

## A. Mathematical Model of Gang Membership and Control

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**Abstract:** *There is an increasing growth in gang membership worldwide and especially our nation Nigeria, this prompted the study as a means to contribute to the social stability of the country. We considered gang membership as an infectious disease that spreads throughout a given population. We formulated a modified SEIR model to look into the transmission dynamics of gang by bringing into consideration, control techniques as measures to reduce the spread and activities of gang members. In analyzing the model, we proved that the disease free-equilibrium is locally and globally asymptotically stable when the reproduction number  $R_0 < 1$  and unstable when  $R_0 > 1$ . The transmission pattern shows that investments in job provisions, technical crafts and programs geared towards recreation and other after-school activities factored into a compartment continues to be the best resource in curbing gang membership and its activities considering from rehabilitation point of view in the numerical simulation.*

**Keywords:** *Mathematical model, reproduction number, infectious disease, transmission dynamics and stability analysis.*

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### 1.0 Introduction

Gangs are self-identified, organized groups of youths/adults, banded together under common interests and a common leader. Gangs are defined by their willingness to use violence to further economic and social goals regarded as menacing to society (M. B. Short, *et al.*, 2014; Lee *et al.*, 2011). The economic goals of gangs maybe related to the establishment and control of drugs or other illicit markets but the social goals of gangs are equal. If not more important: the need to build a reputation that should be feared and respected. Due to these quests of gang members, they engage in crimes like: armed robbery, homicide, auto theft and drain cities, drug dealing, and pose safety threats to community members and governments of tight resources (Hegemann, *et al.*, 2011). It is well known that peers and their pressures play a central role in adolescence as these facts have been ascertained by many sociologists and psychologists. As youths move through adolescence, they gravitate towards cliques that provide support, assurance, protection and direction. Now, when a group provides the social and emotional basis/platform for antisocial behaviours, this group becomes susceptible to gang lifestyle. Susceptibility to peer pressure reaches its highest level in people with low confidence and a lack of social interaction skills (Young *et al.*, 2007). This attacks the troubled youth more and people living in an environment of poverty (ghettos), who have low academic achievements, low school commitment, or people who have gone through the educational system but are still finding issues with their footings. Some youths join a gang by thinking that the gang represents their disadvantaged structural position in society, hence justifying their ill actions as a resolution to their problem. Gang members

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have secluded locations known as set spaces, where they spend and plan their schedule from. We therefore can consider a gang as a node embedded in Euclidean space. In this lies the strength of mathematical modelling. Mathematical models of infectious diseases predict future outbreaks of the disease and suggest intervention measures that have to be taken to control the disease. This idea helps us to consider this work (gang membership) as an infection that multiplies due to interaction of young naïve youths with the delinquent ones via verbal and non-verbal but enticing means of communication. Sooknanan *et al* (2013) modelled the impact of various crime-fighting strategies to determine their influence on gang membership as a means of assisting policy-makers in the development of effective gang policy controls. Young *et al* (2013), focused on contagion of gang membership and juvenile crimes among youths who are particularly susceptible to peer pressure. Through study, it is known that youths who agree to join a gang often endure violent initiation rituals such as beatings, murdering a way of showcasing hardness and other forms of malicious acts. When they come in, are faced with incarceration and other inhumane ways of living. Issues about gangs have become complex as there are institutionalized gangs and non-institutionalized gangs. The institutionalized gangs are born out of the quest for drive to see that the government lives up to its standards and the implementation of the promises made by the government on the assumption of duty. Most of these institutionalized gangs have existed over generations and have a strong organizational structure. But due to the inability to properly management of there quests of theirs by the government, legal bodies and armed forces are now forced into violent behaviours that become menacing to society. Some of these gangs are: the institutionalized gangs of Chicago and the institutionalized gangs of Nigeria such as the Eastern Society Network (ESN), the Oduduwa of the Yoruba tribe and Myatti Allah of the Hausa tribe and the non-institutionalized gangs are the cult groups seen in the colleges/universities of higher learning and the occult groups formed by the grand

masters from the lower platforms. There have been several models on gang membership and its spreading, and some notable studies examining the effectiveness of prevention strategies for at-risk youths. The fear, violence and horror that go with gangs are enormous and call for serious attention. As the government seeks a solution to the menace of gangs and gangsters', we in this work form a model that shows the mode of initiation and the corrective tool of counselling, proper education and good youthful upbringing as the control measure for gang membership.

### 1.1 Model assumptions

- (i). Association to delinquent peers is one of the strongest risk factors for gang membership. This association therefore leads to an infection hypothesis of growth in the gang subpopulation.
- (ii). Our work considers the dynamics of gang formation in a susceptible heterogeneous population.
- (iii). There is homogeneous mixing of members of the population.
- (iv). Due to yearly formation of gang, recruits exist or inflow of susceptible youths as well as exist via rehabilitation, permitting a demographic process to take place.

