6.1 INTRODUCTION

Medical workers and health authorities are diverting substantial efforts and resources into trying to predict and control of diseases of many types. Mathematical modeling to these situations play an important role which help to decide how resources are associated. Therefore, it is important to study how a disease will develop and spreads. Direct effect of industrial pollution on the surrounding vegetarian and life can always be determined by estimating the extent of pollution in a given industrial domain or land area. Therefore the pollution may be in terms of chemical or biological gases, fumes, polluted land and water resources.

However, in addition to above types of pollutants there are many indirect pollutants in industrial habitat areas which also cause health hazards. The affected population becomes prone to catch viral and bacterial infections and is susceptible for various diseases. It is also to be noted that the spread of infectious disease is not uniform in the entire industrial population. For a general consideration we take workers of different categories are found because of differences in the financial status. Due to unhygienic equipment, a sort of social pollution is prevalent in much industrial pollution. For this type of population low paid unskilled workers with generally, transmitted
diseases in the industrial area are very common. But the sections other than the above have better living conditions and medical care, the recovery rate is better.


The simplest scenario for an epidemic is one in which the population is split into two parts, one is the infected (who are also all infectious) and the other is part that is
susceptible to being infected. There are no people who are immune and all the infected people are infectious and vice versa. The infection is spread by contact between members of the community in which there is no removal from circulation by death recovery or isolation. Ultimately, therefore all susceptible become infected.

Although these assumptions are simplified for many practical cases, they are applicable to the type of situation where disease is highly infectious but not sufficiently serious for cases to be withdrawn by death or isolation and no infective becomes clear of infection during the main parts of epidemic. This might be the case for some of the milder infections of the upper respiratory tract. To achieve the goal of the model we construct a growth model to deterministic situation with reference to homogeneously mixed group of individuals of size N + a and we suppose that the epidemic is started of at time $t = 0$ by ‘a’ individuals become infected, the remaining N individuals of all being susceptible but as yet uninfected.

We consider a population in which a small number of members become capable of spreading infectious diseases. The population under consideration is divided into disjoint classes will vary with time t. The susceptible $S(t)$ class consists of those individuals who can catch the disease but not yet infective $I(t)$. The infective class consists of those who are transmitting the diseases to other. The removable class $R(t)$ consists of those who are removed from the susceptible – infective interaction by recovery with immunity, isolation or death. The fraction of the total population of these classes are denoted by $S(t)$, $I(t)$ and $R(t)$ respectively as susceptible, infectives and removals.
In India many industrial townships have public hospitals and health centres which are used by the lower cadre of industrial workers. These facilities are provided by the state or voluntary organizations. Due to lack of awareness and other problems the centres themselves are means of population and cause of the spread of infection.

The contagious disease (the sexual transmitted disease) in industrial town is also very common [83]. The infections of these diseases passed on from one person to other either due to use of unhygienic equipment or through sexual contact. Social norms in lower class industrial workers are not observed and a sort of social population is prevailed in many industrial colonies. However such situations are observed only in temporarily migrated unskilled labour and other sections who exploit the situations. Thus for such diseases, the compartments in the population can be easily observed. The sections other than the above have better living conditions and medical care. Therefore the occurrence of these infectious diseases in these sections is less density wise and the recovery rate if possible is better or the epidemiological model.

Various mathematical and statistical investigations are used in medicine for the computations of populations for epidemic models and estimation of new probable infectives. In the present study we have employed effectiveness technique by introducing numerical method using Runge- Kutta- Fehlberg - 5th [ R - K - F ]order method for solving the arising differential equation after using mathematical model. The results for S, I, R are interpreted for the deterministic approach.
6.2 FORMULATION

In formulating the mathematical model we have made the following assumptions.

