study.

I would like to appreciate the work of my supervisor madam Taligoola diligently guided me during my research. May the Almighty reward her abundantly.

Special thanks go to my parents for their love and support in form of school fees for my education.

I would like to appreciate my beloved brother and sisters; Razin, Shamirah, Rania, Radhia, Nadia. Nabila for their encouraging company and support. It was key for my success. My friends Sharif, Abdu, Ronald, Julius, Ocen, Dasan, Daniel, Hanifah, for their incredible contribution towards my education.

I also wish to thank my lecturers for their advice, knowledge and guidance towards my education. May the good God bless them.

Finally I take this opportunity to acknowledge the efforts of my respondents who availed all the necessary data during this research study.
ABSTRACT

In this research work, Mathematical Model for Measles Transmission Dynamics in Luweero District of Uganda, SVEIR model was developed and analyzed. The model consists of five non linear ordinary differential equations. The effective reproductive number, (the number of secondary infections when a single effective individual is introduced into a population where a proportion is protected) was obtained. Further the disease free and endemic equilibrium where obtained and analyzed for stability. Numerical simulation of the various state variables where obtained using mat lab software. And it shows that the vaccination is capable of reducing the number of susceptible when the coverage is high.
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CHAPTER ONE

1.0 The Introduction.
This chapter expounded on the background of the study, statement of the problem, objectives of the study, significance and scope of the study.

1.1 Background of the study
Measles is an airborne disease that affects mainly children aged between 6 months to 10 years although it can also affect adults. It is spread through air and contact with an infected person. Some early symptoms of measles include high fever, hacking cough, and red swelling eyelids, muscle and body aches, irritability, running nose, watery eyes and finally rashes. Approximately 20 percent of people with measles develop one or more complications associated with the disease. These early symptoms of measles usually last three to four days, although they can last as little as one day or as long as eight days, before the measles rash begins. One to two days before the rash appears, small red lesions with blue-white centers appear on the inside of the mouth and tongue, and occasionally on the whites of the eyes or inside the intestines.

The spread of measles in Luweero sub regions has been attributed to a number of factors. The ministry of health says that some of these factors include the high influx of refugees from neighboring DR Congo, religious cults like the Triple 6, cultural attachments and attitudes towards health services. Religious cults in Luweero have also been blamed for stopping unsuspecting parents from immunizing their children against the killer diseases.(serwadda, 2016).

Measles is highly contagious and remains a leading cause of childhood mortality. In the pre-vaccine era an estimated 130 million cases occurred each year. At the World Health Assembly in 2005, an ambitious global goal for measles control was established as part of the Global Immunization Vision and Strategy document that is, to achieve a 90% reduction in measles mortality by 2010 compared with 2000. Information on measles disease, its differential diagnosis, transmission, immunity and the vaccine can be found in the WHO Module on best practices for measles surveillance the WHO position paper on measles vaccine, and the WHO immunological basis for immunization measles module.
Current WHO guidance on measles outbreak response published in 1999 emphasized that most outbreaks were either detected too late or spread too rapidly to allow for an effective immunization response. This recommendation was based primarily on a literature review of manuscripts published on the impact of immunization control activities on measles outbreaks in middle- and low-income countries from 1977 to 1993. The validity of these recommendations was reviewed given recent evidence published and from filed experience on the impact of outbreak response immunization on measles outbreaks. This review of the evidence was carried out as follows:

1.2 Problem statement
Despite the effort and progress made by the world health organization and the ministry of health in Luweero district to eradicate measles, it has rebounding in Luweero district since 2004. The health sector reported an increasing number of measles cases since April 2015. And the district requested for an assistance to control the spread of this viral infection. Measles is a very dangerous viral infection on human health; it causes muscle pains, whole body fever, and malaise, loss of appetite, too much sneezing dry cough and terrible skin rush. It is from these alarming effects that the researcher seeks to find outs the remedy of this infection. And to dispose its impacts in Luweero district central Uganda.

1.3 The purpose of the study
The purpose of this research study is to identify the transmission dynamics of measles in Luweero district central Uganda and to examine the effects of vaccination via mathematical modelling.

1.4 Objectives of the study
The objectives of the study are as follows

1. To establish the causes and effects of measles infection
2. To establish the symptoms of measles infection
3. To know how measles can be cured and prevented
4. To develop the mathematical model for the dynamic of the disease.

5. To obtain the equilibrium state of the model.

6. To analyze the equilibrium state for stability.

7. To obtain the basic effective reproductive number

1.5 Research questions

1. What are the causes and effects of measles infection?
2. What are the symptoms of measles infection?
3. How can measles be cured and prevented?
4. What is the mode and rate of transmission of measles?
5. What is the equilibrium state of the model?
6. What is the stability of the equilibrium state?
7. How do you obtain the basic effective reproductive number?

1.6 Significance of the study

The study about the impacts and causes as well as the control measures of measles infection is very relevant to various groups and individuals not only in Uganda but in the world at large.

To begin with this study will help the national bureau of statistics to know the virology and medical background of measles as necessary for national health evaluation specifically on this disease.

The survey will assist the ministry of health in determining measures to solve and prevent the spread of diseases and infections related to measles in the region.

The study will benefit students in Kampala International University, and the general public at large as it will add to existing work in their library on measles and infectious disease. This research work will also have a significant role in the community toward awareness and public
caution about the spread of the infections, the government therefore can rely on these findings to help the groups and individuals concerning this disease.

1.7 Scope of the study

1.7.1 Contextual scope

Contextually, this research work is focus on investigating the impacts of measles epidemics, its causes and symptoms plus its prevention measures in Luweero district.

1.7.2 Geographical scope

Luweero district is located in central Uganda, bordered by Kampala district in the north, Kalangala district in the south, Mukono district in the west and Nakaseke district in the east. It is located at approximately 100km from Kampala.

1.7.3 Time scope

This study will be carried out in the period of six months that is from October 2018 to April 2019.
2.0 Virology and medical background

2.1.1 Virology

Viruses are obligate intracellular parasites. To understand the complexity of virus replication and to develop potent antiviral drugs, there is a growing need for a systematic analysis of the underlying molecular mechanisms. Although the kinetics of protein expression has been established for many viruses and a model of the dynamics of virus transcription and replication for bacteriophage Q has been proposed, knowledge in this area is very limited for all eukaryotic viruses. The researcher has found out that the first kinetics model of the transcription and replication of a negative-sense, single-stranded RNA virus.

Measles virus (MV) belongs to the order Mononegavirales. The replication strategy involves a viral RNA-dependent RNA polymerase (vRdRp), which uses as a template a nucleocapsid (NC) made of a single strand of RNA in tight complex with the nucleoprotein (N). The negative-strand genome contains six transcription units encoding the N, phospho (P), matrix (M), fusion (F), hemagglutinin (H), and large (L) or polymerase protein, in that order. The transcription units are flanked by short leader (Le) and trailer (Tr) sequences containing the genomic promoter (on the minus strand) and the antigenomic promoter (on the plus strand), respectively. The P gene also encodes the nonstructural V and C proteins by RNA editing and alternative overlapping open reading frame, respectively.

Schematic view of MV genomic organization, main RNA species, and nucleotide positions of gene starts and gene ends 15,894-nucleotide-long genome. Sense and antisense primers with their first nucleotide positions are indicated by black and grey arrows, respectively. Antisense primers above mRNAs and above antigenome are identical.

Every N protein binds to 6 nucleotides so that the N polymer entirely covers the 15,894-nucleotide genome and makes it resistant to nucleases. NC comprises 2,649 N, ~300 P, and ~20 to 50 L proteins and is enclosed within a membrane envelope containing H and F glycoproteins. After fusion with the plasma membrane at neutral pH, intact NCs are released into the
cytoplasm, where they serve as a template for both primary transcription from and replication of the genomic RNA. Transcription is initiated from a single promoter at the 3' end of the genome (for a review, see reference by vRdRp made of L and P proteins. Immediately prior to an intergenic junction, the transcriptase recognizes a gene end signal and terminates the synthesis of the upstream mRNA. Then, it either recognizes the gene start (GS) signal of the downstream gene and begins the synthesis of mRNA or fails to do so and detaches from the template. As a consequence, there is an attenuated gradient of viral transcription. Sometimes, the transcriptase fails to recognize the intergenic junction, and read-through leads to the generation of bicistronic mRNAs. Following an ill-defined signal, the vRdRp switches to a replicative mode and uses the upstream replication genomic promoter to synthesize a full-length positive-sense RNA strand. Concomitant encapsidation during RNA elongation probably explains the rarity of viral recombination events observed within the order Mononegavirales and makes the RNA resistant to small interfering RNA (RNA interference silencing). NC likely associates with cellular (co)factors, which contribute to the overall vRdRp functionality.

The homo-oligomeric P protein acts as a bridge between L and the NC template P protein also binds newly synthesized, monomeric forms of the 60-kDa N protein (N°) to form a soluble (N°-P) complex. The N°-P complex is thought to be the substrate used by the vRdRp to initiate the encapsidation of the nascent RNA chain during replication.

Study of the functions and regulations of the vRdRp has been hampered so far by the lack of a convenient in vitro acellular or cellular RdRp assay and by our very poor knowledge of the kinetics of the vRdRp during virus infection. By using reverse transcription-quantitative PCR (RT-QPCR), we reliably quantify MV RNA species and describe the kinetics of their accumulation during the infection.

