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Mathematical Propagation for the Treatment and Vaccination of a Generalized Well-Posed SEIR Infectious Model

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ABSTRACT

In this paper, we propagated a generalized theoretical executable investigation for an improved SEIR mathematical model for infectious diseases. The model was constructed to determine a solution for a system of ordinary differential equations described in a deterministic immune population and studied under designated bilinear control functions. Analytic predictions for the system well-posedness was quantitatively conducted using theory of ordinary differential equations in conjunction with Lipschitz condition. An expression is obtained for the state-space and numerical computations determined. Results show that with induced bilinear control functions, rapid rejuvenation of the recovered and the susceptible was tremendously achieved. Moreso, the model exhibited compatibility for varying infectious diseases, provided there exists coherency to designated control functions. Therefore, the application of an improved generalized SEIR model under bilinear control functions is priori innovative for the amelioration and treatment of infectious diseases when compared with results of existing SIR models.

Keywords: *Generalized-SEIR-model, system-well-posedness, bilinear-control-functions, Lipschitz-condition, existence-uniqueness, state-space.*

2010 Mathematics Subject Classification (MSC): *93A30, 93C15, 34H15, 65L20*

1. Introduction

Transmissible infectious diseases are becoming of significant global health concern as there have imposed significant devastations on varying organs of mankind. Though this present investigation does not capture a particular infectious disease, rather making attempt to present a simplified generalized infectious diseases model, we shall as well, focus on general reviews of infectious diseases and possible compactible mathematical models.

Historically, infectious diseases are categories of transmissible viruses, pathogens, bacteria, fungi and protozoa, most of which are considered as airborne diseases as well as vector carriers [1,2]. Commonest among other infectious diseases include but not limited to: influenza, West Nile, filariasis, malaria, chikungunya, dengue, mumps, whooping cough, measles, smallpox, chickenpox, SARs, COVID-19 and many more [3,4,5]. Of interest, is the fact that the invisibility and indistinguishable nature of most infectious disease transmissions have made these classes of diseases a serious threat to mankind and hence, a source of global concern [6,7]. The transmission of infectious diseases could be hypo-transmissibility (human-to-human) or hyper-transmission (environment-to-human) with air and sex as the commonest windows [8,9,10]. The control and treatment of infectious diseases have been among other methods, the use of vaccines and designated treatment and/or chemotherapies. Essentially, vaccines are regarded as pathogenic micro-organisms, which simulates the immune system, leading to the build-up of antibodies against foreign microbes [1,14].

That is, the application of vaccines enhances the immune system against specific microbes, thereby acting as a protective mechanism to the population against specific diseases.

Remarkably, the understanding of disease transmission, control and treatment as well as prevention has been among other methods, the use of mathematical modeling, which is considered instrumental in the studying of the dynamics of infectious diseases. An early simplified mathematical model has been the SIR (Susceptible-Infectious-Recovered) models and recently, enhanced with the incorporation of the Exposed class - the SEIR mathematical models. For instance, one of the earliest comprehensive models, proposed by Anderson and May in the 1980s, introduced a basic SIR model for studying of hepatitis B infection dynamics. The study demonstrated how vaccination could significantly reduce the prevalence of chronic HBV [15]. Basic differential equations were explored and stability analysis conducted. The result of which was innovative with incisive leeway to decision making. Furthermore, [5]. conducted stability analysis and optimal vaccination of infectious epidemic model, using the SIR mathematical model. The study investigated the model equilibria and established the existence of optimal control for the system with results, which was a significant to the understanding of the dynamics and treatment of infectious diseases. [16], provided a more improved SIR model with some realistic assumptions. That model was subjected to four classified conditions and tested for accuracy using Visual Basic (VB). Results of simulations showed that due to those conditions, infections declined rapidly, leading to high recovery rate when compared with other existing SIR models. Perturbingly, the emergence of COVID-19 further fronted the use of SIR mathematical models. For instance, in India, as a case study, SIR mathematical model was initiated in the investigation of the occurrence of COVID-19 [18]. In that study, the analysis of the model explored the Euler's method. The results of model simulations indicated that a systematic spread of the virus was eminent, leading to possible outbreak. Remarkably, the result suggested that the SIR model via Euler's method was an efficient tool for prediction of disease transmission and prevention.