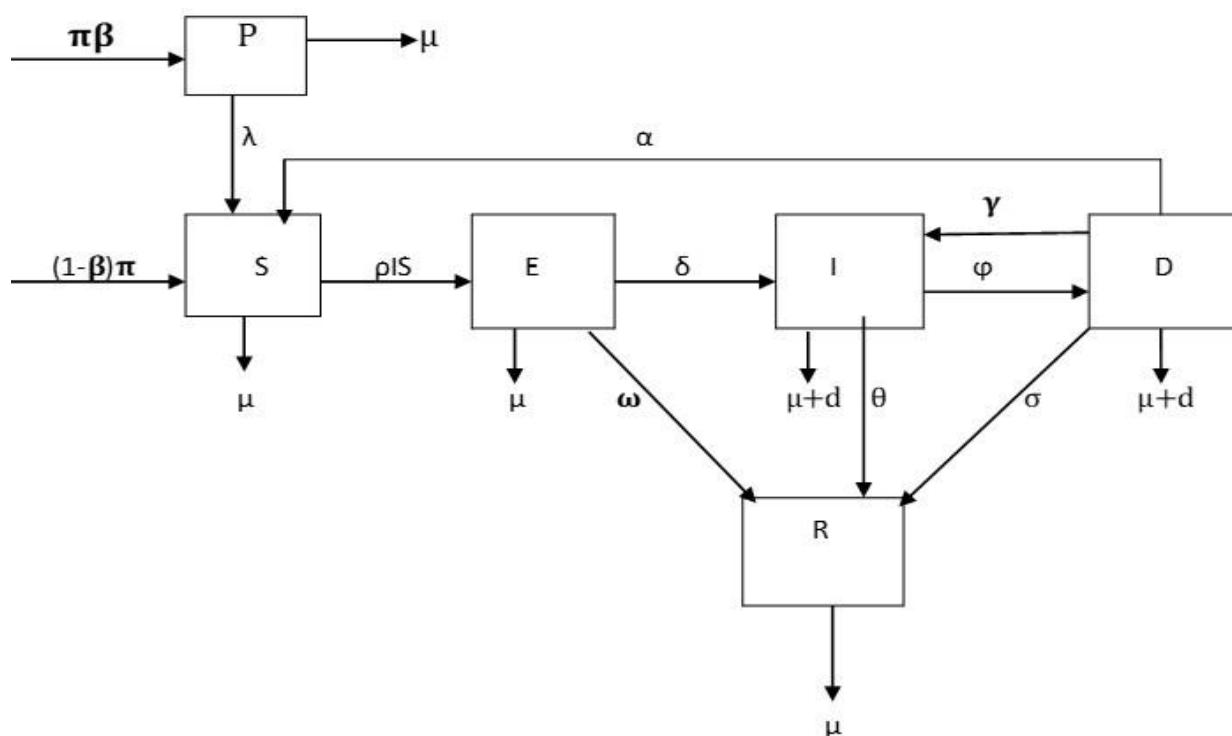
### 1.2 Model formulation

We form a deterministic epidemiological model with a system of six ordinary differential equations to examine the dynamics of the system. The total population ( $N$ ) at a given time  $t$  is divided into six classes as follows: The class ( $P$ ) represents normal youths within the population with no interest in gang membership. The class ( $S$ ) represents youths susceptible to gang membership. The class ( $E$ ) represents youths exposed to gang involvement or fring members, who have not pledged their allegiance to the group. The class ( $I$ ) represents youths who are core gang members and their leaders. Individuals in class ( $D$ ) are youths who have been sent to correctional facilities via conviction of crimes and sentencing. The class ( $R$ ) individuals are youths who recovered through psychological rehabilitation. So that the total population ( $N(t)$ ) is represented as follows:



$(N) = (P) + (S) + (E) + (I) + (D) + (R)$   
 Youths are recruited into the population through immigration and maturity from childhood to youthful age at a rate  $\pi$ . A proportion of these youths moves into the normal class at a rate  $\beta$ . Due to weakness of inner strength, via family problems and other issues of life, lose their ability to continue as chaste youths and become susceptible to gang lifestyle at a rate  $\lambda$ . Youths in this class, age out of the population through death, manhood and family formation at a rate  $\mu$ . Recruitment into class S, comes from the proportion of youths taken into the population who do not think of maintaining a normal youthful life but have yearnings of becoming gang members at a rate  $(1 - \beta)\pi$  and youths weaned from normal class at a rate  $\lambda$ . Susceptible youths become exposed to gang lifestyle through verbal communication and nonverbal but enticing means of communication with the core gang

members at a rate  $\rho$ . When released from the correctional facility, some youth do not possess the ability to live right and thus relapse to susceptible class at a rate  $\alpha$  and age out at a rate  $\mu$ . Peer pressure, and mingling with delinquent youths get susceptible youths to be exposed to gang activities via their yearnings and so become inherent to gang membership at a rate  $\rho$ . Intense interaction with delinquent youths and core gang members at a rate  $\delta$  brings exposed youths into gang membership. At this stage though, counselling, proper attention, or monitoring, the exposed youths can be salvaged psychologically into class R at a rate  $\omega$  and youths in the exposed class, age out of the population through manhood and family formation at a rate  $\mu$ . Youths are talked and or enticed into gang membership at a rate  $\delta$ , led by gang peers and delinquent youths.



**Fig. 1: The compartmental flow diagram of the model, showing the rate of infectiousness ranging from the sub-classes in the population.**

Through proper education on its benefits and effects, they are removed from this class to the recovered/rehabilitated class at the rate  $\theta$  while adamant to corrective measures and lifestyle lands them into correctional facilities through crime conviction and sentencing at a rate  $\phi$ .

Youths in correctional facilities when released relapse back to I class at a rate  $\gamma$ . They age out at a rate  $\mu$  or die as members due to gang activities at a rate  $d$ . Recruitment into correctional homes/facilities is done through crime convictions at a rate  $\phi$ . When proper



counseling is done on the youths in this class in or at the point of release from the facility with good character change, they can be received into the recovered class at a rate  $\sigma$  or relapse back to the susceptible class at a rate  $\alpha$  or relapse back to core gang membership at a rate  $\gamma$  when released. They age out at a rate  $\mu$  or die as members due to gang activities at a rate  $d$ . Exposed youths withdraw from gangster membership at a rate  $\omega$ . Core gang members are recovered at a rate  $\theta$ . Gang members in correctional facilities are recovered at a rate  $\sigma$  when released from such homes. Youths age out from this class through manhood and family formation at a rate  $\mu$ .

### 1.3 Model equations

The interaction between model classes can be represented by the following system of first-order differential equations:

$$\frac{dP}{dt} = \beta\pi - (\lambda + \mu) \tag{1}$$

$$\frac{dS}{dt} = (1 - \beta)\pi + \alpha D + \lambda P - (\rho I + \mu)S \tag{2}$$

$$\frac{dE}{dt} = \rho IS - (\delta + \omega + \mu)E \tag{3}$$

$$\frac{dI}{dt} = \delta E + \gamma D - (\varphi + \theta + \mu + d)I \tag{4}$$

$$\frac{dD}{dt} = \varphi I - (\alpha + \sigma + \gamma + \mu + d)D \tag{5}$$

$$\frac{dR}{dt} = \omega E + \theta I + \sigma D - \mu R \tag{6}$$

**Table 1: Variables and parameters description**

<b>P</b>	<b>Normal youths.</b>
<b>S</b>	Susceptible youths.
<b>E</b>	Exposed or fringe members.
<b>I</b>	Core gang members.
<b>D</b>	Gang members in correctional facilities through convictions and sentencing to crimes.
<b>R</b>	Individuals recovered from gang membership via psychological rehabilitation.
<b><math>\Pi</math></b>	The general rate at which youths come into the population.
<b><math>\beta</math></b>	The proportion of individuals recruited into the normal youth compartment.
<b><math>(1 - \beta)</math></b>	The complementary proportion of youths that move into the susceptible youth compartment.
<b><math>d</math></b>	Death rate caused by gang activities
<b><math>\alpha</math></b>	The rate at which youths in D compartment relapse back to S compartment after release.
<b><math>\gamma</math></b>	The rate at which youths in D compartment relapse back to I compartment after release.
<b><math>\sigma</math></b>	The rate at which youths in D compartment get rehabilitated.
<b><math>\theta</math></b>	Per capita rate of rehabilitation of core gang members.
<b><math>\omega</math></b>	Salvaging rate of exposed youths.
<b><math>\mu</math></b>	The rate at which youths age out of the population
<b><math>\lambda</math></b>	Transition rate from normal youth compartment to susceptible compartment.
<b><math>\varphi</math></b>	The rate at which core gang members are sent to correctional facilities.
<b><math>\rho</math></b>	Effective contact rate with G, leading to gang membership
<b><math>\delta</math></b>	The rate at which the exposed youths move to the core gang class.

