

The Spread of Contagious Disease (Measles) Using Sir Model

(A Case study of Yobe State Specialist Hospital, Damaturu)

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Abstract

Original Research Article

Based on the findings so far, HAM generates series which converge speedily after some iteration. The epidemic has set up affecting the positive fraction of the community host with an average of 996 infected persons daily. The epidemic level was slow in the first place but later it increases exponentially indicating a sign of danger to the said community of host. Sequel to the finding obtained in this study it is obvious that the results obtained have depicted the number of people likely to be infected over a period of time and make a reasonable forecast of how many people to be infected in a certain time to enable a proper decision and supply in case of an outbreak (measles) like this in a given society of host with known population number. Although the study has not made it to our consumption the death that occurred since inception of the epidemic the number of recovered people and the inclining and declining, in the trend of their immune system during measles when on treatment. Further study could be conducted by any interested candidate to make clarification of these not known parameters enlisted above by the study.

Keywords: SIR Model, Measles, Differential Equation.

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INTRODUCTION

No doubt, the history of mankind has been shaped by the pitiless outbreaks of infectious diseases pandemics, the whole nation and civilizations have been occupied most of the map through the ages. In Athens from 425 to 430 BC set of the periclean golden era, the “Cocoliztli” epidemic which occurred during the 16th Century resulted in 13 million death decimating Mesoamerican native population, the black bubonic plague burst in Europe in 1348 and is estimated to have killed over 25 million people just in five (5) years. The pandemic influenza several of (1918–1919) swept through America, Europe, Asia and Africa Smashing the global. The death toll was around 40 million people. Two to one year less fever pandemic followed in the next decade. The (1957) and 1963 influenza pandemic resulted in two and one million death respectively.

In the last decade emergency and re-emerging pandemic such as Aids, Malaria, Measles and Tuberculosis cause death to millions of people each year.

According to the UNAIDS report on the global AIDS epidemic an estimated 35 million people

including 3.4 million children were living with the HIV worldwide at the end of (2010) while the related death and near infection were 1.8 and 2.7 million respectively, Problems stems mainly from two reasons. Many infectious diseases caught children easier than others and named as childhood disease. Such diseases attack within age of 5 years. One fact is that in this period children are very much attached with their fellows, so disease spread rapidly. Some most common childhood diseases are measles disease. SIR Model SIR Model is considered as a basic epidemic model. Most of the childhood diseases that propagate in population adjust quite simply into this model. Usually, diseases caused by a virus such as influenza, measles, and chickenpox, are of SIR type.

STATEMENT OF THE PROBLEM

I sighted this topic “the spread of contagious disease (Measles) using SIR Model” to enable make a prediction or determine the rate at which made clearing by the work.

RESEARCH QUESTION

For the fact that SIR Model depict the relationship between parameters and their changes with

respect to time hence the need to answer these questions.

- Are there possible that there is a big outbreak infecting a positive fraction that preventing of the disease host community?
- What is the effect of vaccination a given community of host prior to the arrival of diseases?
- What are the epidemic levels?
- How many will get infected if the epidemic takes off?

Research Objectives

To determine the rate at which a contagious disease (Measles) Spread with time and be able to project it impact over a short period of time.

SIGNIFICANT OF THE STUDY

The emergence and re-emergence of Measles outbreak in the temperate state of this country that causes death to millions of children especially those below five (5) years of age is main point of concerned. Hence the need for this research. Significant of the study

RESEARCH HYPOTHESIS

A SIR is a model that predicts the exponential growth rate of measles in a community of host if no isolation was made especially in a closed population.

METHODOLOGY

A Homotopy Analysis method of solving differential equation is employed completely so that the result obtained could be use as a tool for solving the problem.

FRAME WORK

This research is mainly the application of differential equation with much emphasis on the nonlinear system of differential equation.

LITERATURE REVIEW

The new analytical technique was proposed by S.J. Liao in 1992 [1]. Homotopy Analysis Method is a general analytical approach use to solve nonlinear equations and solutions are obtained in the form of series. HAM has a great potential to solve strongly nonlinear problems in science and engineering such as the viscous flows of non-Newtonian fluids, nonlinear heat transfer, finance problems, Riemann problems related to nonlinear shallow water equations, projectile motion, Glauert- jet flow, nonlinear water waves, groundwater flows and Laplace equations with certain boundary conditions.