2.1.2 Medical back ground

Measles is a respiratory disease caused by a virus. The disease and the virus that causes it share the same name. The disease is also called rubeola. This virus normally grows in the cells that line the back of the throat and lungs.
The virus is an enveloped virus (100–200 nm in diameter), with a core of single-stranded RNA. Two membrane envelope proteins are important in pathogenesis. They are the F (fusion) protein, which is responsible for fusion of virus and host cell membranes, viral penetration, and hemolysis, and the H (hemagglutinin) protein, which is responsible for adsorption of virus to cells. There is only one antigenic type of measles virus. The virus is rapidly inactivated by heat, light, acidic pH, ether, and trypsin. It has a short survival time (less than 2 hours) in the air or on objects and surfaces.

It is very rare in countries and regions of the world that are able to keep vaccination coverage high. In North and South America, Finland, and some other areas, endemic measles transmission is considered to have been interrupted through vaccination. There are still sporadic cases of measles in the United States because visitors from other countries or US citizens traveling abroad can become infected before or during travel and spread the infection to unvaccinated or unprotected persons. Worldwide, there are estimated to be 20 million cases and 164,000 deaths each year. More than half of the deaths occur in India. People who do not have immunity to it (not vaccinated or who never had it before) are at an increased risk of being infected when exposed to a person with it.

2.2 Transmission, signs and symptoms

2.2.1 Transmission

The virus is highly contagious and can be spread to others from four days before to four days after the rash appears. It is so contagious that if one person has it, 90% of the people close to that person who are not immune will also become infected.

The virus lives in the mucus in the nose and throat of the infected person. When that person sneezes or coughs, droplets spray into the air. The droplets can get into other people’s noses or throats when they breathe or put their fingers in their mouth or nose after touching an infected surface. The virus can live on infected surfaces for up to 2 hours. Measles is a disease of humans; the virus is not spread by any other animal species.
2.2.2 Signs of measles

The measles vaccine is widely available and is said to have dropped global rates of measles by over 75 percent. Complications from measles are fairly common. Some can be serious. People most at risk are patients with a weak immune system, such as those with HIV, AIDS, leukemia, or a vitamin deficiency, very young children, and adults over the age of 20 years. Older people are more likely to have complications than healthy children over the age of 5 years.

Signs can include:

- Diarrhea
- Vomiting
- Eye infection
- Respiratory tract infections, such as laryngitis and bronchitis
- Difficulty breathing
- Ear infections, which can lead to permanent hearing loss
- Febrile seizures

Patients with a weakened immune system who have measles are more susceptible to bacterial pneumonia. However this can be fatal if not treated. The following less common complications are also possible:

Hepatitis: Liver complications can occur in adults and in children who are taking some medications.

Encephalitis: This affects around 1 in every 1,000 patients with measles. It is an inflammation of the brain that can sometimes be fatal. It may occur soon after measles, or several years later.

Thrombocytopenia, or low platelet count, affects the blood’s ability to clot. The patient may bruise easily.

Squint: Eye nerves and eye muscles may be affected.
2.2.3 Symptoms

The symptoms of measles generally begin about 7-14 days after a person is infected, and include: blotchy rash, fever, cough, runny nose, red, watery eyes (conjunctivitis), feeling run down, achy (malaise), tiny white spots with bluish-white centers found inside the mouth (Koplik’s spots). A typical case begins with mild to moderate fever, cough, runny nose, red eyes, and sore throat. Two or three days after symptoms begin, Koplik’s spots may appear inside the mouth. Three to five days after the start of symptoms, a red or reddish-brown rash appears. The rash usually begins on a person’s face at the hairline and spreads downward to the neck, trunk, arms, legs, and feet. When the rash appears, a person’s fever may spike to more than 104 degrees Fahrenheit. After a few days, the fever subsides and the rash fades.

2.3 Diagnosis, treatment and prevention.

2.3.1 Diagnosis

The symptoms of acute measles are so distinctive that laboratory diagnosis is seldom required. However, as the vaccination program progresses, atypical forms of measles have emerged and laboratory diagnosis may be required.

Microscopy - production of multinucleate giant cells with inclusion bodies is pathognomonic for measles. During the prodrome phase, such cells are detectable in the NPS (nasopharyngeal secretions). This is more rapid and practical than virus isolation.

Immuno fluorescence - direct and indirect immune fluorescence have been used extensively to demonstrate MV antigens in cells from NPS specimens. This technique can also be applied to the urine as such cells may be present in the urine 2 to 5 days after the appearance of the rash. (Although like mumps, measles virus is also excreted in the urine, this route is unlikely to play a significant role in the spread of the virus infection.)

Virus isolation - measles virus can be isolated form a variety of sources, e.g. throat or conjunctival washings, sputum, urinary sediment cells and lymphocytes. Primary human kidney (HEK) cells are the best, although primary monkey kidney can be used as well. Continuous cell
lines such as vero cells can also be used although they are not as efficient as primary cell lines. A CPE develops between 2 to 15 days, and consist of either a broad syncytium or a stellate form with inclusion bodies visible. The presence of measles can be confirmed by haemadsorption. In acute measles, the isolation rate is difficult and the success rate is low. Isolation is most likely to be successful from material taken in the prodrome phase but not in the later stages after the rash has developed. Therefore isolation should only be attempted in complicated cases such as suspected SSPE where the lymphocytes may carry the virus, and in immunocompromised individuals developing pneumonia.

**Serology** - diagnosis of measles infection can be made if the antibody titres rise by 4 fold between the acute and the convalescent phase or if measles-specific IgM is found. The methods that can be used include HAI, CF, neutralization and ELISA tests. Neutralization tests are the most sensitive but are not practical to perform. CFTs have a reduced sensitivity and thus are not useful for immune status screening.

**Diagnosis of SSPE** - the presence of measles specific antibodies in the CSF is the most reliable means of laboratory diagnosis of SSPE. Demonstration of MV-specific antibodies in the CSF may be sufficient with, if necessary, demonstration of MV-specific restricted heterogeneity by is electric focusing. Virus isolation from SSPE brain tissue is complicated. Alternately, brain biopsy material can be examined microscopically for inclusion bodies and virus antigen by immune fluorescence.

Syncytial formation caused by measles virus in cell culture (Courtesy of Linda Standard, University of Cape town, S.A.)

### 2.3.2 Treatment of measles

Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A
doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

People should avoid smoking near the child.

Sunglasses, keeping the lights dim or the room darkened may enhance comfort levels, as measles increases sensitivity to light.

If there is crustiness around the eyes, gently clean with a warm, damp cloth.

If there is crustiness around the eyes, gently clean with a warm, damp cloth. Cough medicines will not relieve a measles cough. Humidifiers or placing a bowl of water in the room may help. If the child is over 12 months, a glass of warm water with a teaspoon of lemon juice and two teaspoons of honey may help. Do not give honey to infants.

A fever can lead to dehydration, so the child should drink plenty of fluids.

A child who is in the contagious stage should stay away from school and avoid close contact with others, especially those who are not immunized or have never had measles.

Those with a vitamin A deficiency and children under 2 years who have measles may benefit from vitamin A supplements. These can help prevent complications, but they should only be taken with a doctor's agreement. If you want to buy vitamin A supplements, then there is an excellent selection online with thousands of customer reviews.

Antibiotics will not help against the measles virus, but they may sometimes be prescribed if an additional bacterial infection develops.

2.3.3 Prevention

With no animal reservoir, it must be possible to eradicate the virus through a controlled vaccination campaign. In the USA, where vaccination of all children is required before
commencing school, case reports have fallen by over 99% but eradication has not been achieved. The following vaccines are available

**Inactivated Vaccine**- this vaccine was intended for use in young children less than 1 year most prone to severe complications. It was thought to be advisable to avoid the use of a live vaccine. It was found that at least 3 doses were needed to elicit a protective antibody response but the antibody levels soon waned. This leave the vaccines open to attack by the natural virus. In some cases, the nature of the partial immunity led to serious hypersensitivity reactions to infection (Atypical measles). The exact mechanism is still uncertain but it was thought that the vaccine lacked an important antigen of the virus and thus immunity was not complete. In view of the above and the fact that antibody levels decline rapidly after administration of the killed vaccine, live vaccination is now generally recommended and individuals previously immunized with the killed vaccine should be re-immunized with the live vaccine. The killed vaccine has now been Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

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2.4 Management of Outbreaks

Measles outbreaks are most deleterious in wards with immune compromised children or adults e.g. children with leukaemia and bone marrow transplant recipients. Measles is definitely as dangerous as VZV in that setting. HNIG should be given to all severely immune compromised children irrespective of their immunization status since it has been reported that severe measles infection can occur in those who had been immunized and had a documented low-level antibody response. Therefore, the routine screening of children for measles antibody before admission is probably unjustified since there would be no difference in the management. The same argument applies to the screening of patients for immunity before the administration of HNIG. The use of live-attenuated vaccine for post exposure prophylaxis is contraindicated. The same protocol applies to immune compromised adults who come into contact with measles. Immuno competent children under 12 months in whom there is a particular reason to avoid measles, such as a recent severe illness, can also be given immunoglobulin. MMR vaccine should then be given after an interval of at least 3 months, at around the usual age is now incorporated as part as the MMR vaccine. As vaccine-induced measles antibody develops more rapidly than following natural
infection, MMR vaccine can be used to protect susceptible contacts during a measles outbreak. To be effective, the vaccine must be administered within three 3 days of exposure. If there is doubt about a child’s immunity, vaccine should be given since there are no ill effects from immunizing individuals who are already immune. Immunoglobulin should be given to those for whom the vaccine is contraindicated.