In analyzing the model, we considered first the invariant nature of the model and the positivity of the solution of the model which shows that all solutions of system (1.1) – (1.6) that originate in  $\mathfrak{R}_+^6$  are bounded and eventually enter the attracting set region  $\Omega = \{(P, S, E, I, D, R) \in \mathfrak{R}_+^6 : N \leq \frac{\pi}{\mu}\}$ . This can be found in full detail in Appendix A and B.

## 2.0 The Model Analysis and Results

### 2.1 The disease free equilibrium (DFE)

To find the disease free-equilibrium denoted by  $E_0$ , we equate the right-hand side of the model to zero and solve for the non-infected class, evaluating it at  $E = I = D = R = 0$ , thus giving:



$$E_0 = (P, S, E, I, D, R) = \left( \frac{\beta\pi}{(\lambda+\mu)}, \frac{\pi(1+\lambda-\beta)}{(\lambda+\mu)}, 0, 0, 0, 0 \right) \quad (7)$$

### 2.2 The basic reproduction number

The basic reproduction number is the threshold parameter that governs the spread of disease. To obtain this, we use the next-generation matrix method so that the spectral radius of the Define  $E_0 = (E, I, D)$

$$f = \begin{pmatrix} \rho IS \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad v = \begin{pmatrix} -(\delta + \omega + \mu)E \\ \delta E + \gamma D - (\varphi + \theta + \mu + d)I \\ \varphi I - (\alpha + \sigma + \gamma + \mu + d)D \end{pmatrix}$$

Taking the partial derivatives of  $f$  and  $v$  at disease free-equilibrium state, we obtain the following:

$$f_1 = \rho IS - (\delta + \omega + \mu)E$$

$$f_2 = \delta E + \gamma D - (\varphi + \theta + \mu + d)I$$

$$f_3 = \varphi I - (\alpha + \sigma + \gamma + \mu + d)D$$

Taking the partial derivatives, we get:

$$\frac{\partial f_1}{\partial S} = \rho I, \frac{\partial f_1}{\partial I} = \rho S, \frac{\partial f_1}{\partial E} = -(\delta + \omega + \mu) \quad \text{and} \quad \frac{\partial f_1}{\partial P} = \frac{\partial f_1}{\partial D} = \frac{\partial f_1}{\partial R} = 0 < \infty$$

$$\frac{\partial f_2}{\partial E} = \delta, \frac{\partial f_2}{\partial D} = \gamma, \frac{\partial f_2}{\partial I} = -(\varphi + \theta + \mu + d) \quad \text{and} \quad \frac{\partial f_2}{\partial R} = \frac{\partial f_2}{\partial S} = \frac{\partial f_2}{\partial P} = 0 < \infty$$

$$\frac{\partial f_3}{\partial I} = \varphi, \frac{\partial f_3}{\partial D} = -(\alpha + \sigma + \gamma + \mu + d) \quad \text{and} \quad \frac{\partial f_3}{\partial P} = \frac{\partial f_3}{\partial S} = \frac{\partial f_3}{\partial E} = \frac{\partial f_3}{\partial R} = 0 < \infty$$

So that

$$F = \begin{pmatrix} 0 & \frac{\pi\rho(1+\lambda-\beta)}{(\lambda+\mu)} & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (8)$$

$$V = \begin{pmatrix} (\delta + \omega + \mu) & 0 & 0 \\ -\delta & (\varphi + \theta + \mu + d) & -\gamma \\ 0 & -\varphi & (\alpha + \sigma + \gamma + \mu + d) \end{pmatrix} \quad (9)$$

Let

$$F = \begin{pmatrix} 0 & a_1 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} b_1 & 0 & 0 \\ -b_2 & b_3 & -b_4 \\ 0 & -b_5 & b_6 \end{pmatrix}$$

Where

$$a_1 = \frac{\pi(1 + \lambda - \beta)}{(\lambda + \mu)}, b_1 = (\delta + \omega + \mu), b_2 = -\delta, b_3 = (\varphi + \theta + \mu + d), b_4 = -\gamma, b_5 = -\varphi, b_6 = (\alpha + \sigma + \gamma + \mu + d)$$

$$V^{-1} = \begin{pmatrix} \frac{1}{b_1} & 0 & 0 \\ \frac{b_2 b_6}{(b_1 b_3 b_6 - b_1 b_4 b_5)} & \frac{b_6}{(b_3 b_6 - b_4 b_5)} & \frac{b_3}{(b_3 b_6 - b_4 b_5)} \\ \frac{b_2 b_5}{(b_1 b_3 b_6 - b_1 b_4 b_5)} & \frac{b_5}{(b_3 b_6 - b_4 b_5)} & \frac{b_3}{(b_3 b_6 - b_4 b_5)} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} \frac{a_1 b_2 b_6}{(b_1 b_3 b_6 - b_1 b_4 b_5)} & \frac{a_1 b_6}{(b_3 b_6 - b_4 b_5)} & \frac{a_1 b_4}{(b_3 b_6 - b_4 b_5)} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The largest Eigen value becomes the reproduction number, giving:

$$R_0 = \frac{a_1 b_2 b_6}{(b_1 b_3 b_6 - b_1 b_4 b_5)} \quad (10)$$

next-generation matrix is given by  $\rho(FV^{-1})$ , where  $F$  is the Jacobian of  $f_i$ , and  $f_i$  is the rate of appearance of new infection in compartment  $i$  and  $V$  is the Jacobian of  $v_j$  where  $v_j$  is the rate of transfer of infections from the infected compartment to another compartment  $j$ , all represented in a matrix form.



If  $R_0 < 1$  we obtain asymptotic stability of the model otherwise, instability.

### 2.3 Stability analysis of the disease free- equilibrium

#### 2.3.1 Local stability analysis of the model

In calculating the local stability of the model, we look at the behaviour of the population near the equilibrium point.

#### Theorem 2

The disease free-equilibrium of the model is locally asymptotically stable if  $R_0 < 1$ , otherwise unstable.