Global supply chains (GSCs) (2010), are complex socio-technical systems, and a key feature of SIR in modern civilization GSCs can link many firms, involve many locations and transportation channels,

concern thousands of part numbers, and be responsible for hundreds of thousands of shipments on an annual basis. Stake-holders include the firms involved in producing the goods, the customers for the goods, and all the firms involved in moving and storing the goods. The cost of GSCs is significant; for example, recent logistics and SIR costs in the US have ranged between 7.7% and 9.3% of GDP [2]. Even small improvements in GSCs have potentially large benefits to society. This helps to explain the sustained strong interest in logistics systems modeling and analysis.

Anderson and May [3], proposed a paper about the Invasion, Persistence and Spread of Infectious Disease within Animal and Plant Communities. A Preliminary Study of the transmission dynamics of measles, the Causative.

Jefferies [4], viewed AIDS as new sexually transmitted disease (STD) which develop in people who are infected with the HIV. Through not all who are infected with HIV go on to develop AIDS, but they may be unknowingly pass the virus onto other. A disease kills people once it has developed.

A. I. Enagi [5], considers a deterministic compartmental model of tuberculosis control strategy adopted by national tuberculosis and leprosy control program. He established the disease free and the endemic equilibrium state and carried out the stability analysis of the disease free and the endemic equilibrium state. He also carried out numerical simulations of the model to have an insight into the dynamics of the model. He found out that the disease free equilibrium state is stable. The numerical simulation showed that it will be very difficult to complete eradicate tuberculosis from Nigeria using this method adopted by national tuberculosis and leprosy control program.

Mugisha *et al.*, [6], formulated mathematical models for the dynamics of tuberculosis in density population required to minimize and therefore eradicate tuberculosis. Both numerical and qualitative analyses were done and the effect of various in the area size and recruitment rates was investigated. Analysis showed that there existence disease free – equilibrium point provided the characteristics area is greater than the probability of survival from latent stage to infectious stage and the number of latent infectious produced but a typical characteristics area per individual should be at least 0.25 square kilometer in order to minimize tuberculosis incidence. His work suggested that characteristic area can as well be looked at as environmental stressor that can lead to tuberculosis.

Jama [7], the tuberculosis (TB) mortality rate has declined by 37% worldwide since 2000, but the disease still remains 1 of the top 10 causes of death, according to the most recent Global TB Report 2017, released by the World Health Organization. Greater

political commitment is needed to address the burden of the disease and meet the goal of ending the TB epidemic by 2030. Tuberculosis was the leading cause of death from a single infectious agent in 2016, ranking above HIV/AIDS. Worldwide, 10.4 million new TB infections were estimated in 2016—10% of which occurred in people with HIV—and about 1.7 million people died of the disease. More than 600 000 new cases of TB with resistance to the most effective first-line drug, rifampicin, were reported, including 490 000 multidrug-resistant TB infections.

Lerner [8], in his paper, revisiting the tuberculosis research of Thomas Holnee (2009 – 2015) [9], examined the association between stress and tuberculosis. He urged that disease may lead to disease immune function and this thus to clinical disease. His study suggested that persons who had experienced stressful situation, such as divorce, death of spouse, or loss of a job were more likely to develop tuberculosis and less likely to recover from it. The scholars in the study also devised a numerical scale (social readjustment rating scale) that qualified stress events with control groups. They also emphasized the need to understand each patient history and view his/her tuberculosis infection as the culmination of a life of emotional hardship. However their study has been criticized by Theorell [10], for its inability to consider the fact that everybody respond differently to stressful situations.

Tracy *et al.*, [11], present a Meta – analysis of the literature on stress and immunity in humans. The result showed substantial evidence for a relation between stress and decrease in functional immune measures (proliferative response tomatoes and natural killer cell activity). The way neuroendocrine mechanism sand health practice might alternate stress has been discussed, evidence for the relations between stress and both functional and enumerative immune measures have been presented. The authors further stated that, stressor duration is important for immune outcomes, and interpersonal events are related to alterations incident immune parameters than social events. However, their work lacked a qualitative and numerical analysis which this study has covered.

