I. INTRODUCTION

An infectious disease is one that can be spread or transmitted from one host to another. Typhoid fever is an infectious disease. There are individuals who are able to transmit their illness but do not exhibit any symptoms. These individuals are called “carriers” and they play an important role in the transmission of the disease. The focus of this study is on infectious disease carriers. An infectious disease that produces long-term asymptomatic carriers is the Typhoid fever caused by the bacteria Salmonella typhi. Typhoid fever reached public notoriety at the beginning of the 20th century with the cases of Mr. N. the “milker” in England and Typhoid Mary in the US. These individuals infected hundreds of people over the decades while they worked in the food production industry and private homes. Even today, Typhoid fever infects 21 million people and kills 200,000 worldwide every year. Asymptomatic carriers are believed to play an essential role in the evolution and global transmission of typhi, and their presence greatly hinders the eradication of Typhoid fever using treatment and vaccination [Roumagnac et al., 2006]. The study of this model is focused mainly on the impact of the effects of carriers of typhoid fever on the human population. Although infectious diseases are present in human populations at all times to some degree, the effects of epidemics are the most noticeable and spectacular. It is possible to mathematically model the progress of typhoid fever to discover the likely outcome of an epidemic [Goldstein et al., 2005]. The reproductive ratio, \( R_0 \), measures the average number of new infections generated by a singly infected typhoid individual during his/her entire infectious period when he/she is introduced into a susceptible population in the presence of the aforementioned intervention strategies [Zhao et al., 2000]. In recent decades, there have been several investigations of infectious diseases using deterministic mathematical models with or without demographic change [Guo, 2005]. In particular Greenhalgh (1996) has studied an infectious disease model with population-dependent death rate using computer simulation. Gao & Hethcote analyzed an infectious disease model with logistic population growth. Zhou & Hethcote have studied a few models for infectious diseases using various kinds of
demographic Hethcote has discussed an epidemic model in which the carrier population is assumed to be constant. But in general the size of the carrier population varies and depends on the natural conditions of the environment as well as on various discharges into it by the human population. The simulations are conducted using a statistical computing program known as R.

II. Literature Review

Modeling the transmission dynamics of typhoid is an important and interesting topic for a lot of Computational mathematical researchers [LaSalle, 1976]. The study of infectious diseases in the past has been focused mainly on their impact on the human population. Although infectious diseases are present in human populations at all times to some degree, the effects of epidemics are the most noticeable and spectacular [Hyman & Li, 2006]. The New World was considered virtually disease-free. Between 1918 and 1921, the Soviet Union incurred about 25 million cases of typhus with a death rate of approximately 10 per cent. From Daniel Bernoulli’s analysis of smallpox (1760), mathematical modeling on epidemic models of infectious diseases has been extended greatly in recent years. He was, by a long way, the first to express the proportion of susceptible individuals of an endemic infection in terms of the force of infection (the annual rate of acquiring an infection) and life expectancy [Guo, 2005]. Hundreds of mathematical models have proven particularly powerful in the study of the effects of bacterial, parasitic and viral pathogens. In the past much understanding has been gained through the use of relatively simple 3 models capturing only the most critical biological mechanisms. Now, the improvements in data capture produce major challenges in developing frameworks capable of utilizing this data to predict the complex patterns of evolution of infectious diseases in increasingly dense and interconnected human populations. Mathematical models and computer simulations have become useful in analyzing the spread and control of infectious diseases [Medley et al., 2001; Naresh et al., 2008; CDC, 2009]. They together, build and test theories that are involved with complex biological systems related disease, getting quantitative conjectures, determining parameter sensitivities due to change and estimating parameters from data. One of the most important reasons that developed countries have become as productive as they are today is that the population remains healthy and disease free [Ghosh et al., 2004; Trotter et al., 2005; Naresh et al., 2008]. This essential task is performed by each country’s health department and is carried out by individuals known as epidemiologists. Without their efforts and their coordination with others in the medical field, it would be very difficult if not impossible to obtain current information regarding important diseases, methods of transmission, methods of control, and the like. Furthermore, information on the incidence or prevalence of diseases and statistics on morbidity and mortality rates, all of which are essential to physicians and other medical personnel to help control and understand diseases, would not be available without the efforts of the epidemiologists [Black, 2004]. Ronald Ross (1932) was an English physician. Ross was a pioneer in developing mathematical models for the study of epidemiology. Anderson Gray McKendrick (1943) Scottish physician and epidemiologist was another pioneer in the use of mathematical methods in epidemiology. McKendrick’s career as a mathematical epidemiologist began in India. In 1914, he published a paper in which he gave equations for the pure birth process and a particular birth-death process. After his return to Scotland he collaborated with Kermack on a notable series of papers. The first paper (1927) gave the differential equations for a deterministic general epidemic. It is important to mention that modeling is very crucial in epidemiology since in most cases we cannot do experiments. It is possible to mathematically model the progress of most infectious diseases to discover the likely outcome of an epidemic or to help manage them by different control programs. In the early 20th century, mathematical methods were introduced into epidemiology by Ronald Ross (1916) and others. Even though the actual data needed for the models might not be accurate or even available, such modeling is still vital in investigating how changes in the various assumptions and parameter values affect the course of the epidemic. Mathematical modeling helps in the set of conditions by which the extinction of susceptible population is reached. Their work suggested that the magnitude of herd protection effects greatly influences the total number of cases avoided and the value of public treatment cost savings. [Kalajdzievska, 2006; Guo & Li, 2006; Kalajdzievska & Michael Y. Li, 2011] developed a mathematical model for assessing the effects of carriers on the transmission dynamics of infectious diseases in general. A lot of work has been done but for the effect of carriers on the transmission of typhoid fever a different approach that would enable Kenya to be the most developed country by ensuring that the population remains healthy and disease free is studied. Thus in this study, the effect of variable carrier population caused by environmental discharges on the spread of typhoid addresses this problem [Kemper, 1978].