#### Proof

The Jacobian matrix of the model at disease free-equilibrium is given by: Let

$$f_1 = \beta\pi - (\lambda + \mu)P$$

$$f_2 = (1 - \beta)\pi + \alpha D + \lambda P - (\rho I + \mu)S$$

$$f_3 = \rho IS - (\delta + \omega + \mu)E$$

$$f_4 = \delta E + \gamma D - (\varphi + \theta + \mu + d)I$$

$$f_5 = \varphi I - (\alpha + \sigma + \gamma + \mu + d)D$$

$$f_6 = \omega E + \theta I + \sigma D - \mu R$$

So that

$$\frac{\partial f_1}{\partial P} = -(\lambda + \mu) \text{ and } \frac{\partial f_1}{\partial S} = \frac{\partial f_1}{\partial E} = \frac{\partial f_1}{\partial I} = \frac{\partial f_1}{\partial D} = \frac{\partial f_1}{\partial R} = 0$$

$$\frac{\partial f_2}{\partial S} = -(\rho I + \mu), \frac{\partial f_2}{\partial I} = \rho S, \frac{\partial f_2}{\partial D} = \alpha, \frac{\partial f_2}{\partial P} = \lambda \text{ and } \frac{\partial f_2}{\partial E} = \frac{\partial f_2}{\partial R} = 0$$

$$\frac{\partial f_3}{\partial S} = \rho I, \frac{\partial f_3}{\partial I} = \rho S, \frac{\partial f_3}{\partial E} = -(\delta + \omega + \mu) \text{ and } \frac{\partial f_3}{\partial P} = \frac{\partial f_3}{\partial D} = \frac{\partial f_3}{\partial R} = 0$$

$$\frac{\partial f_4}{\partial E} = \delta, \frac{\partial f_4}{\partial D} = \gamma, \frac{\partial f_4}{\partial I} = -(\varphi + \theta + \mu + d) \text{ and } \frac{\partial f_4}{\partial R} = \frac{\partial f_4}{\partial S} = \frac{\partial f_4}{\partial P} = 0$$

$$\frac{\partial f_5}{\partial I} = \varphi, \frac{\partial f_5}{\partial D} = -(\alpha + \sigma + \gamma + \mu + d) \text{ and } \frac{\partial f_5}{\partial P} = \frac{\partial f_5}{\partial S} = \frac{\partial f_5}{\partial E} = \frac{\partial f_5}{\partial R} = 0$$

$$\frac{\partial f_6}{\partial E} = \omega, \frac{\partial f_6}{\partial I} = \theta, \frac{\partial f_6}{\partial R} = -\mu, \frac{\partial f_6}{\partial D} = \sigma \text{ and } \frac{\partial f_6}{\partial S} = \frac{\partial f_6}{\partial P} = 0$$

$$JE_0 = \begin{pmatrix} -p_1 & 0 & 0 & 0 & 0 & 0 \\ p_2 & -p_3 & 0 & p_4 & p_5 & 0 \\ 0 & 0 & -p_6 & p_7 & 0 & 0 \\ 0 & 0 & p_8 & -p_9 & p_{10} & 0 \\ 0 & 0 & 0 & p_{11} & -p_{12} & 0 \\ 0 & 0 & p_{13} & p_{14} & p_{15} & p_{16} \end{pmatrix}$$

Using elementary row transform, the eigenvalues become:

$$\lambda_1 = -\mu, \lambda_2 = -(\lambda + \mu), \lambda_3 = -(\alpha + \sigma + \gamma + \mu + d), \lambda_4 = -(\varphi + \theta + \mu + d), \lambda_5 = -(\delta + \omega + \mu) \text{ and } \lambda_6 = -\mu$$

Since all the eigenvalues have a negative real path, we conclude that  $R_0 < 1$  showing that the disease-free -equilibrium is locally asymptotically stable.

#### 2.3.2 Global stability analysis of the model

#### Theorem 3

The disease free-equilibrium of the system (3.1)- (3.6) is globally asymptotically stable whenever  $R_0 < 1$  but unstable if  $R_0 > 1$ .

#### Proof

By using the comparison theorem2 for global stability, we have that the comparison method is  $|F - V|$ . From equations 4.5 and 4.6 and  $\xi$ , representing the characteristic polynomial, we obtained the following,



$$|(F - V) - \xi| = \begin{vmatrix} -(\delta + \omega + \mu) - \xi & \frac{\rho\beta\pi}{(\lambda + \mu)} & 0 \\ \delta & -(\varphi + \theta + \mu + d) - \xi & \gamma \\ 0 & \varphi & (\alpha + \sigma + \gamma + \mu + d) - \xi \end{vmatrix} = 0$$

$$\frac{\gamma\varphi(\lambda + \mu)(\delta + \xi + \omega + \mu) + (d + \alpha + \gamma - \xi + \sigma + \mu)(-\delta\rho\beta\pi + (\lambda + \mu)(d + \theta + \xi + \varphi + \mu) + (\delta + \omega + \mu))}{\lambda + \mu} = 0 \tag{12}$$

From the above equation, since the transition rate from non-susceptible youths to susceptible youths represented by  $\lambda$ , is on the decrease as time increases, we conclude that  $R_0 < 1$  which proves the global asymptotic stability of the model.

### 3.0 Discussion

This research work is geared toward possible ways to curb the menace of gangs in society. Considering gangs as an infectious disease is a means of emphasizing how their impact is on the general society at large. The gang model formed considered various forms of its initiation, ways the government has taken to deal with the culprits and the means or modality to be taken to ensure that youths are being engaged continually in productive work, to reduce idleness that could lead to the desire to join gang. From the stability analysis, we found out that with the disease free-equilibrium, the local and global stability of the model is attained when the reproduction number  $R_0 < 1$  and the global stability analysis of the disease free-equilibrium  $E_0$  when  $R_0 < 1$ , assures us that the disease can be controlled if measures that can reduce the reproduction number below unity are implemented, which we were able to showcase from the numerical simulation of the general model and the individual subclasses of the population. In doing this, we used the parameters presented in Table 1 to simulate system (1)-(6) and to investigate the impact of the proposed treatment concept on the subclasses of the population. Most of the parameter values are raised from the literature sources. From the general graph, it is shown

that a decrease in the quest for gang membership applies as time progresses positively, showing that with time, society will be cleansed from this ravaging desire in the youths. Also, through the rehabilitation of deviants, we observed a good increase in the recovery class which is a plus on the economy of the society as reasonable manpower will be released in favour of the growth of the system. This shows that it is possible to crumble or even completely eradicate gang movement from all levels of operation if adequate efforts are made to keep the youths positively active with both technical and technological threads that will keep their minds on the need to make good impacts with what they are doing for relevance rather than seek it from the wrong approach. The individual graphs represent the progression each class of the population faces as time increases.