According to Davidson [12], the World Health Organization (WHO) estimated that by the year 2000, there would be 20 million persons infected with HIV, the cumulative total since beginning of the epidemic being 40 million. About 90% of cases are in developing countries unable to afford the expensive medical care required to control progress of the disease. Recent serological test demonstrated HIV seropositive in an African male. In a blood sample taken in 1947 so far Sub-Sahara African has been hardest hit with the HIV epidemic with the prevalence between 15-50 years of age.

Adamu [13], stated that in 1992, 192 countries reported over 0.5 million AIDS cases. In the same year, about 10-12 million people were globally estimated to be HIV infected (including 3 million women and 1 million children) with AIDS developing in about 2-3 million of them. In Nigeria, 3.5 million people are HIV infected including 0.5 million children. In 1999, the sero-prevalence shows that Benue State has the highest rate 16.8%, Jigawa State was the lowest rate 1.7% while Yobe State has the rate 1.9%, and Damaturu Local Government has the highest rate 1.0% in Yobe state.

According to Walsh [14], HIV can infect cells, which carry a protein called CD4 on their surface. Their primary target is the T4 or T helper cells of the Immune system, but it also affects macrophages, cells in the mucus membrane and the both. One reverse transcriptase converts its RNA to DNA that is then integrated into the DNA of the host cell. HIV particles one then assembled & in the cytoplasm of the cell and escape by budding through the cell membrane, killing the cells are invaded and destroyed the body Immune system as weakened making the individual prone to the variety of opportunistic infection, malignant disease and neuro-psychiatric complications. There is strong association between the number of CD4, lymphocyte and the development of life threatening illness.

Coventry at 1999, defined discrimination as making unfair difference in ones treatment of people while stigmatization was defined as branding someone as disgraceful.

According to Chapman [15], individuals who outwards signs of their physical condition are stigmatized by the society that they are treated differently. The averted glance, the imminent remark away from physical contact and the widespread remark all serve to demonstrate that the individual is different and does not fit into the society perception of an acceptable person.

Jatau 1992, noted that the discrimination and stigma caused the patient to construct a new of life around a new set of attitude, a new set of social relationship and activities. He further states that the public perception response to victims situation as a direct expression of his defect. He suggested ways to handle such stigma and the patients. Treat the conditions symptomatically so as to remove or minimize its impacts help the patients to his/her disability and the condition they find themselves. Provide the needs of a patients so as to give him/her a feeling of belonging. The society should be made to perceive the victims as being sick instead of making them feel isolated and unwanted.

METHODOLOGY

Here the method and the process of computing the values in data obtained are done here in this chapter. Therefore, below is the data obtained from the Primary health care management Board Yobe State. And epidemic of Measles in Daya primary village unit to Fika local government Yobe state in the months of May, 2018.

SIR MODEL

SIR model is considered as a basic epidemic model. Most of the childhood diseases that propagate in population adjust quite simply into this model. Usually, diseases caused by a virus such as influenza and

measles are of SIR type. This model was proposed by Kermack and Mckendrick in 1927. Many epidemiological diseases could be described by SIR model. Consider the flow of SIR model with constant vaccination strategy.

Where μ, β, γ and π are considered as positive parameters. Furthermore, we assumed that vaccination is 100% effective and the natural death rates μ and birth rate π are not same, this cause N to be not constant. A susceptible will move to I- compartment when comes in contact with an infected individual, an infected individual move to R compartment after recovery. Vaccinated individuals are also coming into R-compartment. Now SIR model can be formulated as

$$\frac{ds}{dt} = (1 - p)\pi N - \beta \frac{SI}{N} - \mu S \dots \dots \dots (1)$$

SIR Model with constant vaccination where

- S = Susceptible individuals
- I = Infected
- R = Recovered people with permanent immunity
- μ = Natural death rate
- β = average contact rate
- γ = Recover rate
- π = Birth rate
- P = new born vaccinated each year ($0 < P < 1$)

$$\frac{dI}{dt} = \beta \frac{SI}{N} - (\gamma + \mu)I \dots \dots \dots (2)$$

$$\frac{dR}{dt} = P\pi N + \gamma I - \mu R \dots \dots \dots (3)$$

Solution of a Vaccination Based SIR Epidemic Model by Homotopy Analysis Method

We know $N = S + I + R$

Adding (1) to (3), we have

$$\frac{dN}{dt} = (\pi - \mu)N \dots \dots \dots (4)$$

We have a case of varying total population.