As an improvement to the SIR models, the recent incorporation of the exposed subpopulation became eminent, leading to: Susceptible – Exposed – Infected – Recovered (SEIR) mathematical models. For instance, using the SEIR mathematical approach, a model that studied the effect of monolytic control function – vaccine use, on the dynamics of infectious diseases was formulated [1]. That study focuses on the impact of mono-vaccine on infectious epidemic with compactible analytic predictions conducted. The results obtained indicated that the application of vaccine as designated disease control, have the potency to inhibit disease outbreak, provided productivity ratio is less than unity.

Remarkably, from resourceful reviews of existing literatures, is the fact that studying of the spread of infectious diseases in time and space, using the SIR – SEIR models are geared towards gaining a better understanding of disease transmissions as well as to evolve predictions and possible evaluations of treatments and control strategies [7,13,17]. Therefore, this present research seeks to possibly institute a number of vital scientific remedies as against the study statement of problem by incorporating a more extended SEIR model from some existing models [1,5]. That is, we present a modified generalized SEIR model that redefined system incidence rate as a function of trilinear state space with the infusion of designated bilinear control functions – designated treatment and/or chemotherapy and vaccination occasioned by realistic assumptions. More importantly, the analysis of desired system is targeted to explore the interplay of bilinear control functions.

In a directional format, the entire investigation is composed of the introductory aspect and the incisive literature review on section 1. Section 2, focuses on the materials and methods constituted by the system statement of problem, formulation of desired model. The system mathematical propagation and analysis are viewed in section 3. In section4, we demonstrate some numerical computations with illustrative simulations. Analyses and discussion of derived results

in comparisons with compactible existing results is overcome in section 5. Finally, we deduce incisive summary and recommendations based on available findings in section 6. Explicitly therefore, the present investigation is geared towards giving an insight to the potencies of both modified SEIR model as a tool and the impact of bilinear controls on a generalized SEIR infectious model.

2. Materials and Methods

The materials and methods for this research is characterized by the system statement of problem and formulation of system model. The model cogently explored a set of Dimensional deterministic ordinary differential equations. Moreso, the materials are constituted by the interplay of 4 – sub-population with thematic investigation evolving round a combination of bi-linear control functions (designated treatment and/or chemotherapy and vaccine). System formulation and mathematical analysis deployed fundamental theory of ordinary differential equations in conjunction with Lipschitz criterion. The aspect of illustrative numerical simulations explicitly explores classical in-bult Runge-Kutta of order of precision 4 in a Mathcad surface.

2.1. Problem statement of the study

Clearly, aligning from existing scientific literatures on transmission of infectious diseases, its treatment and controls mechanisms, it's obvious that mathematical modeling have become a vital tool. The use of mathematical modeling of infectious diseases had its bases from the use of the SIR - SEIR models. For instance, adjudging from relatively compactible models to the intense of this present investigation, is the SEIR model developed with linear vaccination that studied the interplay of varying compartments of designated sub-population [1]. Remarkably, a critical review of this model revealed some incisive scientific lapses, which forms the nucleus of this present research. These includes:

i. That the aforementioned model was devoid of any treatment and/or chemotherapy.
ii. Incidence rate was only a function of two consequential interactive processes (only the infectives and recovered).
iii. The model never accounted for the mathematical well-posedness of derived model.

Therefore, incorporating these aforementioned lapses, the present investigation seeks to depict a modified generalized deterministic mathematical model that redefine the modified incidence rate as a function of the exposed compartment. Essentially, the present investigation is geared towards presenting an insight to a generalized modified SEIR infectious mathematical dynamics with the infusion of designated bilinear control functions – designated treatment and/or chemotherapy and vaccination. Moreso, the study seeks not only to importantly verify the well-posedness of the anticipated complex proposed model but also, investigate the positivity of the ascribed state-space of the model and the uniqueness of solution using Lipschitz condition.

Definition: (*Lipschitz condition*)

In particular, Lipschitz condition is defined as: let $x, x' \in \Re$ be real variables and let $f : [a,b] \to \Re$ be a function. Then, f is said to satisfy the Lipschitz condition if there is a constant M such that $|f(x) - f(x')| \le M|x - x'| \quad \forall$ $x, x' \in [a,b]$, where M is the Lipschitz constant.