Figure 1: Transfer Model for Transmission Dynamics for Typhoid Fever
III. Governing Equations for the Model

The model takes the form,
\[
\begin{align*}
S' &= b - d_1S - S(\beta I + \gamma I) - \theta S \\
I_{c}' &= pS(\beta I + \gamma I) - (d_2 + \alpha)I_c \\
I' &= (1 - p)S(\beta I + \gamma I) - (d_3 + \pi)I + \alpha I_c \\
R &= \pi I + \theta S - d_4
\end{align*}
\]
(1)
With \(S > 0, I > 0, I_c > 0, R > 0\).

It is noted that disease carriage is different from disease latency in that individuals in the carrier state are infectious while those in the latent period are not. This model is thus different from the other traditional models that incorporate disease latency. Carriers are allowed to become symptomatic over their life time. This model is different from the carrier model in that new infections are allowed to be either symptomatic or asymptomatic with certain probabilities. Finding the equilibria \((S^*, I_{c}^*, I^*)\) from equations,
\[
\begin{align*}
b - d_1S^* - S^*(\beta I^* + \gamma I^*) - \theta S^* &= 0 \\
pS^*(\beta I^* + \gamma I^*) - (d_2 + \alpha)I_c^* &= 0 \\
(1 - p)S^*(\beta I^* + \gamma I^*) - (d_3 + \pi)I + \alpha I_c^* &= 0
\end{align*}
\]
(2)
Model (1) always has a disease-free equilibrium \(P_0 = \left(\frac{b}{\pi + \gamma}\right)\). An endemic equilibrium \(P^* = (S^*, I_{c}^*, I^*) \) satisfies \((S^*, I_{c}^*, I^*)\). The equilibrium equation shows that a unique \(P^* \) exists with,
\[
S^* = \frac{pd_3\beta + (d_2 + \alpha)\gamma + p(\pi - d_2)\gamma}{(d_3 + \pi)\gamma}(d_3 + \pi)
\]
(3)
Then \(R_0\) is a threshold parameter that determines the number of equilibria. If \(R_0 \leq 1\) then \(P_0\) is the only equilibrium in \(\Gamma\); if \(R_0 > 1\), then there are two equilibria, \(P_0\) and a unique endemic equilibrium \(P^*\). Rewriting \(R_0\) in (3) as
\[
R_0 = \frac{\beta}{(d_3 + \pi)\gamma}(d_3 + \pi)
\]
(4)
This agrees with the intuition that higher transmissibility increases the basic reproduction number. To see the effect of \(p\) on \(R_0\) it is noted that,
\[
\frac{\beta R_0}{dp} = \frac{\beta}{(d_3 + \pi)\gamma}(d_3 + \pi)
\]
and thus
\[
\frac{\partial R_0}{\partial p} > 0 \text{ if } \beta > \frac{d_3 + \pi}{d_3 + \pi} \gamma
\]
(5)
The effect of diagnosis rate \(\alpha\) on \(R_0\) can also be analyzed. Straightforward computation gives
\[
\frac{\partial R_0}{\partial \alpha} = \frac{p\beta}{(d_2 + \alpha)^2} \frac{\gamma}{(d_3 + \pi)(d_2 + \alpha)^2} \frac{d_3 + \pi}{\alpha}
\]
and thus \(\frac{\partial R_0}{\partial \alpha} > 0\) if the same condition (5) holds.