The vaccination programme has been most effective in the USA, where measles immunization is compulsory. The incidence rate has also declined dramatically in the UK but without the rigorously pursued vaccination as practiced in the US, it is not likely to be as effective as that in N. America. In the third world, malnutrition aggravates measles infection and there are 900,000 measles related deaths per year. Vaccination in these areas has failed to yield dramatic results. The problem is that the vaccine is usually given at 12 months of age (it should not be given in younger individuals because the presence of maternal antibodies may lead to a poor response.) but infection in these areas often occurs earlier in life. Vaccination should therefore be performed on younger children than in the developed world. However, this must be balanced with the fact that the success rate is lower in younger children (50-75% in 6-month-old-children as opposed to 95% for 12-month-old children.). Measles is highly infectious and has a very high attack rate and thus it would be extremely difficult to eradicate the virus altogether through vaccination.

2.5.0 Life cycle and pathogenesis

2.5.1 Measles virus infection cycle
Potential routes of measles virus dissemination to the brain. There are three potential routes of entry of measles virus into the brain. Since the virus can infect neurons it has been proposed that access to the brain via nerve bundles in the olfactory bulb may occur. Alternatively, the virus can replicate in capillary endothelial cells and thus may infect brain capillaries and release viral particles directly into the brain parenchyma. Thirdly, infection of monocytes in peripheral blood may allow transport of virus directly across the blood-brain barrier since monocytes periodically transmigrate into the brain and differentiate into resident perivascular macrophages or microglial cells.

Trans-synaptic spread through the olfactory bulb
Since measles virus can infect and spread through neurons it is possible that the virus may access the brain parenchyma by trans-synaptic migration along the nerve bundles in the olfactory bulb. Although a unique receptor on neurons remains to be identified, it may be possible for measles virus to penetrate olfactory neurons located in the nasal epithelium. Following penetration of a neuron, measles virus can migrate along a nerve bundle via cell-to-cell spread and ultimately enter the brain parenchyma. This is thought to be mediated by micro-fusion events between the axon and dendrites of adjacent neurons involving viral fusion proteins (F proteins) and membrane proteins (neurokinin-1) expressed on the surface of neurons.

Bone-marrow derived monocytes are recruited to the brain where they mature into resident tissue macrophages (perivascular macrophages) or specialized brain macrophages (microglial cells). It is possible that measles virus can infect peripheral blood monocytes through the CD150 receptor. Some of these infected cells may enter the brain via the post-capillary venules (by diapedesis) and thereby transport measles virus across the blood-brain-barrier. It is unlikely that virus particles can directly penetrate the brain because of the blood-brain barrier. Tight-junctions between capillary endothelial cells and the presence of a basement membrane prevents diffusion of large molecules into the brain.

Transmigration of monocytes across the blood-brain barrier
Although the receptor remains to be identified, measles virus can replicate in endothelial cells lining blood vessels. It is therefore possible that replication of measles virus in endothelial cells of brain capillaries may allow infectious virus particles to bud directly into the brain parenchyma.

Measles virus replication in brain capillary endothelial cells
Measles virus potentially gains access to the brain either via the trans-synaptic spread through nerve bundles in the olfactory bulb, release of virus from infected brain capillary endothelial cells or transmigration of infected monocytes across the blood-brain barrier. Once measles virus has penetrated the brain, primary target cells include perivascular macrophages, microglial cells and neurons. Infection of neurons does not result in budding of virus particles from the cell membrane and cell death.
Virus can however spread between neurons in a cell-to-cell manner although this process takes place very slowly. Often brain complications of latent measles virus only manifest much later following primary infection.

**Measles virus infection of macrophages, microglial cells and neurons**

Mouse models of measles virus infection of the brain have shown that immune control of infected neurons is dependent on IL-12, CD4+ helper T cells and antigen presenting cells (microglial cells and macrophages). Antigen presenting cells secrete IL-12 that promotes the differentiation of Th1 CD4+ helper T cells that secrete IFN-γ. Neurons express receptors for IFN-γ that induces innate anti-viral responses. Neurons do not express HLA class I receptors but expression can be induced by IFN-γ stimulation. However, CD8+ cytotoxic T cells do not kill infected neurons most likely due to the immune privilege mechanisms that protect neurons from immune damage. Usually measles virus infection of the brain is controlled, but severe encephalitis can occur where neurons are damaged by excessive activation of immune cells and pro-inflammatory cytokine production.

**Non-cytolytic control of measles virus replication in neurons**

Acute infection with measles virus is associated with a strong Th1 cell-mediated immune response which resolves infection in the periphery. However, during acute infection and for a number of weeks following clearance of measles virus, individuals have increased susceptibility to secondary infections. This is partially due to lymphopenia that occurs during acute infection, most likely due to loss of immune cells due to infection and syncytia formation (giant cells), however cell numbers soon recover. A switch from Th1 cell-mediated responses to Th2 has been proposed as the reason for the longer lasting immune suppression. This may be due to a lack of secretion of IL-12 by and an increase in IL-4 and TGF-β production by antigen presenting cells. Infected antigen presenting cells do not produce IL-12. Increased IL-10 secretion by Th2 CD4+ helper T cells and regulatory T cells suppresses Th1 responses and may predispose individuals to secondary infections.

**Immuno suppression during measles virus infection**
It has been shown that measles virus can spread between neurons in a cell-to-cell manner, but this process occurs very slowly. Initial penetration of neurons may still depend on a specific receptor which remains to be identified. However, after infection of a neuron, virus can then spread to adjacent neurons at the synapse from the dendrite to the axon (retrograde). The transport of virus from infected neurons to adjacent uninfected neurons is thought to be mediated by micro fusion events at the cell membrane where viral F proteins bind neurokinin-1 receptors expressed on neurons which facilitates membrane fusion. Viral ribo nuclear protein complexes containing viral RNA accumulate at the cell membrane and can pass through to the adjacent cell. In neurons, there is incomplete assembly of virus particles at the membrane and budding of virus does not occur. This prevents antibody detection of free virus and promotes viral latency in the brain.

2.5.2 Pathogenesis

Measles first gains access to the body via the upper respiratory tract or the conjunctiva. The virus quickly spreads to the immediate lymph nodes. Destruction of the lymphoid tissues leads to a profound leucopenia. A primary viraemia ensues which is responsible for spreading the virus throughout the rest of the R-E system and the respiratory system. A secondary viraemia follows whereby the virus is further spread to involve the skin, the viscera, kidney and bladder. The Koplik's spots and the rash in measles are thought to result from a delayed hypersensitivity reaction, the virus antigen being absent from the lesion itself.

**Acute measles panencephalitis** - It is likely that CNS involvement, even in uncomplicated measles, is common. Transient EEG abnormalities are detected in 50% of patients. Measles virus is rarely isolated from the brain of a patient with acute measles panencephalitis. Therefore, current theories favour an autoimmune reaction as the possible cause of CNS damage.

**Subacute measles encephalitis** - arise only in patients with severe immune disorder. Therefore it is not usually accompanied by the formation of antibodies in the CSF. Infectious virus has not been isolated by conventional methods, suggesting defects in replication. Recently biological studies on brain tissue from a case of SME revealed that the envelope proteins were missing from the brain tissue and only the N and the P protein were consistently detected.
SSPE - in SSPE, the virus is first thought to gain entry to the CNS during the viraemia. Once there, it establishes a low-grade persistent infection. It is not known whether viral replication itself, or immune pathological mechanisms are responsible for the development of lesions. In SSPE, free infectious virus particles have never been isolated from the brain or the CSF, although some viral antigens may be found. Giant cells which are characteristic of acute measles infection are also absent. However, viral nucleocapsids are present in the cytoplasm. Therefore, some defect must exist in the virus replication process that prevents maturation. In the absence of free infectious particles, the infection may spread slowly by infectious nucleocapsids from cell to cell.

Antibodies in the CSF are oligoclonal as opposed to the polyclonal response seen in the sera. This suggests that antibody in the CSF is made locally by a much smaller population of lymphocytes which have invaded this compartment. The M-protein is not recognized by the antibodies present in the CSF. SSPE brain lesions have M, N and P proteins present in infected cells whereas the envelope proteins are missing. The measles mRNAs isolated from SSPE patients showed a high rate of mutations, the highest rate of mutation in the M gene, followed by the F, H, P and N genes. In some cases, infectious MV particles may be recovered if the brain cells are co-cultured with tissue culture cells susceptible to measles virus. In other cases though, the block is only partially overcome and the agent remains cell associated. In this case, although MV envelope mRNAs are present, the envelope proteins are not synthesized. Another hallmark of SSPE is the hyperimmune response to measles antigens that include neutralizing antibodies in the serum and the CSF. In spite of this, the infection cannot be controlled. CMI is much more important than the humoral response in clearing measles virus infection. There is no evidence to suggest that the CMI is impaired in patients who develop SSPE.

Natural immunity to measles is known to last at least 65 years. In 1781 measles disappeared from the Faroe islands following an epidemic and was not reintroduced until 1846. Individuals old enough to have experienced the disease 65 years previously were still protected. This unusual persistence of immunity suggests that measles virus may normally persist inside the body, possibly in lymphocytes so that immunity is restimulated from within.
2.6 Review of literature

As of 2008, four of the six WHO regional offices have a measles elimination goal (AMRO, EURO, EMRO, WPRO) and two have a mortality reduction goal (AFRO, SEARO). It is in these latter two regions where most of the global measles deaths occur. Worldwide, since the year 2000, the estimated number of measles deaths has been reduced by 74%. However, despite this remarkable progress in controlling measles, there were an estimated 197 000 deaths in 2007.