1. An infectious disease which introduced into a closed population.

2. It is required how many individual will catch the disease.

3. Any individual gets recovered from the disease will have the permanent immunity.

We introduce the population as \( I(t) \) - infectious, \( S(t) \) - susceptible, \( R(t) \) - removable and \( C(t) \) - carriers such that

i. Population consists of \( I \) and \( S \) only.

ii. \( S \) becomes infected and remains infected.

iii. \( I + S = C \) (constant), nobody is leaving the place.

iv. \( I \) and \( S \) are function of \( t \) (time).

v. No control mechanism.

Mathematical study of the infectious diseases including epidemics due to industrial pollution is mostly confined to homogenous population consisting of single group. As for as population model is concerned, we have considered and analyzed biological populations with two or more compartments in the same species. The same model is carried out to investigate on the effect on human population.

In view of the above assumption the classical SIR model for the spread of infections diseases is given by

\[
\frac{dS}{dt} = S' = -\beta SI
\]

\[
\frac{dI}{dt} = I' = \beta SI - \frac{1}{\alpha}I
\]  

(6.1)
\[
\frac{dR}{dt} = R' = \frac{1}{\alpha} I
\]

If recovery does not give immunity, then the model is taken as SIS model, since the individual move from susceptible class to the susceptible class upon recovery. Then from equation (6.1), we choose

\[
\frac{dS}{dt} = -\beta SI + \frac{1}{\alpha} I
\]

\[
\frac{dI}{dt} = \beta SI - \frac{1}{\alpha} I
\]

It follows immediately for the population of fixed size,

\[S + I + R = N + \alpha \quad \text{(constant)}\]

Here \(R\) consists of members removed through recovery with immunity.

Further it is assumed that, the average number of contacts sufficient to transmit infection per infective in unit time is a constant. Then the number of contact per unit area is \(\lambda I\) and the probability that each contact is with susceptible is \(S/K\). Then the infection rate is \(\beta SI\) with \(\beta = \frac{\lambda}{K}\).

By noting that the basic concept in epidemiology is the existence of thresholds. These are critical values for the quantitative assessment such as contact number, population size or vector shows the initial population is infected due to person to person contact otherwise density that must be exceeded in order for an epidemic to occur or for a disease to remain epidemic.

Assuming births only in the susceptible class proportional to the size of the class and deaths in each class proportional to the number of members in the class. Keeping the total population size constant. Then equations (6.1) are modified as,
\[
\frac{dS}{dt} = -\beta SI + \frac{1}{L} (K - S)
\]
\[
\frac{dI}{dt} = \beta SI - \frac{1}{\gamma} I - \frac{1}{L} I
\]
\[
\frac{dR}{dt} = \frac{1}{\gamma} I - \frac{1}{L} R
\]

(6.3)

For the cases of the industrial population, we shall use the compartment modeling to discuss interaction between them.

For the spread of population and infection in industrial population we consider the case of sexually transmitted diseases including AIDS.

We choose

- \(S_p(t)\) - number of members of a population susceptible to pollution.
- \(S_d(t)\) - number of members of a population disease to pollution.
- \(I_p(t)\) - number of members of population infectives to pollution.
- \(I_d(t)\) - number of members of a population in pollution infectives to disease.
- \(R_e(t)\) - number of members removed from being reinfected.

\(\beta_p(t)\) - pollution infection rate.
\(\beta_d(t)\) - disease infection rate.

Then the nonlinear differential equations are,

\[
\frac{dS_p}{dt} = -\beta_p S_p I_p
\]
\[
\frac{dI_p}{dt} = \beta_p S_p I_p
\]
\[
\frac{dS_d}{dt} = -\beta_d S_d I_d
\]

(6.4)
\[
\frac{dI_d}{dt} = \beta_d S_d I_d - \gamma I_d \quad \text{and}
\]
\[
\frac{dR_d}{dt} = \gamma I_d
\]

Initial conditions are,
\[
S_p(0) = S_{p_0} > 0 \quad I_p(0) = I_{p_0} > 0 \quad R_d(0) = R_{d_0} = 0 \quad (6.5)
\]
\[
S_d(0) = S_{d_0} > 0 \quad I_d(0) = I_{d_0} > 0
\]

We let \( N(t) \) denote the total population size with
\[
S_p + I_p = N + a \quad (6.6)
\]
\[
S_d + I_d + R_e = I_d
\]

It is required to assume that the individuals under the influence of pollution remain infected for ever. There is no recovery from the pollution. Also it is to be considered that, the effect of pollution on the human population under consideration is assumed to be much slow in comparison to the effect of virus. A person infected by pollutants immediately receives the disease infection if exposed to the same. The number of primary infectives does not vary significantly during the spread of infectious diseases.