### 4.0 Conclusion

The contagious state of the gang lifestyle, plays a central role in allowing gangs to be treated as an infectious disease. We have developed a model with six variables for the spread of gang in a heterogeneous population and its control. Some variables and parameters have been introduced as a way to look into the behavioural activities of gangs and ways to help these youths gain back their confidence and reasons to live normally by bringing in the correctional facilities and rehabilitation homes/programs with the notion of building back these delinquent youths into the population. The mathematical analysis of the model presents the formulation of the reproduction number, and its sensitivity analysis conducted.

Fig 2: The general gang population graph against time  $t$ .



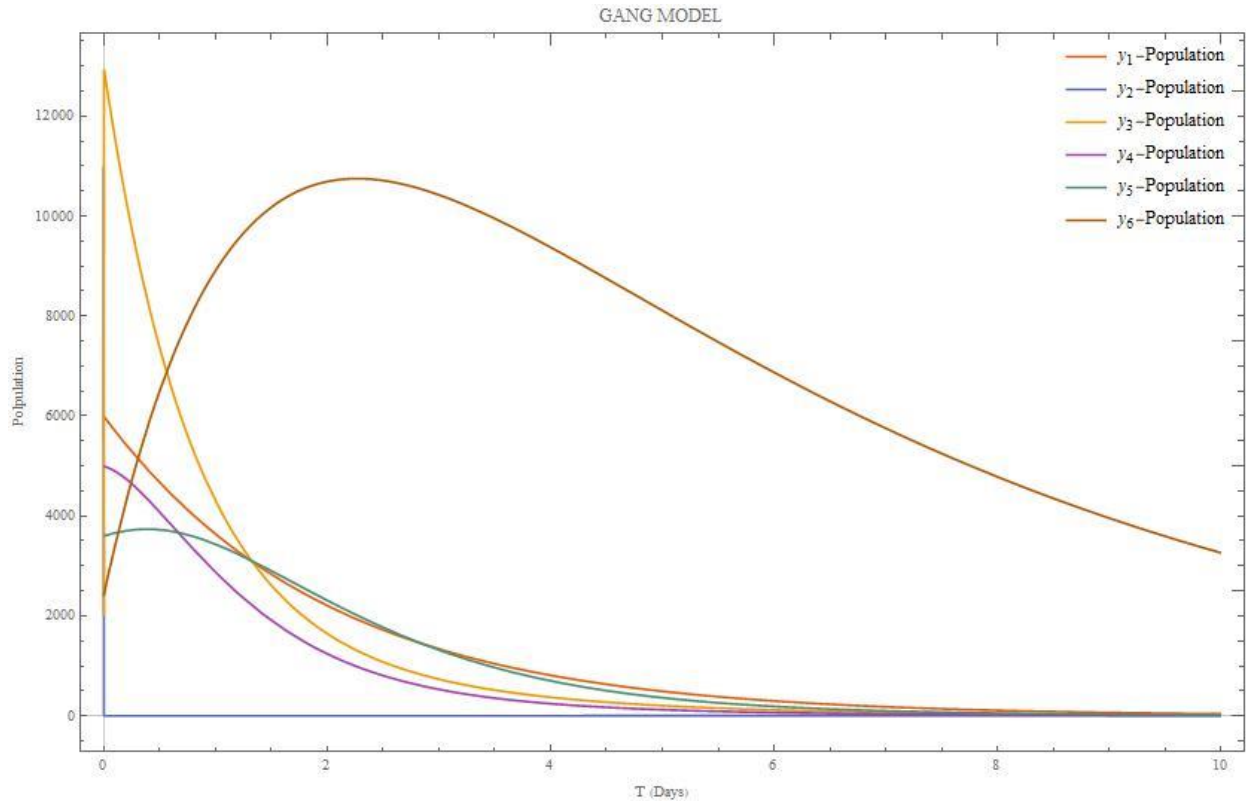


Fig 3: The plot of normal population against time.

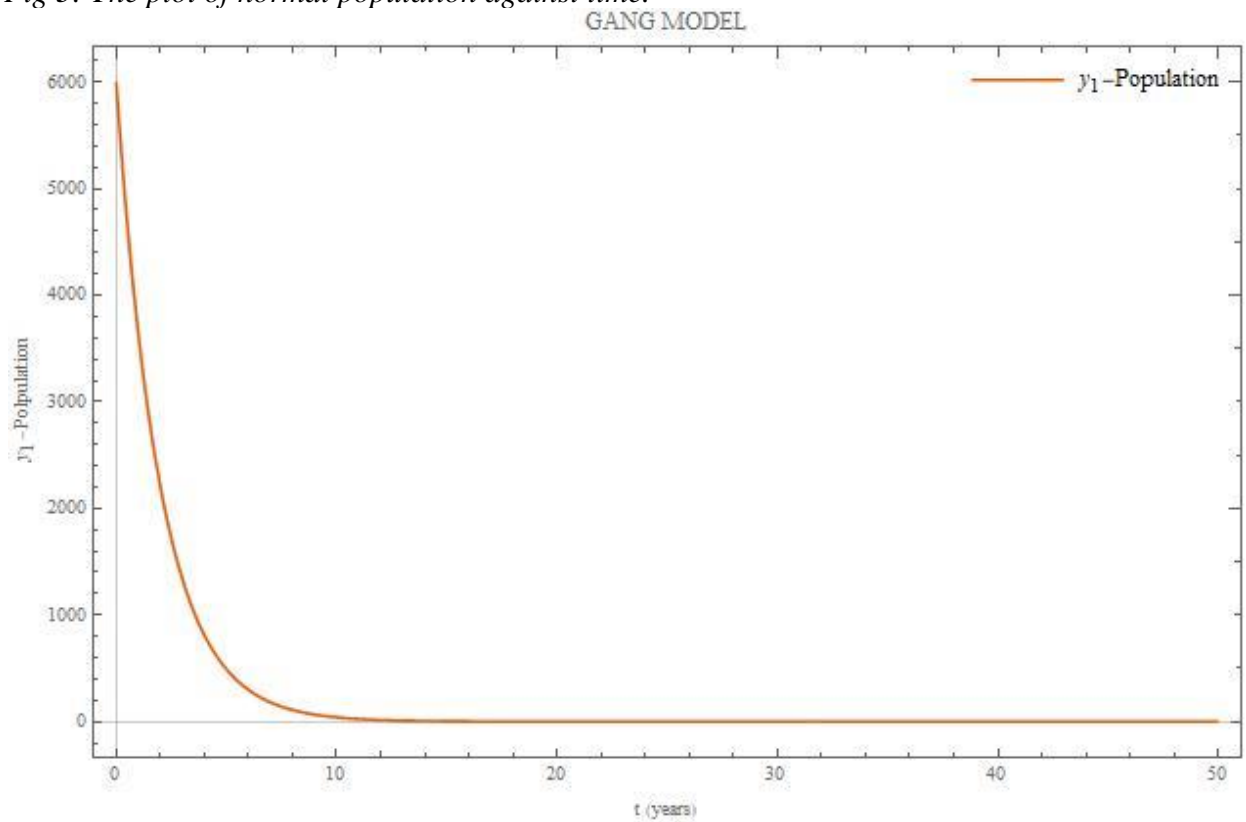


Fig 4; The plot of susceptible population against time.