However, during the past years, three important changes have occurred which necessitate the revision of the recommendation of the above mentioned 1999 WHO document:

The expanded use of a second opportunity for measles immunization through nationwide mass-vaccination campaigns in high burden countries has resulted in marked reductions in measles incidence associated with reduced community acceptance of large outbreaks.

Endorsement of the International Health Regulations (2005) highlighted the importance of timely detection and response to events that are of potential international public-health concern.

Recent literature on the impact of outbreak response immunization found measles epidemics in endemic, pre catch-up supplementary immunization activity (SIA) settings can last 3–9 months providing adequate time to mount a focused, high quality campaign.


Live attenuated measles-containing vaccines are recommended as a 2-dose series in the United States, with the first dose administered at 12–15 months. They are effective, immunogenic, and generally well tolerated; however, an increased risk of fever and febrile seizures has been described 1–2 weeks after immunization, and the risk is greater after receipt of the combined measles–mumps–rubella–varicella (MMRV) than the MMR vaccine. Most children in the United States receive their first dose of a measles-containing vaccine between the ages of 12 and 23 months. Some vaccine-hesitant parents delay administration of the first dose of a measles-containing vaccine toward the end of the second year, believing that with increasing age the
vaccine will be better tolerated; however, there are minimal data on the risk of adverse events associated with alternate vaccine administration schedules. Thus, examining the safety of measles-containing vaccines administered at different ages during the second year of life is important.

Utilizing the Vaccine Safety Datalink (VSD), the authors performed a retrospective cohort study of children aged 12–23 months who received their measles-containing vaccine between January 2001 and December 2011. The VSD is a collaborative project between the Centers for Disease Control and Prevention and managed care organizations throughout the United States, capturing epidemiological, clinical, and vaccination data on approximately 3% of the U.S. population (8.8 million persons) annually for the purpose of vaccine safety research. The outcomes of interest were fever and seizures within 7–10 days after vaccine administration. Events were identified through Emergency Department and hospital ICD-9 codes, and subjects were categorized as 12–15 or 16–23 months of age at time of vaccination. Using a risk-interval analysis, the incidence of fever and seizures during the risk interval (days 7–10) was calculated and compared with that during the control interval (days 11–42). The relative risk of post immunization fever and seizures was compared between age groups, using a Poisson regression model. Secondary analysis compared the risk of post immunization events as related to the type of vaccine used.

A total of 840,348 children were included in the analysis; 69.4% received the separate MMR and varicella vaccines (MMR + V), 14.3% the combined MMRV, and 16% were immunized against MMR only. The majority were immunized at 12–13 months of age. Overall, the incidence of fever and seizures during days 7–10 was significantly greater than during the control interval in all age groups. The risk of seizures during the 7- to 10-day risk interval was significantly greater among children 16–23 months of age (relative risk, 6.5; 95% confidence interval [CI], 5.3–8.1; attributable risk, 9.5 excess cases per 10,000 doses; 95% CI, 7.6–11.5) than among children 12–15 months of age (relative risk, 3.4; 95% CI, 3.0–3.9; attributable risk = 4.0 excess cases per 10,000 doses; 95% CI, 3.4–4.6). Similarly, the relative risk of post immunization fever was significantly greater among older children (relative risk, 5.9; 95% CI, 5.4–6.5) than among younger children (relative risk, 4.4; 95% CI, 4.3–4.6); however, the attributable risk associated with fever was not significant.
In the analysis of vaccine type, MMRV vaccine was associated with a 1.4-fold increase in the risk of fever and 2-fold increase in the risk of seizures compared with MMR vaccine administered with or without varicella vaccine in both younger and older children.

The authors concluded that measles-containing vaccines are associated with a lower increased risk of seizures when administered at 12–15 months of age. These findings support the timely immunization of children with the first dose of measles-containing vaccines at 12 months of age.


*Staphylococcus aureus* (SA) is a common cause of community-associated (CA) skin and soft-tissue infections (SSTIs). Patients with staphylococcal SSTIs are commonly colonized with the organism; and current prevention strategies focus on decolonization and eradication of carriage. Nevertheless, the relation between isolates recovered from sites of infection as compared to sites of colonization is unclear, especially in community settings. Furthermore, CA SA infections are known to occur in household clusters, with transmission occurring in up to 40% of households of index patients. In the current study, the authors aimed to describe the molecular epidemiology of SA infection and colonization in children with SA SSTI and determine the relatedness of strains among their household contacts who were colonized with SA.

Rodriguez and colleagues used samples that were obtained in a prospective clinical trial comparing 2 decolonization strategies in patients with CA SA-SSTIs. Index patients were aged 6 months to 20 years, presented with a SA infection, and had documented SA colonization of the nares, axillae, and/or inguinal folds. Specimens from similar sites were also obtained from household members to assess SA colonization. Colonization cultures were repeated for the index case only at 1, 3, 6, and 12 months after enrollment. Molecular typing by repetitive sequence-based polymerase chain reaction was used to compare infecting and colonizing isolates.

A total of 163 cases and 562 household contacts were included in the analysis. Forty percent of cases and 21% of household contacts had a history of SSTIs in the year before study enrollment. Most index cases (110 of 163, 67%) were found to have at least 1 baseline colonizing strain that
matched their infecting strain, although 71 (44%) were colonized with more than 1 strain. Almost half of the index cases (n = 75, 46%) had at least 1 household contact colonized with a concordant strain. Of the 53 index cases with discordant infecting and colonizing strain(s), 15 (28%) had 1 or more household contacts with a colonizing strain that matched the index case's infecting strain. A total of 27 distinct strains were observed, with households containing 1–6 different strains. Familial strain-similarity was observed in 105 families (64%).

The authors concluded that 33% of patients presenting with CA SA-SSTI are colonized with 1 or more strains that differed from the one causing the infection, and most of these patients were colonized with a strain that could not be linked to a colonized household member. These results suggest that despite household carriage, non household exposures continue to be associated with a substantial proportion of new acquisition of SA. These findings may help explain why personal or household decolonization strategies are not always successful in preventing SA-SSTIs.


Respiratory tract infections (RTI) are common in the pediatric population. Even though most cases are self-limiting with a very small risk for complications, RTI account for up to a third of pediatric primary care visits. Furthermore, antimicrobials are often prescribed, in spite of little supportive data. One of the main drivers of parents' decision to seek medical advice and pediatricians' prescription of antibiotics is deviation from the expected duration of disease. However, estimates of the expected time course of symptoms of common RTI are highly variable and not always evidence based. The authors conducted a systematic review of the literature on the duration of symptoms of the most common RTI.

Studies were included in the review if they reported on children with acute RTI, were conducted in developed countries, provided data on time to resolution of symptoms, and included a placebo or no-treatment arm, which served as the basis for analysis. A comprehensive search of the literature, followed by data extraction and quality assessment, was performed by 2 authors. The primary outcome was time to resolution of symptoms in 50% and 90% of patients.
In all, 23 randomized trials and 25 observational studies were identified as eligible for inclusion.

**Earache** Data were available for 1409 patients presenting with a main complaint of earache. Based on pooled results, 50% of children's symptoms resolved by day 3 and 90% by day 9.

**Sore throat** In 344 children presenting with a main complaint of sore throat, mean duration of symptoms was 2–6.7 days. Due to lack of consistent reporting, polled data for time to 90% resolution was not reported.

**Cough** Data on 1763 children resulted in the following duration of symptoms:

- **Acute cough** In 50% of patients the cough resolved by day 10 and in 90% by day 21.

- **Croup** Mean duration of 2–3 days, with 50% resolved by day 1 and 80% resolved by day

- **Bronchitis** In different studies, the mean time to resolution was 8–15 days. The pooled time to resolution in 50% of patients was 13 days, and 90% was estimated at 21 days.

- **Common cold** Data on 5327 children were included. Mean duration in different studies was 7–15 days. Based on pooled data, the time to resolution of symptoms in 50% of children was 10 days and in 90% was estimated at 15 days.

The authors conclude that the duration of symptoms in many common RTI is considerably longer than 7 days. This information should help parents and clinicians make informed decisions in treatment of children with RTI.


Community-acquired pneumonia (CAP) is a common cause of pediatric hospitalizations. Despite guidelines published in 2011 by PIDS/IDSA that recommend empiric treatment of children hospitalized with uncomplicated CAP with narrow-spectrum antimicrobial agents (ampicillin or penicillin G), uncertainty about the causative pathogen contributes to the continued use of empiric broad-spectrum antibiotics such as third-generation cephalosporins. The objective of this
The study was designed to compare the effectiveness of empiric therapy with narrow-spectrum antibiotics with empiric therapy with broad-spectrum antibiotics in children hospitalized with uncomplicated CAP.

Queen and colleagues conducted a retrospective, multicenter study, including patients 60 days to 18 years of age with uncomplicated CAP, admitted to 4 medical centers in 2010. Patients were excluded if they were treated with unusual regimens, required intensive-care unit care within 2 days of admission, or were untreated for >48 hours after initial presentation. Main outcomes were length of stay, duration of fever, rates of readmission within 7 days, supplemental oxygen use and overall cost of stay. A propensity score to predict likelihood of receiving broad-spectrum antibiotics was developed and used to match subjects treated with narrow versus broad-spectrum regimens and to adjust for potential confounding by indication.

A total of 492 patients were included: 256 (52%) were treated with a narrow-spectrum antibiotic. In the adjusted analysis, children treated with a narrow-spectrum regimen had a 10-hour-shorter length of stay than those treated with a broad-spectrum regimen (P = .04). There was no significant difference in duration of supplemental oxygen use, duration of fever, standardized daily cost, or readmission rate within 7 days.