From this analysis it can be seen that parameter \(p\) and \(\alpha\) have opposite effects on \(R_0\).

3.1. Stability of the Disease-Free Equilibrium

To examine the local stability of the disease-free equilibrium \(P_0\) we evaluate the Jacobian matrix at \(P_0\) as follows
\[
J(P_0) = \begin{bmatrix}
-d_1 - \theta & -\beta & 0 \\
0 & -d_2 + \alpha & \gamma \\
0 & (1 - p)d_3 + \pi + \alpha & (1 - p)\gamma
\end{bmatrix}
\]
(6)
One eigenvalue of \(J(P_0)\) is \(\lambda_1 = -(d_1 + \theta) < 0\). The other two eigenvalues \(\lambda_2, \lambda_3\) are eigenvalues of the \(2 \times 2\) matrix
\[
A = \begin{bmatrix}
\beta b & (d_1 + \theta) - d_2 + \alpha \\
0 & (1 - p)\gamma
\end{bmatrix}
\]
and thus \(\lambda_2, \lambda_3\) are stable if \(A < 0\). Therefore the disease-free equilibrium \(P_0\) is stable if \(R_0 < 1\).

3.2. Stability of the Endemic Equilibrium \(P^*\)

If \(P_0 > 1\), then \(P^*\) is globally asymptotically stable with respect to the interior of \(\Gamma\).

Using Lyapunov function \(V\) of the form,
\[
V(S, I, I_c, I) = x_1(S - S^* \ln S) + x_2(I - I^* \ln I) + x_3(I - I^* \ln I)
\]
(7)
Where \(x_1, x_2, x_3 > 0\) are constants to be specified. It is noted that \(V\) has a global minimum at \(P^*(S^*, I^*, I_{c}^*)\) and \(V(S, I, I_c, I) - V(P^*)\) is positive definite. It is shown that suitable constants \(x_1, x_2, x_3\) can be chosen such that the Lyapunov derivative of \(V\) is negative definite with respect to \(P^*\).

Direct calculation and applying the identity \(b = d_1 S^* + \theta S^* + \beta I_{c}^* + \gamma I_{c}^* S^*\) lead to
\[
\frac{dV}{dt} = x_1(S - S^*) + x_2(I - I^*) + x_3(I - I^*)
\]
Positive constants \(x_1, x_2, x_3\) are chosen as
\[
x_1 = 1, x_2 = -\frac{(d_3 + \pi)(\beta S^* + \gamma S^*)}{(d_2 + \alpha)(d_3 + \pi)}, x_3 = \frac{\gamma S^*}{(d_3 + \pi)}
\]
(8)
Re-grouping terms in \(\frac{dV}{dt}\) such that \(\frac{dV}{dt} = V_1 + V_2 + V_3\), where
\[
V_1 = (d_1 + \theta)S^* \left(2 - \frac{S^*}{S}\right),
V_2 = x_1(\beta S^* I_{c}^* + \gamma I_{c}^* S^*) + x_2(d_2 + \alpha)I_{c}^*
+ x_3(d_3 + \pi)I^*
\]
(9)
It can be seen that \(V_1 \leq 0\) from the inequality \(x + \frac{1}{x} \geq 2\) for all \(x > 0\) and that \(V_1 = 0\) if and only if \(S = S^*\). It can also be shown that \(V_2 + V_3 \leq 0\) by examining \(V_2\). Using the values for \(x_1, x_2, x_3\) in (7), relations in (8), and the equilibrium relation
\[
(pd_2 + \alpha)I_{c}^* = (1 - p)(d_3 + \pi)I^*,
\]
(10)
V_2 \text{ is rewritten as,} \\
V_2 = 2px_3yI'S^* + 2(1-p)x_2\beta I^*_cS^* + 4px_3\beta I^*_cS^* + 3(1-p)\alpha \frac{S}{(pd_2 + \alpha)}yI^*_cS^*. \hspace{1cm} (10)