DIMENSIONLESS TRANSFORMATION

We want to convert the varying total population into a constant total population, for this we have to choose new variables

$$i = \frac{I}{N} \quad r = \frac{R}{N} \quad s = \frac{S}{N}$$

Now total population is constant i.e., $0 = dt dN$, from equation (4), we have birth rate equal to death rate $\pi = \mu$

Putting respective values in (1), (2) & (3), new system is

$$\frac{ds}{dt} = (1 - p)\pi - \beta si - \pi s \dots \dots \dots (5)$$

$$\frac{di}{dt} = \beta si - (\gamma + \pi)i \dots\dots\dots(6)$$

$$\frac{dr}{dt} = P\pi + \gamma i - \pi r \dots\dots\dots(7)$$

QUALITATIVE ANALYSIS

We will analyze system in two categories

1. Infection free equilibrium ($i = 0$)
2. Endemic equilibrium ($i \neq 0$)

Subsystems in the closed set form are

$$\Gamma = \{(s, i) \in R^+ | 0 \leq s + i \leq 1\}$$

To find fixed points, from eq (5) & (6)

$$0 = (1 - p)\pi - \beta si - \pi r \dots\dots\dots(8)$$

$$0 = \beta si - (\gamma + \pi)i \dots\dots\dots(9)$$

Case I. Infection free equilibrium

When disease dies out naturally then from eq (9)

$$(\beta s - \gamma - \pi) \neq 0 \ \& \ i = 0$$

From eq (8); $s = (1 - p)$

The solution comes on an infection free equilibrium E_0 asymptotically

$$E_0 = (1 - P, 0)$$

Reproduction number and basic reproduction numbers are:

$$R_0 = \frac{\beta}{\gamma + \pi} \ \& \ R_v = \frac{\beta(1 - p)}{\gamma + \pi} \text{ respectively}$$

This is a threshold which determines the stability of equilibrium.

Case II. Endemic Equilibrium

An unstable disease free equilibrium i.e., $R_v > 1$ give rise to endemic equilibrium E_u . Again from eq (9);

$$\begin{aligned} 0 &= (\beta s - \gamma - \pi)i \\ (\beta s - \gamma - \pi) &= 0 \ \& \ i = 0 \\ \Rightarrow s &= \frac{(1 - p)}{R_v} \end{aligned}$$

From of eq (8);

$$\begin{aligned} i &= \frac{\pi}{\beta} \left(\frac{(1 - P)}{s} - 1 \right) \quad \text{or} \\ i &= \frac{\pi}{\beta} (R_v - 1) \end{aligned}$$

So, we have endemic equilibrium of the form

$$E_u = \left(\frac{(1 - p)}{R_v}, \frac{\pi}{\beta} (R_v - 1) \right)$$

STABILITY ANALYSIS

The infection free equilibrium E_0 is locally stable if $R_v < 1$ and endemic equilibrium E_u is unstable. Conversely for $R_v > 1$, endemic equilibrium E_u is stable and infection free equilibrium E_0 is unstable. In both cases local stability of equilibrium give rise to Global stability in the particular domain of s and i . An examination of local stability of the model's equilibria reveals that there is a critical vaccination proportion

$$P_c = 1 - \frac{1}{R_0} \Rightarrow P_c = \frac{\beta - \gamma - \pi}{\beta}$$

P_c governs the system as follow

1. For relatively large vaccination level i.e., $P_c > P$ infection free equilibrium is locally stable with the coordinates

$$s = 1 - p \ \& \ i = 0$$

While endemic equilibrium is unstable.