6.3 ANALYSIS

For the system of equations (6.4), we can estimate the values \( S_p, S_d, I_p, I_d \) at any time intervals of their initial values without removals.

Solving for \( S_p, I_p, S_d, I_d \) equations (6.4) and (6.6) in reference to growth model.

Solving for \( S_p \) with separating the variables with reference to equations (6.4) and (6.6) and using partial fractions, we obtain,
\[
\frac{1}{N_p} \left[ \log \left( \frac{S_p}{N_p - S_p} \right) \right] = -\beta_p t + C_1 \tag{6.7}
\]

At \( t = 0, I = 1, S = N \)

\[
\frac{1}{N_p} \left[ \log \left( \frac{S_p}{N_p - S_p} \right) - \log \left( \frac{S_{p_0}}{N_p - S_{p_0}} \right) \right] = -\beta_p t
\]

\[
\frac{S_p}{S_{p_0}} \frac{(N_p - S_{p_0})}{(N_p - S_p)} = e^{-\beta_p t N_p} \tag{6.8}
\]

\[
S_p = \frac{(N_p - S_{p_0}) e^{-\beta_p t N_p}}{K \quad \text{where} \quad K = \frac{(N_p - S_{p_0})}{S_{p_0}} \tag{6.9}
\]

Solving for \( I_p \) with separating the variables with reference to equations (6.4) and (6.6) using partial fractions, we obtain,

\[
\frac{1}{N_p} \left[ \log \left( \frac{I_p}{N_p - I_p} \right) \right] = \beta_p t + C_2 \tag{6.10}
\]

At \( t = 0, I = 1, S = N \)

\[
\frac{1}{N_p} \left[ \log \left( \frac{I_p}{N_p - I_p} \right) - \log \left( \frac{I_{p_0}}{N_p - I_{p_0}} \right) \right] = \beta_p t \tag{6.11}
\]

\[
\frac{I_p}{I_{p_0}} \frac{(N_p - I_{p_0})}{(N_p - I_p)} = e^{-\beta_p t N_p} \tag{6.12}
\]

\[
I_p = \frac{(N_p - I_p) e^{-\beta_p t N_p}}{K \quad \text{where} \quad K = \frac{(N_p - I_{p_0})}{I_{p_0}} \tag{6.13}
\]

Solving for \( S_d \) with separating the variables with reference to equations (6.4) and (6.6) using partial fractions, we obtain,
\[
\frac{1}{N_d} \left[ \log \left( \frac{S_d}{N_d - S_d} \right) - \log \left( \frac{S_{d_0}}{N_d - S_{d_0}} \right) \right] = -\beta_d t + C_3
\]  

(6.14)

At \( t = 0, I_0 = 1, S = N \)

\[
\frac{1}{N_d} \left[ \log \left( \frac{S_d}{N_d - S_d} \right) - \log \left( \frac{S_{d_0}}{N_d - S_{d_0}} \right) \right] = -\beta_d t
\]  

(6.15)

\[
\frac{S_d (N_d - S_{d_0})}{S_{d_0} (N_d - S_d)} = e^{-\beta_d t \cdot N_d}
\]  

(6.16)

\[
S_d = \left( \frac{N_d - S_{d_0}}{K} \right) \frac{e^{-\beta_d t \cdot N_d}}{S_{d_0}}
\]  

where \( K = \frac{(N_d - S_{d_0})}{S_{d_0}} \)  

(6.17)