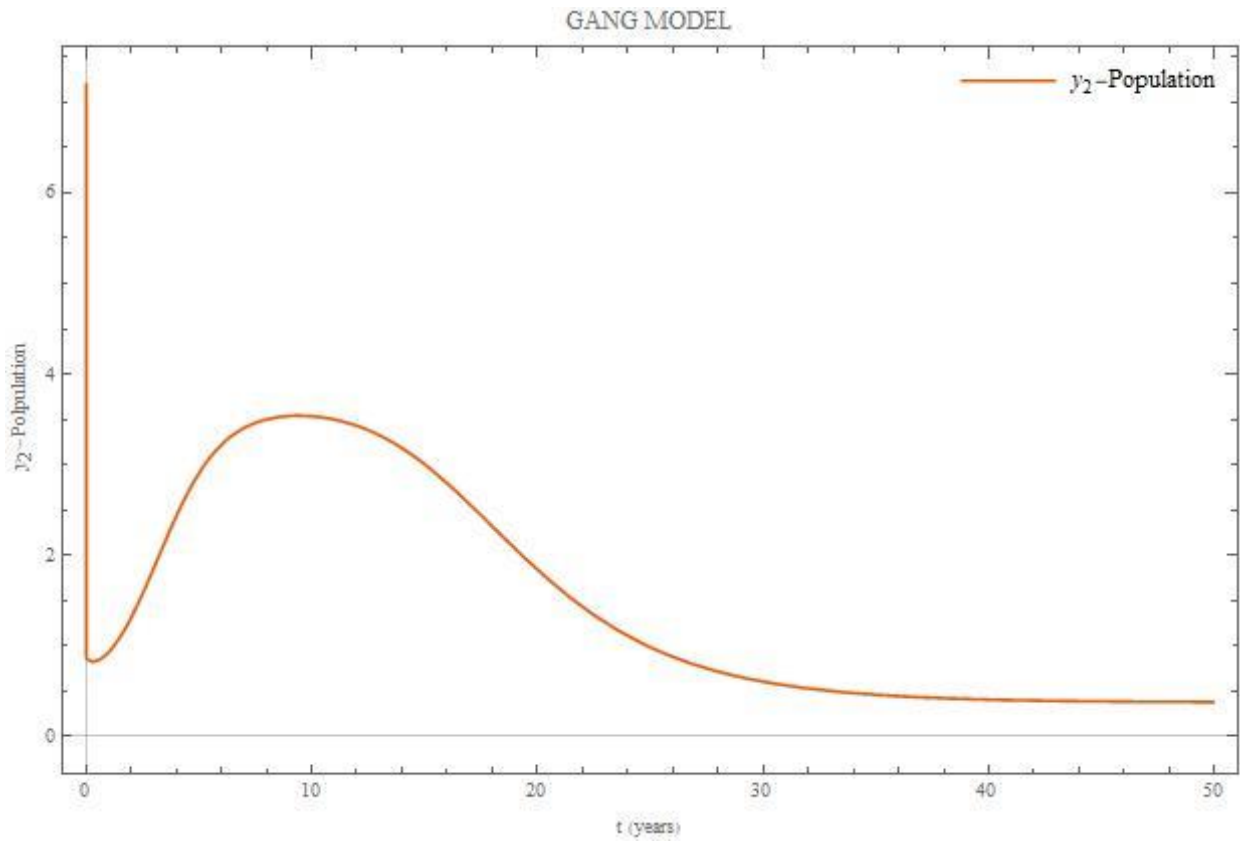


Fig 5: The plot of the exposed population against time.

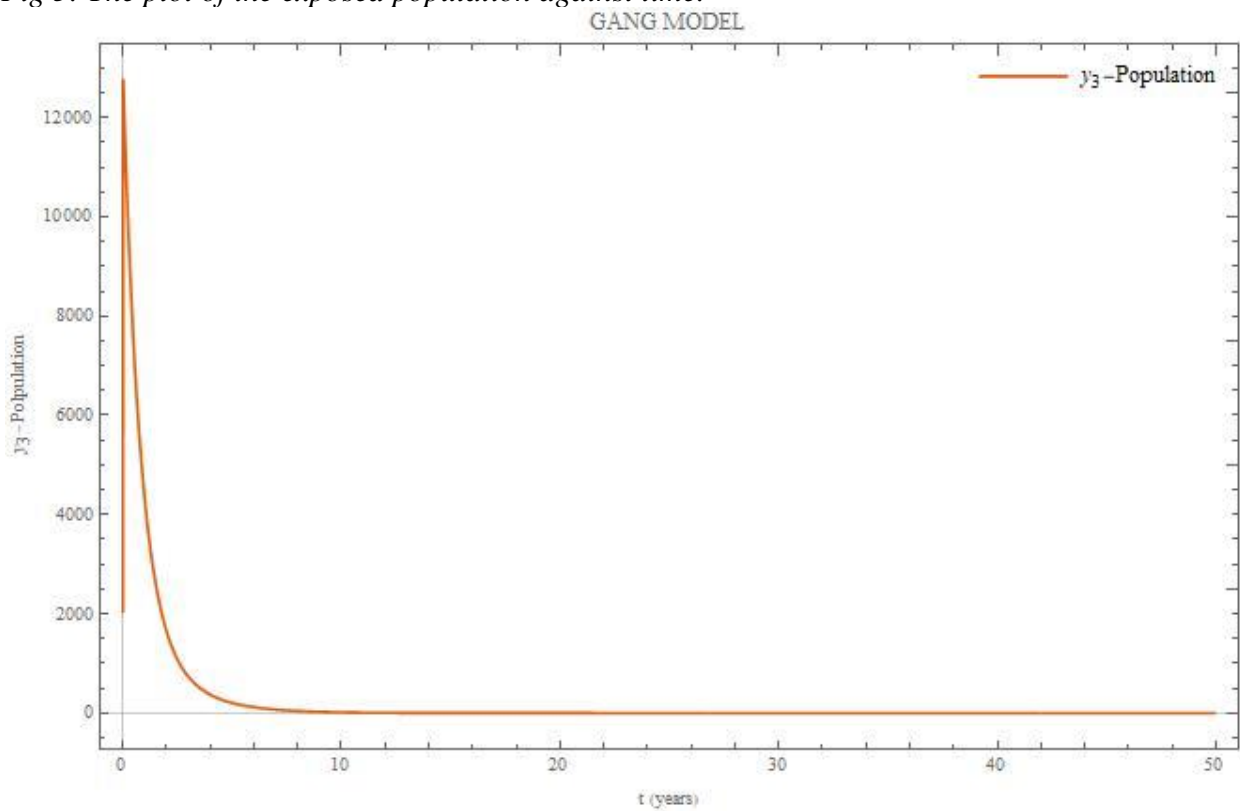


Fig 6: The plot of core gang members against time.