The authors conclude that among children hospitalized with uncomplicated CAP, narrow-spectrum antibiotic coverage is associated with outcomes that are comparable to those of broad-spectrum antibiotics. These results support the recommendations to use narrow-spectrum treatment in children hospitalized with CAP.
3.0 Introduction
This section of the study indicates how the research is going to be carried out. It shows the research design, type of data and sources of data, the model formulation, Schematic diagram, Parameters of the model

3.1 Research Design
This study used descriptive survey research design. According to Jackson (2009) descriptive research is used to obtain information concerning the current status of the phenomenon to describe „what exists” with respect to variables or conditions in a situation.

The focus of the study is to model the dynamics of measles Luweero district using data from the selected health centers. Qualitative data will be collected from different medical personals via interviews, while quantitative data will be collected from staff members of the selected health centers in Luweero district.

3.2 mathematical model of infectious diseases
The Modeling of infectious diseases is a tool which has been used to study the mechanisms by which diseases spread, to predict the future course of an outbreak and to evaluate strategies to control an epidemic (Daley and Gani, 2005).

The first scientist who systematically tried to quantify causes of death was (Graunt, 1662). The bills he studied were listings of numbers and causes of deaths published weekly. Graunt’s analysis of causes of death is considered the beginning of the “theory of competing risks” which according to (Daley and Gani, 2005) is “a theory that is now well established among modern epidemiologists”.

The earliest account of mathematical modeling of spread of disease was carried out in 1766 by Daniel Bernoulli. Trained as a physician, Bernoulli created a mathematical model to defend the practice of inoculating against smallpox (Hethcote, 2000). The calculations from this model
showed that universal inoculation against smallpox would increase the life expectancy from 26 years 7 months to 29 years 9 months (Bernoulli and Blower, 2004).

Daniel Bernoulli’s work preceded our modern understanding of germ theory, it was followed by (Hamer, 1906) model on measles but it was not until the research of (Ross, 1911) into the spread of malaria, that modern theoretical epidemiology began. This was soon followed by the work of (McKendrick and Kermack, 1927) whose paper a Contribution to the Mathematical Theory of Epidemics was published in 1927. A simple deterministic (compartmental) model was formulated in this paper. The model was successful in predicting the behavior of outbreaks very similar to that observed in many recorded epidemics (Brauer and Castillo-Chavez, 2001).

3.2 Model formulation and analysis

In this section a deterministic, compartmental mathematical model to describe the transmission dynamics of measles is formulated. It is assumed that the population is homogeneously mixing and reflects increasing dynamics such as birth and immigration, individual can be infected through direct contact with an infectious individual, on recovery the individual obtains permanent infection-acquired immunity that is an individual cannot be infected again.

The total population \( N \) is divided into the following epidemiological classes: Susceptible, \( S \) (Individuals who may get the disease); Exposed or Latent, \( E \) (Individuals who are exposed to the disease); Infected, \( I \) (Individuals who have the disease and are able to transfer it to others); Recovered, \( R \) (Individuals who have permanent infection-acquired immunity) and Vaccinated, \( V \) (Individuals who have received vaccine at birth plus migrant individuals who receive vaccine on arrival). If there is an adequate contact of a Susceptible individual with an Infective individual then transmission may occur, thus the susceptible individuals may join the Exposed class, \( E \) at the rate \( \alpha \). When Latent period ends, exposed individuals may progress to the Infectious class, \( I \) at rate \( \sigma \). After some treatment, infectious individuals may recover and join the recovery class, \( R \) at rate \( \gamma \). Since the disease is fatal, infected individuals may die due to the disease at the rate \( \delta \) or die naturally at rate \( \mu \). The recovered class, \( R \) consists of those with permanent infection-acquired immunity. Basically our new model is an \( SVEIR \) model
Figure 1. Schematic diagram of measles transmission dynamics.

The model equation.

From the model formulation and schematic diagram we here by present the model equations.

\[
\frac{dS}{dt} = \beta N (1 - \tau \varepsilon) - \alpha \frac{SI}{N} - \eta (1 - \tau \varepsilon)S - \mu S \tag{1}
\]

\[
\frac{dV}{dt} = \beta N (\tau \varepsilon) + \eta (1 - \tau \varepsilon)S - \mu V \tag{2}
\]

\[
\frac{dE}{dt} = \alpha \frac{SI}{N} - (\sigma + \mu)S \tag{3}
\]

\[
\frac{dI}{dt} = \sigma E - (\gamma + \delta + \mu)I \tag{4}
\]

\[
\frac{dR}{dt} = \gamma I - \mu R \tag{5}
\]

Where

\( S(t) \) – population of susceptible individuals at time \( t \).

\( V(t) \) – population of vaccinated individuals at time \( t \).
E (t) – population of exposed individuals at time t.
I (t) – population of infected individuals at time t.
R (t) – population of reserved individuals at time t.
N – Total population at time t.

The following are parameters

β – Birth rate of the total population.
εv– Vaccine efficacy.
τ – Vaccine coverage in the community.
α – Transmission rate.
Π – Proportion of susceptible vaccinated at other time not at birth.
μ – Natural death rate of human in the district.
σ – Proportion rate from exposed to infected stage.
δ – death rate due to measles infection.
γ – recovery rate of infected individuals.
CHAPTER FOUR

DATA PRESENTATION ANALYSIS AND INTERPRETATION OF FINDINGS

4.0 Data analysis
1. The objectives of the study are as follows
2. To establish the causes and effects of measles infection
3. To establish the symptoms of measles infection
4. To know how measles can be cured and prevented
5. To develop the mathematical model for the dynamic of the disease.
6. To obtain the equilibrium state of the model.
7. To analyze the equilibrium state for stability.
8. To obtain the basic effective reproductive number

This chapter includes the model analysis of measles in this study

4.1 Causes and effects of measles infection

Measles is a respiratory disease caused by a virus. The disease and the virus that causes it share
the same name. The disease is also called rubeola. This virus normally grows in the cells that line
the back of the throat and lungs.

The virus is an enveloped virus (100–200 nm in diameter), with a core of single-stranded RNA.
Two membrane envelope proteins are important in pathogenesis. They are the F (fusion) protein,
which is responsible for fusion of virus and host cell membranes, viral penetration, and
hemolysis, and the H (hemagglutinin) protein, which is responsible for adsorption of virus to
cells. There is only one antigenic type of measles virus. The virus is rapidly inactivated by heat,
light, acidic pH, ether, and trypsin. It has a short survival time (less than 2 hours) in the air or on
objects and surfaces.

It is very rare in countries and regions of the world that are able to keep vaccination coverage
high. In North and South America, Finland, and some other areas, endemic measles transmission
is considered to have been interrupted through vaccination. There are still sporadic cases of
measles in the United States because visitors from other countries or US citizens traveling abroad can become infected before or during travel and spread the infection to unvaccinated or unprotected persons. Worldwide, there are estimated to be 20 million cases and 164,000 deaths each year. More than half of the deaths occur in India. People who do not have immunity to it (not vaccinated or who never had it before) are at an increased risk of being infected when exposed to a person with it.

4.2 symptoms of measles infection

The symptoms of measles generally begin about 7-14 days after a person is infected, and include: blotchy rash, fever, cough, runny nose, red, watery eyes (conjunctivitis), feeling run down, achy (malaise), tiny white spots with bluish-white centers found inside the mouth (Koplik’s spots). A typical case begins with mild to moderate fever, cough, runny nose, red eyes, and sore throat. Two or three days after symptoms begin, Koplik’s spots may appear inside the mouth. Three to five days after the start of symptoms, a red or reddish-brown rash appears. The rash usually begins on a person’s face at the hairline and spreads downward to the neck, trunk, arms, legs, and feet. When the rash appears, a person’s fever may spike to more than 104 degrees Fahrenheit. After a few days, the fever subsides and the rash fades.

4.3 cure and preventive measures for measles infection

Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child’s temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

People should avoid smoking near the child.
Sunglasses, keeping the lights dim or the room darkened may enhance comfort levels, as measles increases sensitivity to light.

If there is crustiness around the eyes, gently clean with a warm, damp cloth.

Cough medicines will not relieve a measles cough. Humidifiers or placing a bowl of water in the room may help. If the child is over 12 months, a glass of warm water with a teaspoon of lemon juice and two teaspoons of honey may help. Do not give honey to infants.

A fever can lead to dehydration, so the child should drink plenty of fluids.

**Inactivated Vaccine**- this vaccine was intended for use in young children less than 1 year most prone to severe complications. It was thought to be advisable to avoid the use of a live vaccine. It was found that at least 3 doses were needed to elicit a protective antibody response but the antibody of age who are most prone to severe complications. It was thought to be advisable to avoid the use of a live vaccine. It was found that at least 3 doses were needed to elicit a protective antibody response but the antibody levels soon waned. This leave the vaccines open to attack by the natural virus. In some cases, the nature of the partial immunity led to serious hypersensitivity reactions to infection (Atypical measles). The exact mechanism is still uncertain but it was thought that the vaccine lacked an important antigen of the virus and thus immunity was not complete. In view of the above and the fact that antibody levels decline rapidly after administration of the killed vaccine, live vaccination is now generally recommended and individuals previously immunized with the killed vaccine should be re immunized with the live vaccine. The killed vaccine has now been Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

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Cough medicines will not relieve a measles cough. Humidifiers or placing a bowl of water in the room may help. If the child is over 12 months, a glass of warm water with a teaspoon of lemon juice and two teaspoons of honey may help. Do not give honey to infants.

A fever can lead to dehydration, so the child should drink plenty of fluids.

A child who is in the contagious stage should stay away from school and avoid close contact with others, especially those who are not immunized or have never had measles.