Solving for \( I_4 \) with reference to equations (6.4) and (6.6)

\[
\frac{dI_d}{dt} = \beta_d S_d I_d - \gamma I_d \text{ with } I_d + S_d = N_d
\]

\[
\frac{dI_d}{dt} = \beta_d (N_d - I_d) I_d - \gamma I_d
\]

\[
\frac{dI_d}{dt} = \beta_d N_d I_d - \beta_d I_d^2 - \gamma I_d
\]

\[
\frac{1}{I_d^2} \frac{dI_d}{dt} = (\beta_d N_d - \gamma) \frac{1}{I_d} = \beta_d
\]  

(6.18)

Equation (6.18) is Bernoulli’s non linear differential equation then we find the integrating factor, the solution for \( I_4 \) is given by

\[
\frac{1}{I_d} e^{(\beta_d N_d - \gamma) t} = \int e^{(\beta_d N_d - \gamma) t} \beta_d \, dt + C
\]

\[
I_d = \frac{\beta_d N_d - \gamma}{\beta_d + (\beta_d N_d - \gamma) e^{-\gamma} (\beta_d N_d - \gamma)}
\]  

(6.19)
We solve the differential equations for $I_p$ and $I_d$ from equation (6.4) using analytical approach to achieve the exact solution.

\[
\frac{dI_p}{dt} = \beta_p S_p I_p \text{ with } I_p + S_p = N + a
\]

\[
\frac{dI_p}{dt} = \beta_p [N + a - I_p] I_p
\]

\[
\frac{1}{I_p^2} \frac{dI_p}{dt} - \beta_p (N + a) \frac{1}{I_p} = -\beta_p
\]

The above equation is Bernoulli's non linear differential equation; the solution for $I_p$ is,

\[
I_p = \frac{N + a}{(N + a) c e^{\beta_p(N+a)t} + 1}
\]

Solving for $I_d$,

\[
\frac{dI_d}{dt} = \beta_d S_d I_d - \gamma_d \text{ with } I_d + S_d = N + a
\]

\[
\frac{dI_d}{dt} = \beta_d (N + a) I_d - \beta_d I_d^2 - \gamma I_d
\]

\[
\frac{1}{I_d^2} \frac{dI_d}{dt} - \left(\frac{\beta_d (N + a) - \gamma}{I_d}\right) = \beta_d
\]

This equation is Bernoulli's non linear differential equation; the solution for $I_d$ is given by,

\[
I_d = \frac{\beta_d (N + a) - \gamma}{\beta_d + \left[\beta_d(N + a) - \gamma\right] c e^{-[\beta_d(N+a)-\gamma]t}}
\]

Expressions for the $I_p$ and $I_d$ show the number of infectives at any time $t$ for infectious due to industrial pollution and for infectious due to diseased one, depends only
on the initial condition at \( t = 0 \) and the infection rate \( \beta \). The equation (6.23) gives the computations for deterministic model.

Solving by numerical method the equation \( \frac{dl_p}{dt} = \beta_p S_p I_p \) from equations (6.4) with \( I_p + S_p = N + a, \ N = 20, \ a = 2, \ \beta_p = 0.02, \ h = 1 \)

Using Runge - Kutta Fehlberg (R - K - F) fifth order or embebbed Runge - Kutta method. We get a clear advantage that it requires six functional evaluations per step.