2.2. Mathematical formulation of model equations

In this sub-section, attempting to overcome the existing study statement of problem, we align with study motiving model [1]. Remarkably, in that model, the population understudy was partitioned into: susceptible $S(t)$, the infectious $I(t)$ and the recovered $R(t)$. The epidemiological derived model is given by

$$\begin{cases} \dfrac{dS(t)}{dt} = vN - vS(t) - \dfrac{\beta I(t)S(t)}{N}, S(0) = S_0 \geq 0 \\ \dfrac{dI(t)}{dt} = \dfrac{\beta I(t)S(t)}{N} - (\gamma + v)I(t), I(0) = I_0 \geq 0 \\ \dfrac{dR(t)}{dt} = \gamma I(t) + vR(t), R(0) = R_0 \geq 0 \end{cases} . \quad (1)$$

The details of eqn. (1) can be accessed as reference above. Now, suppose we extend model (1) with the incorporation of modified exposed compartment to consider a SEIR mathematical deterministic dynamic infectious model, then we have subpopulations denoted by susceptible $S_p(t)$, the exposed $E_p(t)$ .the infectious $I_p(t)$ and the recovered $R_p(t)$. Since our population is a complete representation of a set of living organisms and posed to be a real-life epidemic outbreak, then the investigation of interplaying varying subpopulation can be subjected to some designated bilinear control functions in the form if compactible treatment and/or chemotherapy γ and inducible vaccination ρ_i, $i = 1,2$ for all $i > 0$ .Furthermore, a reliable infectious mathematical model is bounded and is sustained by clear and succinct assumptions. That is, the guiding assumptions of this study, which align with existing assumptions are as contained in assumption of the model.

Assumptions of the model

The following have been considered as the assumptions of the model:

i. Population interaction is homogeneous, i.e. $N(t) = S_p(t) + E_p(t) + I_p(t) + R_p(t) = 1$

ii. System birth rate must surpass natural clearance rate, i.e. $b_p > \mu$ for all $(b_p, \mu) \geq 0$

iii. Only the susceptible and infectious receive vaccination, i.e. $\rho_i \geq 0$ for all $i = 1,2$ such that $\rho_2 \square \rho_1$.

iv. Only the infectious are exposed to treatment and/or chemotherapy, i.e. $\gamma \geq 0$.

v. Only the infectious die due to infection, i.e. $\alpha \geq 0$.

vi. Infection could re-occur after prolong vaccination, if $\rho_i \leq 0$.

Therefore, by extending the study motivating model (1) and using aforementioned assumptions, the epidemiological SEIR model equations are derived as seen in the differential dynamics of model (2):

$$\begin{cases} \dfrac{dS_p(t)}{dt} = b_p + \omega R_p - \beta_i(\hat{N})S_p - \rho_1 S_p - \mu S_p \\ \dfrac{dE_p(t)}{dt} = \beta_i(\hat{N})S_p - \sigma E_p - \mu E_p \\ \dfrac{dI_p(t)}{dt} = \sigma E_p - (\gamma + \rho_2)I_p - (\mu + \alpha)I_p \\ \dfrac{dR_p(t)}{dt} = (\gamma + \rho_2)I_p + \rho_1 S_p - (\omega + \mu)R_p \end{cases} , \quad (2)$$

where

$$\beta_i(\hat{N}) = \frac{\sum_{i=1}^{3}\left(\beta_1 c_1 E_p + \beta_2 c_2 I_p + \beta_3 c_3 R_p\right)}{N} \quad (3)$$

with $N(t) = S_p(t) + E_p(t) + I_p(t) + R_p(t) = 1$ and $\hat{N}(t) = E_p(t) + I_p(t) + R_p(t)$. Notably, eqn. (2) holds, provided the initial conditions $S_p(t_0) > 0, E_p(t_0) > 0, I_p(t_0) > 0$ and $R_p(t_0) > 0$ for all $t \geq t_0$ is satisfied. Moreso, eqn. (3) is the model incidence rate also known as the force of infection or system mass action. That is, eqn. (3) is defined by a trilinear novel incidence coefficient i.e. $\frac{\beta_i c_i}{N}\left(E_p + I_p + R_p\right)$ for all $i = 1,2,3$.

Therefore, for a more explicit representation of model (2) in conjunction with outlined model assumptions, the schematic representation of derived model is as in fig. 1.