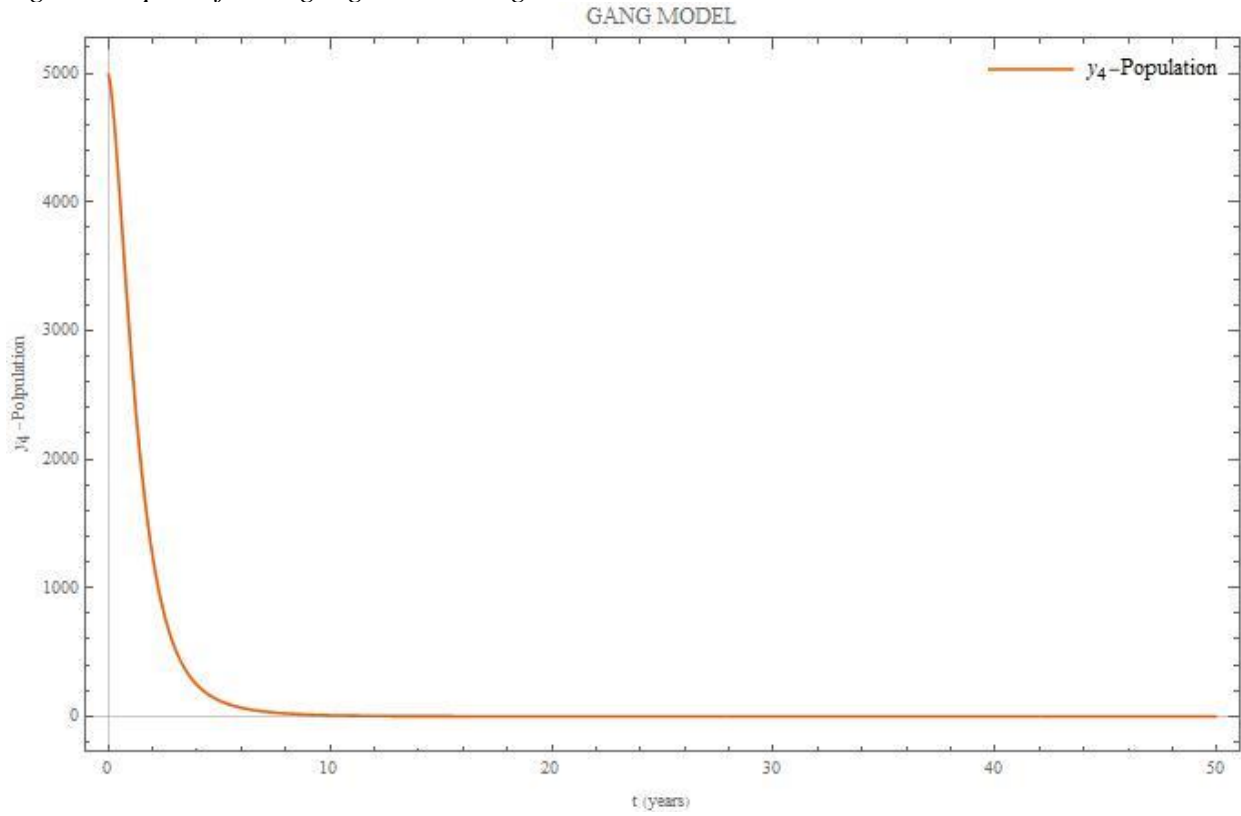


Fig 7: The graph of individuals in correction against time.