\[
(I_p)_{i+1} = (I_p)_i + \frac{2825}{27648} \left\{ f(t, I_p) \right\} + \frac{18575}{48384} \left\{ f \left( t + \frac{3}{10} h, I_p + \frac{3}{40} k_1 h + \frac{9}{40} k_2 h \right) \right\} + \frac{13525}{55296} \left\{ f \left( t + \frac{3}{5} h, I_p + \frac{3}{10} k_1 h + \frac{9}{10} k_2 h + \frac{6}{5} k_3 h \right) \right\} + \frac{277}{14336} \left\{ f \left( t + h, I_p - \frac{11}{54} k_1 h + \frac{5}{2} k_2 h - \frac{70}{27} k_3 h + \frac{35}{27} k_4 h \right) \right\} + \frac{1}{4} \left\{ f \left( t + \frac{7}{8} h, I_p + \frac{1631}{55296} k_1 h + \frac{175}{512} k_2 h + \frac{44275}{110592} k_3 h + \frac{253}{4096} k_4 h \right) \right\} + \frac{1}{10} \left\{ f \left( t + \frac{1}{5} h, I_p + \frac{1}{5} k_1 h \right) \right\} + \frac{1}{10} \left\{ f \left( t + \frac{1}{10} h, I_p + \frac{3}{40} k_1 h + \frac{9}{40} k_2 h \right) \right\} + \frac{1}{10} \left\{ f \left( t + \frac{3}{5} h, I_p + \frac{3}{10} k_1 h - \frac{9}{10} k_2 h + \frac{6}{5} k_3 h \right) \right\} + \frac{1}{10} \left\{ f \left( t + h, I_p - \frac{11}{54} k_1 h + \frac{5}{2} k_2 h - \frac{70}{27} k_3 h + \frac{35}{27} k_4 h \right) \right\}
\]

Where \( k_1 = f(t, I_p) \)

\[ k_2 = f \left( t + \frac{1}{5} h, I_p + \frac{1}{5} k_1 h \right) \]

\[ k_3 = f \left( t + \frac{3}{10} h, I_p + \frac{3}{40} k_1 h + \frac{9}{40} k_2 h \right) \]

\[ k_4 = f \left( t + \frac{3}{5} h, I_p + \frac{3}{10} k_1 h - \frac{9}{10} k_2 h + \frac{6}{5} k_3 h \right) \]

\[ k_5 = f \left( t + h, I_p - \frac{11}{54} k_1 h + \frac{5}{2} k_2 h - \frac{70}{27} k_3 h + \frac{35}{27} k_4 h \right) \]
Solving by numerical method the equation \( \frac{di_d}{dt} = \beta_d S_d I_d - \gamma I_d \) from equations (6.4)

with \( I_d + S_d + R_e = N + a \), \( N = 20 \), \( a = 2 \), \( \beta_d = 0.02 \), \( h = 1 \)

\[
(l_d)_{i+1} = (l_d)_i + \begin{bmatrix}
\frac{2825}{27648} f(t, I_d) + \frac{18575}{48384} f\left(t + \frac{3}{10}, I_d + \frac{3}{20} k_1 h + \frac{9}{20} k_2 h\right) \\
+ \frac{13525}{55296} f\left(t + \frac{3}{5}, I_d + \frac{3}{10} k_1 h - \frac{9}{10} k_2 h + \frac{6}{5} k_3 h\right) + \\
\frac{277}{14336} f\left(t + h, I_d - \frac{11}{54} k_1 h + \frac{5}{2} k_2 h - \frac{70}{27} k_3 h + \frac{35}{27} k_4 h\right) \\
+ \frac{1}{4} f\left(\frac{14325}{110592} k_4 h + \frac{253}{4096} k_5 h\right)
\end{bmatrix} h
\]

(6.25)

Where \( k_1 = f(t, I_d) \), \( k_2 = f\left(t + \frac{1}{5} h, I_d + \frac{1}{5} k_1 h\right) \)

\( k_3 = f\left(t + \frac{3}{10} h, I_d + \frac{3}{4} k_1 h + \frac{9}{4} k_2 h\right) \)

\( k_4 = f\left(t + \frac{3}{5} h, I_d + \frac{3}{10} k_1 h - \frac{9}{10} k_2 h + \frac{6}{5} k_3 h\right) \)

\( k_5 = f\left(t + h, I_d - \frac{11}{54} k_1 h + \frac{5}{2} k_2 h - \frac{70}{27} k_3 h + \frac{35}{27} k_4 h\right) \)

\( k_6 = f\left(t + \frac{7}{8} h, I_d + \frac{1631}{55296} k_1 h + \frac{175}{512} k_2 h + \frac{575}{13824} k_3 h + \frac{44275}{110592} k_4 h + \frac{253}{4096} k_5 h\right) \)