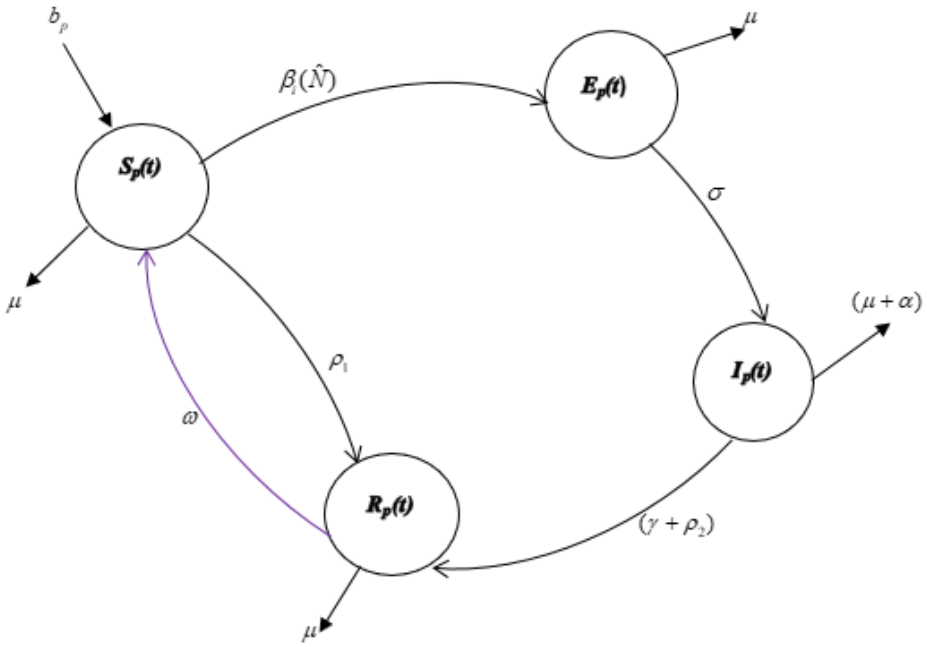


Fig. 1: Schematic representation of a SEIR model under bilinear control functions

A critical descriptive and review of model (2) alongside fig.1 is depicted by tables (1 & 2)

Table 1: Description of state components for model (2)

State space	Description of dependent variables
$S_p(t)$	Susceptible subpopulation not yet infected
$E_p(t)$	Exposed subpopulation at asymptomatic stage of infection
$I_p(t)$	Infectious subpopulation at symptomatic stage
$R_p(t)$	Recovered subpopulation due to bilinear control functions

Table 2: Description of constants and parameter for model (2)

Parameters & symbols	descriptions Parameters & constants
b_p	Natural birth rate, i.e. $b_p > 0$
μ	Natural death rate, i.e. $\mu \geq 0$
$\beta_i, _{i=1,2,3}$	Incidence rate (mass action)
ω	Proliferation of recovered population, i.e. $\omega \geq 0$
σ	Rate at which the exposed become infected, i.e. $\sigma \geq 0$
$\rho_{i,i=1,2}$	Rate of vaccination of $S_p(t)$ and $I_p(t)$
γ	Rate at which the infectious receive treatment
α	Clearance rate due to infection, i.e. $\alpha \geq 0$

Furthermore, the epidemiological description of model (2) as in fig. 1, are as follows: from the first equation of the model, describing the biological implication, we note that the differential behaviour of the susceptible increment is defined by incoming natural birth rate b_p, together with proliferating recovered population under declined immunity ω. Population mutation in this compartment is due to continuous interactions with the varying infectious subpopulations denoted by $-\beta_i(\hat{N})$; the rate of immunization of the susceptible $-\rho_1$ and declined due to natural clearance rate $-\mu$. Taking on the second equation of the model, which depicts the differential derivative of the exposed compartment, the sustaining population here, is the mass action defined by the incidence rate $\beta_i(\hat{N})$. This subpopulation gradually depleted as there become infectious at a rate $-\sigma$ as well as natural death rate $-\mu$.