2. For relatively weak vaccination i.e., $P_c < P$, endemic equilibrium is locally stable with the coordinates

$$s = \frac{(1 - p)}{R_v} \ \& \ \frac{\pi}{\beta} (R_v - 1)$$

The Jacobian matrix at Endemic equilibrium E_u .

$$J = \begin{pmatrix} -\pi R_v & -(\gamma + \pi) \\ \pi(R_v - 1) & 0 \end{pmatrix}$$

$$trcJ = -\pi R_v$$

$$\det J = \pi R_v(\gamma + \pi) - \pi(\gamma + \pi)$$

As we know

$$trcJ \pm \frac{\sqrt{(trcJ)^2 - 4(\det J)}}{2}$$

On putting values, we have

$$\lambda_{1,2} = \frac{-\pi Rv \sqrt{(-\pi 2Rv)^2 - 4[\pi Rv(\gamma + \pi) - \pi(\gamma + \pi)]}}{2}$$

For small values of π & γ we neglect the last term under the square root sign

$$\lambda_{1,2} \approx -\frac{\pi}{2} Rv \pm \frac{1}{2} \sqrt{\pi 2Rv^2 - 4Rv\pi(\gamma + \pi)}$$

For asymptotically stable, value under square root will be negative i.e.

$$Rv \leq \frac{4(\gamma + \pi)}{\pi}$$

The endemic equilibrium E_u is locally asymptotically stable if

$$1 < Rv \leq \frac{4(\gamma + \pi)}{\pi}$$

We have complex eigenvalues with negative real part. So E_u can be treated as a spiral sink. This can be explained as initially susceptible are increasing and we have few infected. Then infection starts spreading and susceptible start to decrease. Disease spread more rapidly than increment in susceptible. As a result we are left with too small number of individuals who are susceptible to disease, the outbreaks ends and susceptible begins to increase again.

Before going on with sir model it is important to understand how epidemics set up in a population for disease conferring long lasting immunity infection. E.g measles. The number of susceptible (s) decrease with time. Before the outbreak of a first measles case the population of susceptible (s) is 100% in the population because everyone is susceptible, the proportion of expose (E), infected (I) and recovered (R) is zero (0), when epidemic start to spread susceptible decrease while immune and infection increase until everyone get immunized.

The potential of infected person in a population depend on the basic in a population depends on the basic reproduction number (R) is define as the average number of person directly infected by an infection disease during his/her entire infection period when he/she enters a totally susceptible persons. The development and the size of infection are determined by (R) that relies on.

1. HAM is different than all perturbation and non-perturbation techniques because of the following facts.

2. Large or small parameters are of no significance in HAM
3. Solution of a Vaccination Based SIR Epidemic Model by Homotopy Analysis Method
4. Convergence of solution can be ensured in a very simple way.
5. We are free to choose base function.
6. The attack rate (risk of transmission per contact)
7. The number of potentially infected contact that is the average person in a population has per unit time.
8. The duration has per the infecting period.

If at any time (R) gets smaller than one that is the disease eventually disappears from the population because on average each infected person cannot ensure transmission of the infection agent to one susceptible. This result in a new waves of infected been lesser amplitude then the proceeding one and finally to disease elimination on other hand it ($R_0=1$) the disease remain endemic as one infection agent to one infections agent to one susceptible on the average. Lastly if ($R_0=1$) epidemic build up. This effort has been established by Kermak and Mc Kendrick and expands body the introduction of infections individual into a community of susceptible does not automatically give rise to an epidemic outbreak.

RESULT AND DISCUSSION

In this section we are used the model formulated in chapter three to compute the result at various time and the result obtained can be discuss to enable used depict the epidemic or otherwise from equation.