Those with a vitamin A deficiency and children under 2 years who have measles may benefit from vitamin A supplements. These can help prevent complications, but they should only be

4.4 Equilibrium state of the model
The model is built using ordinary differential equations in which the independent variable is expressed explicitly, called autonomous differential equation, i.e., equation of the form:

$$\frac{dx}{dt} = f(x,t), x \in \mathbb{R}^n$$

$$t \in [0, +\infty) \in \mathbb{R}, f: \mathbb{R}^n \times \mathbb{R} \rightarrow \mathbb{R}^n$$

(7)

where the function $f$ can depend on the independent variable $t$.

The solutions of the equation $f(x) = 0$ are called the equilibrium points or steady state solution of the ordinary differential equation (ode) (6). It is the state of a system whose configuration or large scale properties do not change over time. For example, in mechanics a system is at equilibrium if the force acting on a body is equal to zero. If dynamic of a system is described by a system of differential equations, then the equilibrium can be estimated by setting the derivatives to zero.
Definition 4.4.1: A point \( \bar{x} \in \mathbb{R}^n \) is called an equilibrium point of the equation (6) if \( f(\bar{x}) = 0 \)

In epidemiology, the equilibrium can be zero, disease – free or endemic. At zero equilibrium, the population tends to extinction, the disease free – equilibrium is the state at which the population comprises of the susceptible and recovered individuals, while in the endemic equilibrium state each compartment of the sub population is greater than zero. It is important we emphasize here that in models with standard incidence and disease induced death we may not be able to explicitly find the coordinates of the equilibrium point, but we can at least show that it exists.

4.4.2 Disease Free Equilibrium (DFE)

There are three types of equilibrium states usually considered in epidemiological modeling; the zero equilibrium, the disease free equilibrium and the endemic equilibrium states. In this work, we consider the disease free equilibrium (DFE), and the endemic equilibrium states. Generally, at the equilibrium states, the rate of change of the state variables with respect to time is zero, i.e.

\[
\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0.
\]

For DFE, i.e. equilibrium points where there is no disease, we define disease compartments as the exposed human, and Infected human compartments that is \( E \) and \( I \) respectively.

To obtain the DFE of the model, we set the right hand side of the model (1)to (6) to zero as given below;

\[
\beta N(1 - \tau e) - \alpha \frac{SI}{N} - \eta(1 - \tau e)S - \mu S = 0
\]

(8)

\[
\beta N \tau e N + \eta(1 - \tau e)S - \mu V = 0
\]

(9)

\[
\alpha \frac{SI}{N} - (\sigma + \mu)S
\]

\[
\beta N(1 - \tau e) - \alpha \frac{SI}{N} - \eta(1 - \tau e)S - \mu S = 0,
\]

\[
\beta N(1 - \tau e) - (\eta(1 - \tau e) + \mu)S = 0
\]

(10)

\[
\sigma E - (\alpha + \delta + \mu)I = 0
\]

(11)
Solving (8) to (12) as the infected compartments tends to zero, we have

$$\gamma I - \mu R = 0 \quad (12)$$

From (8)

$$\beta N (1 - \tau e) - \alpha \frac{SI}{N} - \eta (1 - \tau e) S - \mu S = 0,$$
$$\beta N (1 - \tau e) - (\eta (1 - \tau e) + \mu) S = 0$$

$$S = \frac{\beta N (1 - \tau e)}{(\eta (1 - \tau e) + \mu)} \quad (13)$$

From (9),

$$\beta N \tau e N + \eta (1 - \tau e) S - \mu V = 0$$
$$\mu V = \beta N \tau e N + \eta (1 - \tau e) S$$

$$V = \frac{\beta N \tau e N + \eta (1 - \tau e) S}{\mu} \quad (14)$$

Substituting (13) into (14) and simplifying, we have

$$V = \frac{\beta N \tau e (\eta (1 - \tau e) + \mu) + \eta \beta N (1 - \tau e)^2}{\mu ((1 - \tau e) + \mu)} \quad (15)$$

We denote the disease free equilibrium with \( E_0 \) such that at

$$E_0 = (S, V, E, I, R) = \left( \frac{\beta N (1 - \tau e)}{(\eta (1 - \tau e) + \mu)}, \frac{\beta N (\tau e (\eta (1 - \tau e) + \mu) + \eta \beta N (1 - \tau e)^2)}{\mu ((1 - \tau e) + \mu)}, 0, 0, 0 \right) \quad (16)$$

### 4.5 Endemic equilibrium state

This is an equilibrium state where at least one of the infected compartments is non-zero. In order to find the Endemic equilibrium for our model equations (1) to (5), the following steps are taken.

We let

$$\lambda = \alpha \frac{I}{N} \quad (17)$$
Be the force of infection of susceptible human at steady state, further, we let \( E_v = (S^*, V^* E^* I^* R^* ) \) represent any arbitrary point of the Endemic Equilibrium of our model.

Substituting (17) into the right hand side of (1) to (5) we have, we have:

\[
\begin{align*}
\beta N (1 - \tau e) - \lambda S - \eta (1 - \tau e) S - \mu S &= 0 \quad (18) \\
\beta N \tau e N + \eta (1 - \tau e) S - \mu V &= 0 \quad (19) \\
\lambda S - (\sigma + \mu) S &= 0 \quad (20) \\
\sigma E - (\alpha + \delta + \mu) I &= 0 \quad (21) \\
\gamma I - \mu R &= 0 \quad (22)
\end{align*}
\]

Solving (18) to (22), we have

\[
\begin{align*}
\beta N (1 - \tau e) - \lambda S - \eta (1 - \tau e) S - \mu S &= 0 \\
\beta N (1 - \tau e) &= \lambda S + \eta (1 - \tau e) S + \mu S \\
\therefore S^* &= \frac{\beta N (1 - \tau e)}{\lambda + \mu + \eta (1 - \tau e)} \quad (23) \\
V^* &= \frac{[\lambda + \mu + \eta (1 - \tau e)]\beta N \tau e + \eta \beta N (1 - \tau e)}{\mu [\lambda + \mu + \eta (1 - \tau e)]} \quad (24) \\
E^* &= \frac{\lambda S}{\sigma + \mu} \quad (25) \\
I^* &= \frac{\lambda S \sigma}{(\sigma + \mu)(\gamma + \mu + \delta)} \quad (26) \\
R^* &= \frac{\lambda \sigma \gamma}{\mu (\sigma + \mu)(\gamma + \mu + \delta)} \quad (27)
\end{align*}
\]
4.6 Stability of the disease free equilibrium

**Theorem 1.** The measles model (1) to (5) is locally asymptotically stable if the effective reproductive number \( R_e < 1 \).

**Proof**

Theorem 1 will be proved by finding the eigen-values of the Jacobian matrix evaluated at DFE.

\[
J_0 = \begin{pmatrix}
-K_1 & 0 & 0 & K_2 & 0 \\
K_3 & -K_4 & 0 & 0 & 0 \\
0 & 0 & -K_5 & K_6 & 0 \\
0 & 0 & K_7 & -K_8 & 0 \\
0 & 0 & 0 & K_9 & -K_{10}
\end{pmatrix}
\]

Where \( K_1 = (\eta(1 - \varepsilon \tau) + \mu) \), \( K_2 = \frac{\alpha S^*}{N^*} \), \( K_3 = \eta(1 - \varepsilon \tau) \), \( K_4 = \mu \), \( K_5 = (\sigma + \mu) \), \( K_6 = \frac{\alpha S^*}{N^*} \), \( K_7 = \sigma \), \( K_8 = (\gamma + \mu + \delta) \), \( K_9 = \gamma \), \( K_{10} = \mu \).

We use the traditional characteristic equation method \( |J_0 - \lambda I_5| = 0 \) as follows

\[
|J_0 - \lambda I_5| = 0
\]

The stability of the disease free equilibrium can be obtained by studying the eigen values of the \( |J_0 - \lambda I_5| = 0 \). If all the eigen values have negative part, then the equilibrium point is locally asymptotically stable. The five eigen values are as follows. The determinant of the matrix \( |J_0 - \lambda I_5| = 0 \)
Simplifying, we have

\[ \lambda_1 = \frac{1}{2} \left( - (\sigma + 2\mu + \lambda + \delta) + \sqrt{(\sigma + 2\mu + \gamma + \delta)^2 - 4(\sigma + \mu)(\gamma + \mu + \delta) - \frac{\sigma\alpha S^*}{N^*}} \right) \]

\[ \lambda_2 = \frac{1}{2} \left( - (\sigma + 2\mu + \gamma + \delta) - \sqrt{(\sigma + 2\mu + \gamma + \delta)^2 - 4(\sigma + \mu)(\gamma + \mu + \delta) - \frac{\sigma\alpha S^*}{N^*}} \right) \]

\[ \lambda_3 = -\mu \]

\[ \lambda_4 = \eta (1 - \varepsilon \tau) - \mu \]

\[ \lambda_5 = -\mu . \]

Since all the Eigen-values of the Jacobin are all negative, we conclude that the DFE of the models locally asymptotically stable whenever \( R_c < 1 \).
Table 1: Parameters of the models, their interpretations and numerical values