On the other hand, the infectious compartment depicted by differential equation three, is sustained by the incoming infectious population σ and the gets depreciated by the rate at which the infectious receive treatment and getting vaccinated $-(\gamma+\rho_2)$ as well as death due to both natural effect and due to infection $-(\mu+\alpha)$. The final equation depicting the differential amplitude of the recovered subpopulation is confined by recovered due to bilinear controls $(\gamma+\rho_2)$ and rate at which the susceptible receive vaccine ρ_1. Here, population declines due to gradual deterioration of immunization. $-\omega$ and natural clearance rate $-\mu$.

Next, following the formulation of system model, it becomes pertinent that we verify the system compositions and its well-posedness. We achieve this by diffusing in our next section, the mathematical properties (system mathematical analysis).

3. Mathematical Analysis of Derived Model

In this section, we focus on the mathematical properties of derived system (2), which is characterized by the evaluations of the epidemiological well-posedness. That is, we show that all the state space exhibits non-negative solutions; determine that there exists boundedness of system solutions and finally, to show that the state space exists and is unique.

3.1. Positivity of system solutions

The concept of positivity of system composition enables the verification that solutions of the system remain non-negative for all $t \geq o$. This task is accomplished using the following theorem.

Theorem 1 (*Positivity od system solutions*)

Suppose $\{S_p(0), E_p(0), I_p(0), R_p(0),\} \in \Re_+^4$ denote initial conditions for system (2) for all $t \geq o$. , then there exists a solution set $\{S_p(t), E_p(t), I_p(t), R_p(t),\} \in \Re_+^4$ for system (2) and is positive for all $t \geq o$.

Proof

The prove of this theorem will access existing results for non-negativity of solutions [8,19,20,21]. In this case, model (2) is then confined in compact form as:

$$\Omega = \left\{(S_p, E_p, I_p, R_p) \in \Re_+^4 : N = S_p(t) + E_p(t) + I_p(t) + R_p(t) \leq \frac{b_p}{\mu}\right\}.$$

Suppose $\left(S_p(t) + E_p(t) + I_p(t) + R_p(t)\right)$ is any solution and having non-negative initial conditions, э $N = S_p(t) + E_p(t) + I_p(t) + R_p(t)$. Then, at zero mortality rate (i.e. $\alpha = 0$), the time derivative of $N(t)$ in the direction of system (2) is computed as:

$$\frac{dN}{dt} = b_p\mu\left(S_p + E_p + I_p + R_p\right) \le \frac{b_p}{\mu} - \mu N$$

for all $\alpha = 0$. Applying the theorem of differential equations and taking the integral factors and imposing initial conditions, the evaluation of the system yields the result

$$N(t) = N(0)e^{-\mu t} + \frac{b_p}{\mu}\left(1 - e^{-\mu t}\right) \ge 0$$

or

$$N(t) = \frac{b_p}{\mu} + \left(N(0) - \frac{b_p}{\mu}\right)e^{-\mu t} \ge 0\,. \tag{4}$$

Taking the limit of eqn. (4) as $t \to \infty$, we obtain

$$\lim_{t\to\infty} N(t) = \frac{b_p}{\mu} \ge 0\,. \tag{5}$$

From eqn. (5), it becomes clear that the solutions of all the state components is bounded in $\Re_+^4$ and is confined in the region Ω, for all $t \in [0,\infty)$. Hence, the required result. □

3.2. Invariant Region of System Solutions

Having shown that non-negativity of solutions exists for system (2), then we further show that this non-negative solution is bounded within the invariant region for all $t \ge 0$. This boundedness of solution is satisfied by the following theorem.

Theorem 2 (*Boundedness of system solutions*)

Let the system be bounded in the closed set $\Re_d = \left\{(S_p, E_p, I_p, R_p) \in \Re_+^4 : N \le \frac{b_p}{\mu}\right\}$. Then, all solutions of this closed set $\Re_d$ is bounded, non-negatively invariant and attracting absolutely for system (2).