Table-1: The initial values and parameters

Case	S_0	i_0	r_0	β	γ	π	P
1	1	0	0	0.9	0.04	0.2	0.9
2	0.9	13	0	0.9	0.04	0.2	0.6
3	0.8	17	0	0.9	0.04	0.2	0.3
4	0.7	19	0	0.9	0.04	0.2	0.0

$$Rv = \frac{\beta(1-P)}{\gamma + \pi} = \frac{0.9(1-0.9)}{0.04 + 0.2} = \frac{0.09}{0.24}$$

$$= 0.375$$

$$Rv < 1, E_0$$

$$\text{Fro } t=0$$

$$\frac{ds}{dt} = (1-P)\pi - \beta s_0 i_0 - \pi s_0 - \frac{\beta(1-P)}{\gamma + \pi}$$

$$= (1-0.6)0.2 - 0.9(1) - 0.2(1)$$

$$= 0.08 - 0.2$$

$$= -0.2 \Rightarrow s = -0.12 = s1$$

$$s(t) = 0.1 - 0.12t + 0.54 \times 10^{-1}t^2 - 2.6 \times 10^{-2}t^3 + 2.6 \times 10^{-3}t^4 - 0.597 \times 10^{-4}t^5 - 0.32444444493 \times 10^{-6}t^6$$

$$i(t) = 0$$

$$r(t) = 0.12t - 0.54 \times 10^{-1}t^2 + 2.6 \times 10^{-2}t^3 - 2.6 \times 10^{-3}t^4 + 0.597 \times 10^{-4}t^5 - 0.312 \times 10^{-5}t^6$$

$$\frac{dr}{dt_{t=0}} = P\pi + -\gamma i_0 - \pi r_0$$

$$\Rightarrow r = 0.12t = r1$$

For

$s_2, s_3, s_4, \dots, i_2, i_3, i_4, \dots$ and r_2, r_3, r_4, \dots

Time Population Fraction

Susceptible Infected Recovered

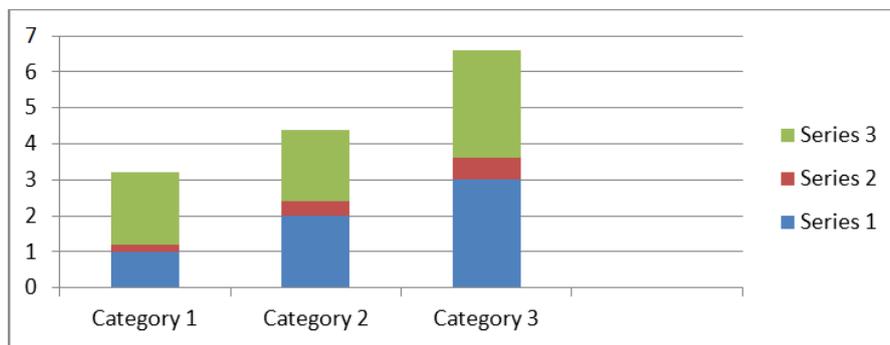


Fig-1: Plot for case 1

From Table, based on Figure-1, there is a decline in the number of non-infected person in the society of host, although, the number of non-infected decline steady and latter reduces sharply showing a threat to the community.

Time Population Fraction

Susceptible

Infected

Recovered

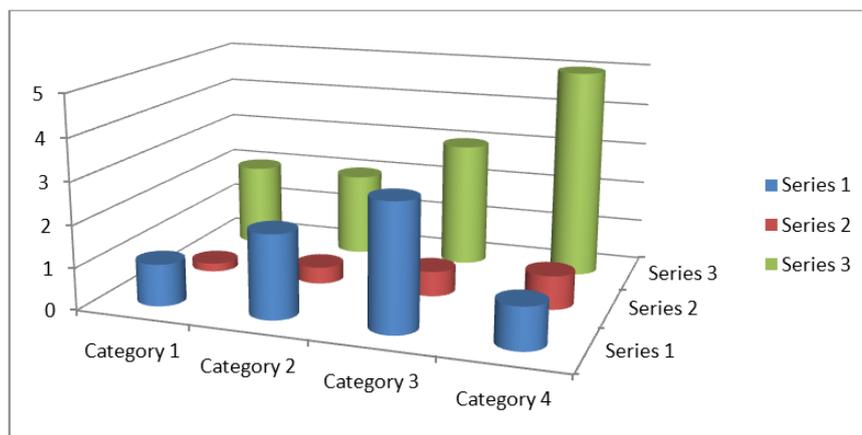


Fig-2: Plot for case 1

However, the Table-2 and the above Figure-1 depicts that an epidemics has set up and the level of the epidemic is totally of the final stayed affecting on average of 286. People in just four days, that is it infected positive is steady from the onset and letter it exponentially increase causing every large damage to

the society. Also the graph below depicted the non-infected people against the time in the same society of host at that period in time.