Writing, V_3 = V_a + V_b + V_c + V_d, with each term representing the expression enclosed in a pair of big square brackets. Each term in V_3 will be estimated by applying the inequality \\
\frac{a_1 + a_2 + \ldots + a_n}{n} \geq (a_1, a_2, \ldots, a_n)^{1/n} \text{ for } a_i > 0.

The following are obtained as follows:

\begin{align*}
V_a &= -x_2(1-p)\beta I^*_cS - \frac{(1-p)x_2\beta I^*_cS^*}{S} \\
&\leq -2\sqrt{(x_2(1-p))^2(\beta I^*_cS^*)^2} \\
&= -2(1-p)\beta I^*_cS^* \\
V_b &= -x_3pyI^*_cS^* \frac{S}{S} \\
&\leq -2\sqrt{(x_3p)^2(yI^*_cS^*)^2} \\
&= -2px_3yI^*_cS^* \\
V_c &= -y \frac{x_2(1-p)yIS^*}{I} - \frac{x_3aI_cI^*}{I} \\
&= -y \frac{(1-p)x_2yI^*_cS^*}{S} \leq -3 \\
V_d &= -(1-y) \frac{x_2(1-p)yIS^*}{I} - \frac{x_35I_cI^*}{I} \\
&= -(1-y) \frac{x_2(1-p)yIS^*}{S}
\end{align*} \hspace{1cm} (11) - (14)

Therefore, \((11) - (14)\) imply \\
\begin{align*}
V_3 \leq -2(1-p)x_2\beta I^*_cS^* - 2px_3yI^*_cS^* - 4px_3\beta I^*_cS^* + 3(1-p)\alpha \frac{S}{(pd_2 + \alpha)}yI^*_cS^*. \hspace{1cm} (15)
\end{align*}

It follows from \((10)\) and \((15)\) that \(V_2 + V_3 \leq 0\) and thus \(\frac{dv}{dt} \leq 0\). Furthermore, \\
\(\frac{dv}{dt} = 0\) if and only if \(V_1 = 0\) and \(V_2 + V_3 = 0\). Using \((10)\) - \((15)\), it can be shown that; \\
\(\frac{dv}{dt} = 0 \Rightarrow (S, I_c, I) = (S^*, I^*_c, I^*_c)\), and thus \(\frac{dv}{dt}\) is negative definite with respect to \(P^*\).

The global stability of \(P^*\) follows from the classical stability theorem of Lyapunov.

3.3. Assumptions of the Model

The following assumptions were taken into considerations:

- The study generalized the whole town.
- In the period of study, most of the clinical typhoid fever cases were treated so most of the infected people recover after treatment.
- Whole population was only slightly decreased due to typhoid fever.
- The number of individuals who come to town is taken to be equal to those moving out of town.

IV. Simulation of Results

The compartment S contains individuals who are Susceptible to typhoid fever infection, compartment I contains individuals who are chronically infected with typhoid fever, compartment I_c contains individuals who are asymptomatic carriers of typhoid fever and have no knowledge of their condition and compartment R contains individuals who have recovered from the fever. A computer code was generated to solve the above equations using computer software known as “R”. The values of our model parameters that were obtained from table 1 are as in table 2 below.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>b</td>
<td>Rate of influx of susceptible</td>
</tr>
<tr>
<td>d_1d_4</td>
<td>Natural death rates</td>
</tr>
<tr>
<td>d_2d_3</td>
<td>Death rates for both natural and disease-caused death</td>
</tr>
<tr>
<td>(\beta)</td>
<td>Transmission coefficient for the carrier compartment (I_c)</td>
</tr>
<tr>
<td>(\gamma)</td>
<td>Transmission coefficient for the symptomatically infected compartment (I)</td>
</tr>
<tr>
<td>(\alpha)</td>
<td>Rate at which carriers develop symptoms</td>
</tr>
<tr>
<td>(\pi)</td>
<td>Rate of recovery</td>
</tr>
<tr>
<td>(p)</td>
<td>Probability of a newly infected individual is asymptomatic</td>
</tr>
<tr>
<td>(\nu)</td>
<td>Vaccination rate</td>
</tr>
</tbody>
</table>