<table>
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<th>Parameters</th>
<th>Meaning</th>
<th>Value</th>
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<td>$\beta$</td>
<td>Birth rate of susceptible human</td>
<td>0.038</td>
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<tr>
<td>$\alpha$</td>
<td>Contact rate of susceptible human when b</td>
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<td></td>
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<td>$\mu$</td>
<td>Natural death of human</td>
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<td>$\sigma$</td>
<td>Progression rate of exposed human</td>
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<td>Trotter &amp; Philipp, 2003</td>
</tr>
<tr>
<td>$\delta$</td>
<td>Death rate of human due to virus infection</td>
<td>0.0</td>
<td>Index mundi, 2018</td>
</tr>
<tr>
<td>$\gamma$</td>
<td>Recovery rate of infectious human</td>
<td>0.14</td>
<td>Trotter &amp; Philipp, 2003</td>
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<tr>
<td>$\varepsilon$</td>
<td>Vaccine efficacy where (0 &lt; $\varepsilon$ &lt; 1)</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>$\tau$</td>
<td>Vaccine coverage</td>
<td>Variable</td>
<td></td>
</tr>
<tr>
<td>$\eta$</td>
<td>The rate at which susceptible human receive vaccine</td>
<td>0.4</td>
<td>Assumed</td>
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</table>

4.7 The basic effective reproductive number ($R_e$)

One of the most important concerns about any infectious disease is its ability to invade a population. Many epidemiological models have a disease free equilibrium (DFE) at which the population remains in the absence of the disease. The basic reproduction number, $R_0$, is a measure of the potential for disease spread in a population, and is inarguably ‘one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory’ (Heesterbeek and Dietz, 1996). The basic reproductive number $R_0$ is the number of secondary infections that one infective individual would create over the duration of the infectious period when population is completely susceptible. On the other hand, the effective reproductive number $R_e$ is the number of secondary infections that one infective individual would create over the duration of the infectious period when a portion of the population is protected. When $R_e < 1$, then the DFE is locally asymptotically stable, and the disease cannot invade the population, but if $R_e > 1$, then the DFE is unstable and invasion is always possible see Hethcote (1978).

Our model is suited for a heterogeneous population in which the vital and epidemiological parameter for an individual may depend on such factors as the stage of the disease, spatial position, etc. however, we assume that the population can be broken into homogeneous
subpopulation or compartment such that individual in a given compartment are indistinguishable from one another.

We use the next generation matrix approach as described by Driessch and Watmough (2005) to derive our effective Reproductive Number $R_e$. Numerous other articles Dietz (1993), Heesterbeek (1992), Roberts (2003), Simon (1992), and Diekmann (1990), are devoted to the calculation of basic reproductive number $R_0$ for different models of various diseases.

Our model has two Infective compartments, namely the Infective ($I$) and Exposed. It follows that the matrices $F$ and $V$ for the new infective terms and remaining transfer terms respectively are given below. Where the entries of $F$ and $V$ are partial derivatives of $f_I(x)$ and $V_I(x)$. For our model, $F$ and $V$ are given below.

$$F = \begin{pmatrix} 0 & \frac{\alpha S^*}{N^*} \\ 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} \sigma + \mu & 0 \\ -\sigma & (\gamma + \mu + \delta) \end{pmatrix}$$

$V^{-1}$ is calculated as follows:

$$V^{-1} = \begin{pmatrix} \frac{1}{\sigma + \mu} & 0 \\ \frac{\sigma}{(\sigma + \mu)(\gamma + \mu + \delta)} & \frac{1}{(\gamma + \mu + \delta)} \end{pmatrix}$$

Such that

$$FV^{-1} = \begin{pmatrix} \frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} & \frac{\alpha S^*}{N^*(\gamma + \mu + \delta)} \\ 0 & 0 \end{pmatrix}$$

Here, the effective reproductive number $R_e$ is the spectral radius (dominant eigen value) of the product matrix $FV^{-1}$, is calculated as follows;

$$|FV^{-1} - \lambda I| = 0$$

But $I = \begin{pmatrix} 1 & 0 \\ 0 & 1 \end{pmatrix}$
\[ \lambda I = \begin{pmatrix} \lambda & 0 \\ 0 & \lambda \end{pmatrix} \]

so that \[ |FV^{-1} - \lambda I| = 0 \] gives:

\[
\begin{vmatrix}
\frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} - \lambda & \frac{\alpha S^*}{N^*(\gamma + \mu + \delta)} \\
0 & -\lambda
\end{vmatrix} = 0
\]

\[-\lambda \left( \frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} - \lambda \right) = 0
\]

Therefore, \( \lambda_1 = 0 \)

And \( \lambda_2 = \frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} \)

\[ R_e = \rho(FV^{-1}) = \frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} \quad (23) \]

4.7.1 Numerical simulation

In this section, we use the Matlab software to plot the graph of the numerical solution of our model equations. The initial condition for each plot is stated, the parameters values are as given in table 1. Figures 1 to 5 are numerical simulation of the measles model given by equations (1) to (5), using the original system variables with parameter values as given in table 1. The simulations were conducted using the Runge-Kutta method (rkf45) embedded in Matlab software.

The rkf45 method is a fourth-order method, meaning that the local truncation error is on the order of \( 0(h^5) \), while the total accumulated error is order \( 0(h^4) \).
Figure 1. Simulation of susceptible population with, parameter values are as given in table 1

The susceptible population according to figure 1 is decreasing with respect to time. This could be due to the vaccination program which is intended to reduce the number of susceptible individuals.

Figure 2: Simulation Vaccinated population with time, parameter values are as given in table 1
The vaccinated population according to figure 2 is increasing with respect to time.

The exposed population according to figure 3 is decreasing with respect to time. This is due to the vaccination program.

Figure 3. Simulation of exposed population with time, parameter values are as given in table 1
The exposed population according to figure 3 is decreasing with respect to time. This is due to the vaccination program.
Figure 4: Simulation of Infected population with time, parameter values are as given in table 1.
Shows the dynamics of recovered compartment with vaccination. It was observed that the population of the recovered individuals at the very beginning raise rapidly as the rate increases and then fall as time increases.

Figure 5: Simulation of recovered population with time, parameter values are as given in table 1.
This rapid decline of the infected individuals may be due to early detection of the measles and partly due to those who revert to the exposed class.
The virus is an enveloped virus (100–200 nm in diameter), with a core of single-stranded RNA. Two membrane envelope proteins are important in pathogenesis. They are the F (fusion) protein, which is responsible for fusion of virus and host cell membranes, viral penetration, and hemolysis, and the H (hemagglutinin) protein, which is responsible for adsorption of virus to cells. There is only one antigenic type of measles virus. The virus is rapidly inactivated by heat, light, acidic pH, ether, and trypsin. It has a short survival time (less than 2 hours) in the air or on objects and surfaces.

It is very rare in countries and regions of the world that are able to keep vaccination coverage high. In North and South America, Finland, and some other areas, endemic measles transmission is considered to have been interrupted through vaccination. There are still sporadic cases of measles in the United States because visitors from other countries or US citizens traveling abroad can become infected before or during travel and spread the infection to unvaccinated or unprotected persons. Worldwide, there are estimated to be 20 million cases and 164,000 deaths each year. More than half of the deaths occur in India. People who do not have immunity to it (not vaccinated or who never had it before) are at an increased risk of being infected when exposed to a person.

Information from the ministry of health at the district level revealed that the government is looking forward to reduce the likely hood of disease outbreak and improve the health sector in the refugee camps as well as in the entire district as a whole in fact the financial budget of 2018-2019 put much emphasis on the health sector and no doubt it will effect on the health department in Luweero district, the government has prospects of improving on Zirobwe health center to a referral hospital to cater for the increasing need for health attention in wakatayi.

Numerical simulations of the model were carried out to graphically illustrate the trend of susceptible, exposed, infected and recovered populations in figure 1, figure 2, figure 3, figure 4 and figure 5 above. The table 1 also shows the set of parameter values and the state variables which were used in order to support the analytical results.

In this report, we have developed a mathematical model for the transmission of measles disease by considering recurrent infection and vaccination. The disease free and endemic equilibrium, basic reproductive number and its stability is presented. Numerical examples show that vaccination is able to prevent the disease from spreading. Further more, we have shown that $R_c < 1$ implies that the disease will Persist which implies that $R_c = 1$ is the threshold between the with it.
CHAPTER FIVE

SUMMARY OF FINDINGS DISCUSSION, CONCLUSION AND RECOMMENDATION

5.0 Introduction
This chapter includes the summary of findings, discussion, conclusion and recommendations for further studies.

5.1 SUMMARY OF FINDINGS AND DISCUSSION
The main participants of this study were employees, and management of Zirobwe health centre IV in Luweero district located in central Uganda and the health centre data base and patient records were referred to in this study.

When research was conducted in viral diseases department, it was found out that measles was the common infectious disease in this health centre in fact results found out that there is outbreak of measles disease in Luweero and the results are remorse to the community.

The respondents from the administrative unit reported that had measles from 2006 but the severe ones broke out in 2013 and 2015. It was reported further that the influx of refugees in Wakataayi region increased the risk of outbreak of measles infection. Therefore the cases of measles have been attributed to the establishment of refugee camps in Wakaatayi by the government. It was noted that these camps were characterized with population pressure/ congestion, poor standards of health and malnutrition. Related to the scientific study about the spread of measles infection, overcrowding of people in the camps provide a fertile ground for the disease to spread like bush fire.

Measles is a respiratory disease caused by a virus. The disease and the virus that causes it share the same name. The disease is also called rubeola. This virus normally grows in the cells that line the back of the throat and lungs.

The virus is an enveloped virus (100–200 nm in diameter), with a core of single-stranded RNA. Two membrane envelope proteins are important in pathogenesis. They are the F (fusion) protein, which is responsible for fusion of virus and host cell membranes, viral penetration, and hemolysis, and the H (hemagglutinin) protein, which is responsible for adsorption of virus to
cells. There is only one antigenic type of measles virus. The virus is rapidly inactivated by heat, light, acidic pH, ether, and trypsin. It has a short survival time (less than 2 hours) in the air or on objects and surfaces.