CONCLUSION

Based on the findings so far, HAM generates series which converge speedily after some iteration. The epidemic has set up affecting the positive fraction of the community host with an average of 996 infected persons daily. The epidemic level was slow in the first place but later it increases exponentially indicating a sign of danger to the said community of host.

RECOMMENDATION

Sequel to the finding obtained in this study it is obvious that the results obtained have depicted the number of people likely to be infected over a period of time and make a reasonable forecast of how many people to be infected in a certain time to enable a proper decision and supply in case of an outbreak (measles) like this in a given society of host with known population number.

Although the study has not made it to our consumption the death that occurred since inception of the epidemic the number of recovered people and the inclining and declining, in the trend of their immune system during measles when on treatment. Further study could be conducted by any interested candidate to make clarification of these not known parameters enlisted above by the study.

A study of this nature needs data from the host societies which were normally monitored and checked by health agencies. Difficulties are often encountered while trying to collect the data for the paper. In conclusion I would like to say that since there is an outbreak in the said society of host a special care unit be built by the government in at least every unit of local government so that we can get proper health care.

REFERENCES

- Liao S. J. (1998). Homotopy Analysis Method: A New Analytical Method for Nonlinear Problems. *Applied Mathematics and Mechanics*, 19(10).
- Zhao, X., Lynch Jr, J. G., & Chen, Q. (2010). Reconsidering Baron and Kenny: Myths and truths about mediation analysis. *Journal of consumer research*, 37(2), 197-206.
- May, M., Gompels, M., Delpech, V., Porter, K., Post, F., Johnson, M., ... & Hill, T. (2011). Impact of late diagnosis and treatment on life expectancy in people with HIV-1: UK Collaborative HIV Cohort (UK CHIC) Study. *Bmj*, 343, d6016.
- Jefferies, J. E., DeMay, R. W., & Lachinyan, G. L. (2014). *U.S. Patent No. 8,768,565*. Washington, DC: U.S. Patent and Trademark Office.
- Enagi, A. I. (2013). A deterministic compartmental model of tuberculosis control strategy adopted by the national tuberculosis and leprosy control program in Nigeria. *Specific journal of science and technology*. 14(1): 342-348.
- Mugisha, J. Y. T Ssematimba, A., & Luboobi, L. S. (2015). Mathematical models for the dynamics of TB in density – dependent populations; the case of internally displaced peoples' camps (IDPCS) in Uganda. *Biostat*, 1(3):217-224.
- Friedrich, M. J. (2017). Tuberculosis update 2017. *Jama*, 318(23), 2287-2287.
- Lerner, M. (2017). *The Mind and Faith of Justice Holmes: his speeches, essays, letters, and judicial opinions*. Routledge.
- Barron, I. H. (2017) can stress cause diseases? Revisiting tuberculosis research on Thomas Holmes. *Ann Intern Med*; 124:673-680.
- Aronsson, G., Theorell, T., Grape, T., Hammarström, A., Hogstedt, C., Marteinsdottir, I., ... & Hall, C. (2017). A systematic review including meta-analysis of work environment and burnout symptoms. *BMC public health*, 17(1), 264.
- Tracy, B. H., & Sheldon, C. (2016). Stress and immunity in Humans. A meta-analytic Review. *Psychosomatic Medicine*, 55:364-379.
- Davidson, R. J., & Irwin, W. (1999). The functional neuroanatomy of emotion and affective style. *Trends in cognitive sciences*, 3(1), 11-21.
- Adamu, B. A. (2001). Resistant starch derived from extruded corn starch and guar gum as affected by acid and surfactants: Structural characterization. *Starch- Stärke*, 53(11), 582-591.
- Walsh, M. (1997). How nurses perceive barriers to research implementation. *Nursing standard (Royal College of Nursing (Great Britain): 1987)*, 11(29), 34-39.
- Chapman, S. J. (1997). Barley straw decomposition and S immobilization. *Soil Biology and Biochemistry*, 29(2), 109-114.