The value of 'h' is so chosen that (h = 1) the approximations give numerical computations are quite satisfactory.
6.4 RESULTS AND DISCUSSION

Mathematical model for spread of infectious diseases using growth model predicts the number of infectives in the population where the disease is highly infectious but not sufficiently serious. The computations for \( I(t) \) showed that the infected individuals remain in the population to spread the disease. The numerical solution by Runge-Kutta Fehlberg method gives the effective accurate values of the parameters to explain the spread of infectious disease when compared with computations by analytical method and exact method. The computations for \( S(t) \) shows that when one in the total population is infected and other is the part that, few members are susceptible to become infected. For no removal case or the recovery rate is low, we can notice the long term effect of pollution in industrial towns on the human population in terms of spread of diseases. If the population parameters \( I(t), S(t) \) are expressed with economic, social and other factors then we can show the expressions for solutions of systems of non linear ordinary differential equations as step by step approximations for \( I(t) \) and \( S(t) \).

It is reasonable to assume that the individuals under the influence of pollutants remain infected for ever this means there is no recovery from the pollution while the recovery of diseases infected members has been considered. The individuals susceptible to disease are those who are already under the influence of pollution. If the effect of pollution on the human population under normal conditions is assumed to be such slow in comparison to the effect of virus. A susceptible becomes infective after a long exposure to the pollution, while a person infected by pollutants immediately receives the disease if exposed to the same. Therefore we assumed that the number of primary infectives do not vary significantly during the spread of infectious disease. The investigation proposes the
rate of infection due to industrial pollutants among I(t), S(t) and R(t) depends on normal conditions as slow but in other conditions it will be little higher and the recovery cases lead to critical case. However for disease in which the recovery from the disease does not mean recovery from the pollution too for some members but for the others permanent recovery implies from disease and as well as from industrial pollution.

Fig.6.1 depicts the flow chart of industrial pollution with reference to industrial pollution view point and biological view point.

Fig. 6.2 shows the nonlinear growth of population infectious to pollution at various time intervals, R - K - F method gives better approximations as shown in figure.

Fig.6.3 gives the better growth of population disease with time intervals by logistic model equations (6.7, 6.10, 6.14, 6.21, and 6.23)

Fig.6.4 explains the exponentially increasing trend of infectives due to industrial pollution ($\beta_p > 0$) and becomes constant for ($\beta_p = 0$)

6.5 CONCLUSION

The study gives an idea that the long term effect of pollution and the population in terms of spread of diseases will cause the various diseases due to increase of industrial pollution and the increase of transmitted diseases. A set of non-linear ordinary differential equations describe solutions of the logistic equations and an attempt by R- K - F method to compute numerically the closeness of the effect of diseases.
Fig. 6.1 Flow chart of industrial pollution and mathematical model
Fig. 6.2 Population infectives to pollution Vs Time

Fig. 6.3 Population disease to pollution Vs Time
Fig. 6.4 Logistic curve for number of infectives against various time intervals

\((\beta_p > 0 \text{ and } \beta_p = 0)\)
Photograph 6.1. Area of industrial pollution at Davangere (Location -1)
(Location of air pollution due to the burning of used tyres releasing \( \text{NO}_2 \), \( \text{CO}_2 \), \( \text{CO} \), \( \text{SO}_2 \) at mandkki batti – Azad Nagar, Davangere)

Photograph 6.2. Area of industrial pollution at Davangere (Location -2)
(Location of air pollution due to the burning of used tyres releasing \( \text{NO}_2 \), \( \text{CO}_2 \), \( \text{CO} \), \( \text{SO}_2 \) at mandkki batti – Azad Nagar, Davangere)
Photograph 6.3. Area of industrial pollution at Davangere (Location -3).
(Sepage of drainage water (untreated) behind the industrial area)

Photograph 6.4. Area of industrial pollution at Davangere (Location -4).
(Sepage of drainage water (untreated) behind the industrial area)