Table 1: The State Variables and Parameters used for the Population

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>b</td>
<td>750</td>
</tr>
<tr>
<td>d_1</td>
<td>0.15</td>
</tr>
<tr>
<td>d_2</td>
<td>0.1503</td>
</tr>
<tr>
<td>d_3</td>
<td>0.1503</td>
</tr>
<tr>
<td>(\beta)</td>
<td>0.125</td>
</tr>
<tr>
<td>(\pi)</td>
<td>0.0625</td>
</tr>
</tbody>
</table>

Figure 2: Simulations of the Susceptible Population at the Very First Time

The graph shows the effects of different initial conditions (levels of typhoid outbreaks cases) where the disease is spreading and no treatment is offered to the population of Kisii town in Kenya on the prevalence of typhoid. The population is decreasing with time. The decrease may be due to a higher rate of contact with infected individuals and also due to consumption of contaminated food. This graph suggests that an increase in human recruitment rate, typhoid transmission and the proportion of individuals who get infected with typhoid greatly influence the magnitude of the...
reproductive number. Hence in this case, treatment will have a greater influence on reducing cumulative typhoid cases.

Figure 3: Simulations of Infected Population

The graph shows the effects of treatment rate on cumulative typhoid cases without carrying research to know the cause of the fever. The graph for Susceptible is as explained in figure 2 above but for the recovery graph gives a picture of Kisii town especially when typhoid fever is a problem to the town and treatment is becoming difficult leading to patients taking longer to recover after sometime and also those who recover decrease after a short while because they do not develop immunity to the disease and therefore they are re-infected again by taking contaminated water from Nyangongo and dara mbili rivers in kisii town. The intersection of the two curves gives a solution to the typhoid fever in kisii town. Hence when all the infected individuals recover fully we have a stable town, therefore, Infected (I) = Recovered (R) i.e Infected (I) – Recovered (R) = 0.

Figure 4: Simulations of the Infected Population

The graph shows the effects of increasing the proportion of individuals who become infectious carriers upon infection on the cumulative new infections in Kisii town. The infectious population is responsive to both changes in treatment as well as changes in the levels of carriers. Increasing the percentage of people receiving treatment causes the infectious population to drop. The stationary point shows the maximum number of people in town getting infected after one and half months before recovering fully from the disease.

Figure 5: Simulations of the Susceptible Population after the Spread of the Disease

The graph shows how Susceptible individuals respond to the typhoid fever upon infection. Switching between different therapies leads to noticeable changes in the number of different bacteria. In this graph the population of the susceptible decreases after about 14 days which is the incubation period of the disease after which fever and rush develop and they the infected stage. After about 40 days the graph remains constant since most of those infected become carriers or do not access proper medical treatment since they are from a poor village like masosa in kisii town and others are street families who have never been attended to medically.

Figure 6: Simulation of the entire population which shows that ; the graph of infected (I) population the infected humans increase first before treatment is administered but after a short while the graph continue to decrease since part of the population is receiving full medical treatment which is a good measure for disease control. The graph for the Removed (R) individuals continue to increase due to more people accessing treatment and are subjected to regular medical check-ups. The graph for Infectious Carriers (I_C) is decreasing since those who were initially unaware of their condition are showing symptoms and getting treatment and others die before they are diagnosed.

The point of intersection between Infectious Carriers (I_C) and Removed (R) shows a situation where those who are Infectious Carriers are dying without getting any treatment. The point of intersection between Susceptible (S) and Removed (R) shows that all those recovering from the disease are immediately getting re-infected and enter the
stage of Infected (I) hence at this point the population of those infected and Infectious Carriers is too high which is also the point of intersection for the Infected and Infectious Carriers remain susceptible again.