It is very rare in countries and regions of the world that are able to keep vaccination coverage high. In North and South America, Finland, and some other areas, endemic measles transmission is considered to have been interrupted through vaccination. There are still sporadic cases of measles in the United States because visitors from other countries or US citizens traveling abroad can become infected before or during travel and spread the infection to unvaccinated or unprotected persons. Worldwide, there are estimated to be 20 million cases and 164,000 deaths each year. More than half of the deaths occur in India. People who do not have immunity to it (not vaccinated or who never had it before) are at an increased risk of being infected when exposed to a person.

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In this report, we have developed a mathematical model for the transmission of measles disease by considering recurrent infection and vaccination. The disease free and endemic equilibrium, basic reproductive number and its stability is presented. Numerical examples show that vaccination is able to prevent the disease from spreading. Further more, we have shown that $R_C < 1$ implies that the disease will Persist which implies that $R_C = 1$ is the threshold between the with it.
\[ R_e = \rho(FV^{-1}) = \frac{\sigma \alpha S^*}{N^* (\sigma + \mu)(\gamma + \mu + \delta)} \]

Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

People should avoid smoking near the child.

Sunglasses, keeping the lights dim or the room darkened may enhance comfort levels, as measles increases sensitivity to light.

If there is crustiness around the eyes, gently clean with a warm, damp cloth.

Cough medicines will not relieve a measles cough. Humidifiers or placing a bowl of water in the room may help. If the child is over 12 months, a glass of warm water with a teaspoon of lemon juice and two teaspoons of honey may help. Do not give honey to infants.

A fever can lead to dehydration, so the child should drink plenty of fluids.

**Inactivated Vaccine** - this vaccine was intended for use in young children less than 1 year most prone to severe complications. It was thought to be advisable to avoid the use of a live vaccine. It was found that at least 3 doses were needed to elicit a protective antibody response but the antibody levels soon waned. This leave the vaccines open to attack by the natural virus. In some cases, the nature of the partial immunity led to serious hypersensitivity reactions to infection (Atypical measles). The exact mechanism is still uncertain but it was thought that the vaccine lacked an important antigen of the virus and thus immunity was not
complete. In view of the above and the fact that antibody levels decline rapidly after administration of the killed vaccine, live vaccination is now generally recommended and individuals previously immunized with the killed vaccine should be re-immunized with the live vaccine. The killed vaccine has now been Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

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A fever can lead to dehydration, so the child should drink plenty of fluids.

A child who is in the contagious stage should stay away from school and avoid close contact with others, especially those who are not immunized or have never had measles.

Those with a vitamin A deficiency and children under 2 years who have measles may benefit from vitamin A supplements.
5.2 Recommendations

Having cited the measles transition dynamics in Luweero district of Uganda and a mathematical model developed and on the basis of the study the following are recommended,

To curb a transmission of measles, people of Luweero district and the whole world should use the inactivated vaccine. This vaccine was intended to be used in young children less than 1 year most prone to severe complications. It was thought to be advisable to avoid the use of alive vaccine. It was found that at 3 doses were needed to elicit a protective antibody response but the antibody levels soon waned. This leaves the vaccines open to attack by the natural virus. In some cases, the nature of the partial immunity led to serious hypersensitivity reactions to infection.

For vaccination against measles vaccine to be effective, the vaccine must be administered within three (3) days of exposure. If there is doubt about a child’s immunity, vaccine should be given since there are no ill effects. From immunizing individuals who are already immune. Immunoglobulin should be given to those for whom the vaccine is contraindicated.

Individuals should be sensitized the basic signs and symptoms of measles for example vomiting, eye infection, diarrhea, respiratory tract infections, such as bronchitis ear infections which can lead to permanent hearing loss and difficulty in breathing, skin rashes runny nose and therefore they should be able to administer basic treatment for example, if the child’s temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, ache and pains.

During the treatment of measles, children under 16 years should not take aspirin. A doctor should advise about acetaminophen dosage, as too much can harm the child, especially the liver.
5.3 AREAS FOR FURTHER RESEARCH
From the findings in this study, the researcher recommends that the future researchers carry out investigations on the following topics.

1. The effects of measles on the development of refugee camps in Zirobwe sub county, Luweero district.
2. The mathematical model of typhoid disease a case study of wakatayi health Centre.
3. The effects of AIDS disease on the education of students and academic performance.
4. The effects of measles vaccine on the health of the people.

5.4 CONCLUSION
With the research about the mathematical model showing the transmission of measles infection.

It shows that the reproductive number (number of people who can contract measles after an infected person has been introduced in the population) can be got by the expression

\[ R_c = \rho(FV^{-1}) = \frac{\sigma \alpha S^*}{N^*(\sigma + \mu)(\gamma + \mu + \delta)} \]

Research has shown that there is no specific treatment. If there are no complications, the doctor will recommend rest and plenty of fluids to prevent dehydration. Symptoms usually go away within 7 to 10 days. The following measures may help:

If the child's temperature is high, they should be kept cool, but not too cold. Tylenol or ibuprofen can help control fever, aches, and pains. Children under 16 years should not take aspirin. A doctor will advise about acetaminophen dosage, as too much can harm the child, especially the liver. There is an excellent selection online if you want to buy Tylenol or ibuprofen.

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A fever can lead to dehydration, so the child should drink plenty of fluids.

A child who is in the contagious stage should stay away from school and avoid close contact with others, especially those who are not immunized or have never had measles.

Those with a vitamin A deficiency and children under 2 years who have measles may benefit from.
QUESTIONNAIRE FOR THE RESPONDENTS
Dear respondents
I am KIYIMBA QURAISH and a student of Kampala International University pursuing a bachelors of science with education. I am carrying out a study research entitled Mathematical model of measles a case study of ZIROBWE health Centre IV LUWEERO district. You are among the respondents randomly selected to provide information. Please you are requested to respond to the questions by ticking on the appropriate box or write a brief statement where applicable. The information provided will be kept confidential and will only be for academic purposes.

SECTION A:
**BIO DATA OF THE RESPONDENTS**

Instructions tick where appropriate

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<td></td>
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<tr>
<td>D</td>
<td>Specify</td>
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SECTION B:
SIGNS OF MEASLES DISEASE

Using a Likert scale of 1 – 3 to rate the following alternatives from A – I where 1- Agree (A), 2- Not Sure (NS), 3 - Disagree (D)

Tick where appropriate

<table>
<thead>
<tr>
<th>STATEMENT</th>
<th>AGREE (1)</th>
<th>NOT SURE (2)</th>
<th>DISAGREE (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diarrhea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Respiratory tract infections, such as laryngitis and bronchitis</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulty breathing</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vomiting</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ear infections, which can lead to permanent hearing loss</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Febrile seizures</td>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>
SECTION C

TREATMENT AND CONTROL OF MEASLES INFECTION
Using a Likert scale of 1 – 3 to rate the following alternatives from A – I where 1- Agree (A), 2- Not Sure (NS), 3 - Disagree (D)

Tick where appropriate

<table>
<thead>
<tr>
<th>STATEMENT</th>
<th>AGREE (1)</th>
<th>NOT SURE (2)</th>
<th>DISAGREE (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Be kept with cool but not too cold</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid smoking near the children</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid contact with infected persons</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crustiness around the eyes, gently clean with a warm damp cloth</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin A supplements</td>
<td></td>
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</tr>
<tr>
<td>Febrile seizures</td>
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</table>
SECTION D

MEASURES OF CONTROLLING MEASLES

Using a Likert scale of 1 – 3 to rate the following alternatives from A – I where 1- Agree (A), 2- Not Sure (NS), 3 - Disagree (D)

Tick where appropriate

<table>
<thead>
<tr>
<th>STATEMENT</th>
<th>AGREE (1)</th>
<th>NOT SURE (2)</th>
<th>DISAGREE (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Inactivated vaccine</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Direct vaccination</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Avoid contact with infected persons</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Advise patients to complete the prescribed doses</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>YEAR</td>
<td>CASES</td>
<td></td>
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<td>-------</td>
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<td>2000</td>
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<td>2016</td>
<td>152</td>
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<tr>
<td>2017</td>
<td>20</td>
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</table>
I want to thank you for taking your time to meet with me today. My name is Kiyimba Quraishand you have been purposively selected to be interviewed because of your strategic position in new vision. This interview is designed to assist me to complete an academic research project on mathematical model of measles a case study of Zirobwe health center IVLuweero district. This research is a partial fulfillment for the award of bachelors of Science with education and the interview will take about 15 minutes. All responses will be kept confidential and will purely be for academic purposes.

What are the causes and effects of measles infection?

What is the mode and rate of transmission of measles?

Examine the mathematical mode of measles infection?

How has this viral infection affecting the people in Luweero District.?

What are the symptoms of measles infection?

What measures can be put in place to cure and prevent to outbreak, spread and prevention this infection?

Thank you for your cooperation.
### APPENDIX C: PROPOSED BUDGET 2018

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<tr>
<th>ITEM</th>
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## APPENDIX D:

### ACTION PLAN

**August 2018-January 2019**

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<tr>
<th>Month &amp; year</th>
<th>August 2018</th>
<th>September 2018</th>
<th>October 2018</th>
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<th>December 2018</th>
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<td>Researcher</td>
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<td>Report writing &amp; approval</td>
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<td></td>
<td></td>
<td>Researcher &amp; supervisor</td>
</tr>
</tbody>
</table>