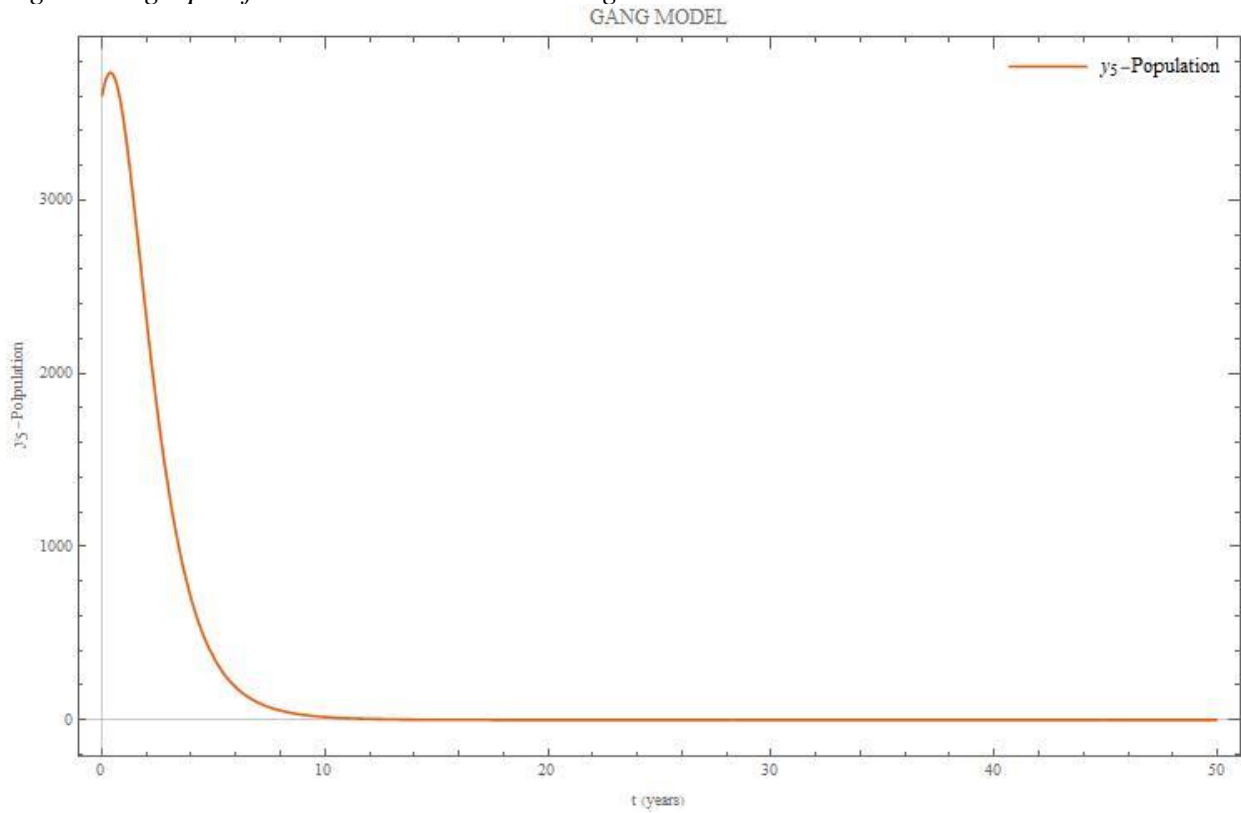
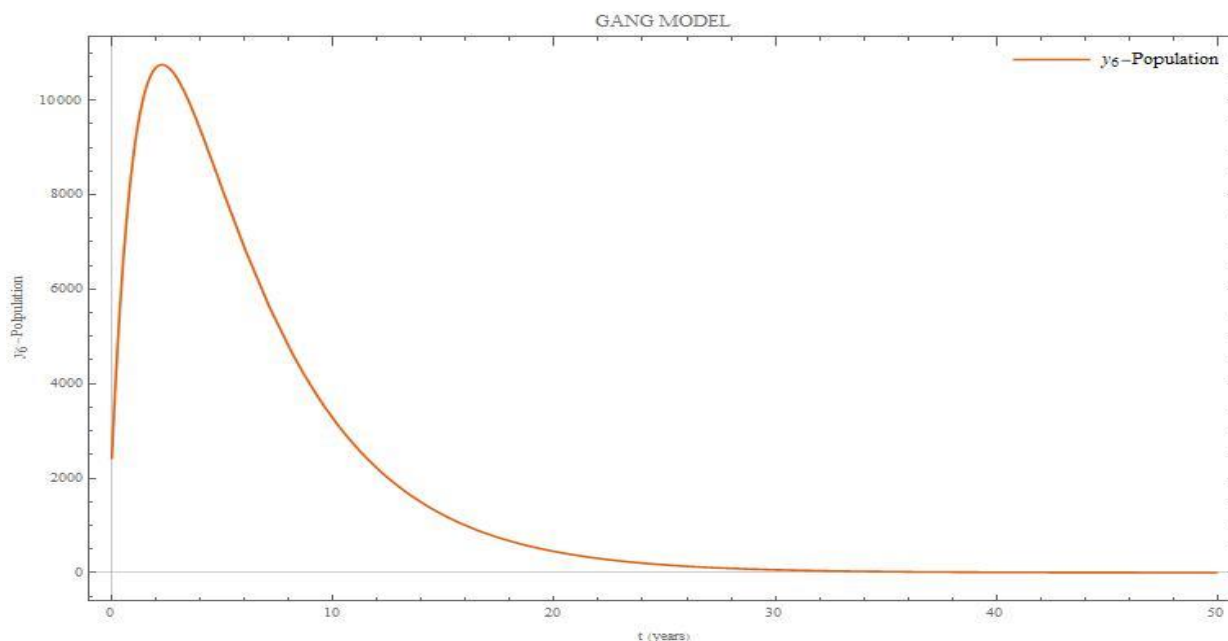


Fig 8: The plot of recovered individuals against time.





**Appendix A: Invariant Region**

With the assumption that the initial condition is nonnegative, that is  $P \geq 0, S \geq 0, E \geq 0, I \geq 0, D \geq 0$  and  $R \geq 0$ , we obtain the invariant region in which the model solution is bounded by considering the total human population.

$$N = P + S + E + I + D + R$$

Then differentiating  $N(t)$  both sides with respect to  $t$  leads to:

$$\frac{dN}{dt} = \frac{dP}{dt} + \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dD}{dt} + \frac{dR}{dt} \quad (13)$$

By combining (1.1) – (1.6), we get:

$$\frac{dN}{dt} = \pi - \mu N - (I + D)d$$

In the absence of mortality due to gang activities, (2.1) becomes

$$\frac{dN}{dt} \leq \pi - \mu N$$

Therefore,

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\pi}{\mu} \dots \quad (14)$$

Thus, the feasible solution set of the system equation of the model enters and remains in the entire region  $\Omega = \{(P, S, E, I, D, R) \in R_+^6: N \leq \frac{\pi}{\mu}\}$ .

Therefore, the basic model is well posed epidemiologically and mathematically, so sufficient to study the dynamics of the basic model in  $\Omega$ .

**Appendix B Positivity of the solutions**

With the initial condition, we show that the solution of the model is also positive.

**Theorem 1**

Let  $\Omega = \{(P, S, E, I, D, R) \in R_+^6: P \geq 0, S \geq 0, E \geq 0, I \geq 0, D \geq 0$  and  $R \geq 0\}$ , then the solutions of the model are positive at  $t \geq 0$ .

**Proof**

From the system of differential equation (1.1) to (1.6), considering (1.1), we have.

$$\frac{dP}{dt} = \beta\pi - (\lambda + \mu)P$$

This implies

$$\frac{dP}{dt} \geq -(\lambda + \mu)P$$

Upon integration and by solving using separation of variable method, we have:

$$\int \frac{dP}{P(t)} \geq - \int (\lambda + \mu) d(t)$$

$$\ln \frac{P(t)}{P(0)} \geq -(\lambda + \mu)t$$

$$\frac{P(t)}{P(0)} \geq e^{-(\lambda + \mu)t}$$

$$P(t) \geq P(0)e^{-(\lambda + \mu)t} \geq 0 \quad (15)$$

Considering (2)

$$\frac{dS}{dt} = (1 - \beta)\pi + \alpha D + \lambda P - (\rho I + \mu)S$$

$$\frac{dS}{dt} \geq -(\rho I + \mu)S$$

Upon integration and by solving using separation of variable method, we have:

$$\int \frac{dS}{S(t)} \geq - \int (\rho I + \mu) d(t)$$

$$\ln \frac{S(t)}{S(0)} \geq -(\rho I + \mu)t$$



$$\frac{S(t)}{S(0)} \geq e^{-(\rho I + \mu)t}$$

$$S(t) \geq S(0)e^{-(\rho I + \mu)t} \geq 0 \quad (16)$$

Similarly if we solve for (1.3) – (1.6), we conclude that the solutions of the model are positive always for  $t \geq 0$ , which completes the proof of the theorem.

### Appendix C: Existence and Uniqueness of the model solution

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