More important, the figure indicates that at least a certain proportion of the population needs to be treated properly in order to avoid deadly typhoid fever in the case of very high density of parasites. Besides that, different levels of treatment strongly influence the fitness between carriers and infectious populations.

V. Conclusion

Typhoid fever continues to be an important cause of illness and death, particularly among children and adolescents in developing countries, where sanitary conditions remain poor. A stable town will be achieved when the basic reproductive number is less than unity i.e \( R_0 < 1 \) and that \( dIc / dt \) remains to be negative. Typhoid fever is becoming a major problem and treatment is becoming increasingly difficult, leading to patients taking longer to recover, suffering more complications and continuing to spread the disease to their family and to their community. A mathematical model for investigating the impact of effects of carriers on the dynamics of typhoid is developed and analyzed. Comprehensive and robust mathematical techniques have been used to analyze the model steady states. It has been established that the model has a disease-free equilibrium which is globally asymptotically stable when the associated reproductive number is less than unity. Sensitivity analysis of the reproductive number has been carried out. Results from the sensitivity analysis of the reproductive number suggest that an increase in human recruitment rate, typhoid transmission by nonsymptomatic and symptomatic individuals, and the proportion of individuals who become nonsymptomatic upon infection have the greatest influence on increasing the magnitude of the associated reproductive number. We also note that treatment has the greatest influence on reducing cumulative typhoid cases. Analytical and numerical results have suggested that an increase of carriers of typhoid cases may outnumber typhoid treatment cases with time, and this will lead to high prevalence of typhoid in the community. The model developed in this paper has limitation(s), which should be acknowledged. Recruited individuals are assumed to be susceptible which might not be the case in some communities. As we have seen, the infectious population is responsive to both changes in treatment as well as changes in the levels of carriers. Increasing the percentage of people receiving treatment causes the infectious population to drop. However, this decrease in the infectious group becomes much less significant as we move from low to high levels of carriers. In other words, treatment can be effective in reducing the number of people that are sick if the effect of carriers is not too high. On the other hand, once effect of carriers becomes prevalent, treatment is less likely to be successful. In this case, mathematical models such as this one can help suggest thresholds in which treatment would be effective and should be used conservatively to avoid creating more problems by aggravating effect of carriers. A cost-benefit analysis of treatment rates can be analyzed using mathematical simulations. In our study, we discovered that increasing treatment yields a smaller decrease of infectious individuals. In other words, doubling the treatment rate may produce significant changes in the size of the infectious subpopulation, but this effectiveness decays as we continue to increase treatment. A balance can be found such that treating a certain percentage of people would yield optimal effectiveness per shilling spent. Throughout our parameter manipulations, we do not change the recovery rate and thus the proportion of people recovering becomes fewer in comparison to those obtaining drugs.

VI. Recommendation

As a result of this research, we are painfully aware of the implications that treatment has on effects of carriers on typhoid fever and all of our simulations have reinforced the strong cause. The following are to be taken into account for us to have a stable kisii town free of typhoid fever:

- There should be proper disposal of the feaces and urine especially the people using bushes-in Nyanchwa hill as toilets and rivers-in town as urinals to prevent the spread of the disease.
- Domestic water should be boiled or chlorinated before drinking to kill the bacteria.
- Hands and cutlery should be washed with clean before eating.
- Fruits should be washed with clean water before being eaten.
- Food handlers should be clean, and should be subjected to regular medical check-ups.
- A lot has to be done in the models in order to ensure that there is no much difference between treating symptoms and creating a more volatile and disease carriage.
- Many more areas of the model can and should be explored to better understand the implications of treatment and how it works.
- Care must be taken when evaluating treatment plans, which is one of the most useful ways to leverage mathematical models.
- Since the simulation program reports the sizes of the susceptible, infected, infective and removed population at every time there is a new event, the program can be used not only to study the outbreak size or to obtain the estimates for the model's parameters, but also to estimate the transmission incidence over time or other parameters of interest such as the outbreak duration distribution.
- The individual distribution can be defined obeying not only individual characteristics but environmental heterogeneities. The environmental effect on the graph can be modeled superimposing a map on the network structure. It is natural to think that for many infectious agents, the individuals in a local network will be more likely to be under similar environmental situations.
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- Masters of science in applied mathematics: (13 completed, 8 ongoing)

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