Proof

Invoking existing results for boundedness for solutions (for example, see: [2,19,23]. Then, form the differential sum of system (2), we have

$$\frac{dN}{dt} = \frac{dS_p}{dt} + \frac{dE_p}{dt} + \frac{dI_p}{dt}\frac{dR_p}{dt}$$

$$= b_p - \mu\left(S_p + E_p + I_p + R_p\right) - \alpha I_p$$

This implies that

$$\frac{dN}{dt} = b_p - \mu N - \alpha I_p\,, \tag{6}$$

where $N(t) = S_p + E_p + I_p + R_p$. Now, in the absence of mortality rate due to infection, then the population understudy is absolutely devoid of infection i.e. $\alpha = 0$. In this case, eqn. (6) becomes

$$\frac{dN}{dt} = b_p - \mu N\,.$$

Applying separation of variables, we have

$$\frac{dN}{dt} + \mu N \le b_p\,.$$

By integrating factor i.e. $IF = e^{\mu t\int dt} = e^{\mu t}$, we obtained

$$e^{\mu t}\frac{dN}{dt}+e^{\mu t}\mu N \le b_p e^{\mu t}$$

or

$$\frac{d}{dt}\left[\mu N e^{\mu t}\right] \le b_p e^{\mu t}.$$

Further integration gives

$$Ne^{\mu t} \le \frac{b_p}{\mu}e^{\mu t}+C,$$

where C is the constant of integration. If we now simplify the above, we get

$$N(t) \le \frac{b_p}{\mu}+Ce^{-\mu t}. \tag{7}$$

Introducing the initial condition $t=0$ to eqn. (7) and solving for C, we have

$$N(0) \le \frac{b_p}{\mu}+C$$

or

$$N(0)-\frac{b_p}{\mu} \le C. \tag{8}$$

Substituting eqn. (8) into eqn. (7), yields

$$N(t) \le \frac{b_p}{\mu}+\left[N(0)-\frac{b_p}{\mu}\right]e^{-\mu t}$$

or

$$N(t) \le N(0)e^{-\mu t}+\frac{b_p}{\mu}\left[1-e^{-\mu t}\right], \tag{9}$$

where $N(0)$ is the initial population at time $t=t_0=0$. This further yield $N(t) \le N(0)$ as $t \to 0$ and $N(t) \le \frac{b_p}{\mu}$ as $t \to \infty$.

Then, by Birkhof and Rota's theorem for differential inequality for all $t \to \infty$, we arrive at $0 \le N(t) \le \frac{b_p}{\mu}$ for all $t \ge 0$, [24]. Now, by well-posedness of system (2), it implies that

$$\frac{dS_p}{dt}=\frac{dE_p}{dt}=\frac{dI_p}{dt}=\frac{dR_p}{dt}=0.$$

This implies that

$$\frac{dN}{dt}=0.$$

Integrating both sides, yields

$$N=C,$$

where C is a constant. But we know that sum of population understudy is unity i.e.

$$N=S_p+E_p+I_p+R_p=1.$$

Then, it is deduced that

$$N=C=1.$$

This surmount to the fact that population is constant, non-negative and equal unity. That is, all feasible solutions of system (2) are constant in the region $\Re_d$ i.e.

$$\Re_d = \left\{(S_p, E_p, I_p, R_p) \in \Re_+^4 : S_p + E_p + I_p + R_p = 1\right\}.$$

Then, we ascertain that the region is bounded, non-negative and attracting. Hence, derived model is mathematically well-posed in the region $\Re_d$ and thus, is epidemiologically viable in $\Re_d$. This completes the proof. □

3.3. Existence and uniqueness of system solution

Here, the investigation shall have explored the concept of Lipschitz condition as a vital tool for the determination of the existence and uniqueness of the system solution.

By definition of Lipschitz condition, let $g:[x,y]\to\Re$ be a function, then g is said to satisfies Lipschitz condition, if there is a Lipchitz constant G such that $|g(s)-g(s')|\le G|s-s'|$ for all $s,s'\in[x,y]$, where G is the Lipschitz constant [19,26]. Now, let the complex set be defined by $\varphi:\Re\to\Re_+^4$ such that

$$t\mapsto\left(S_p(t)+E_p(t)+I_p(t)+R_p(t)\right) \text{ and } Q:\Re\to\Re_+^4$$

such that

$$\varphi(t)\Rightarrow Q(\varphi(t))=\left(\left(S_p^*(t)+E_p^*(t)+I_p^*(t)+R_p^*(t)\right)\right).$$

Then,

$$\varphi(t)=Q\left(\varphi(t),\varphi(0)\right)=\varphi_0.$$

Exploring the above definition, the following theorem holds for existence and uniqueness of system solution.

Theorem 3 (*Existence and Uniqueness*)

Let system (2) be a typical function said to be continuous and satisfies definition 1. Then, the existence and uniqueness of solution for system (2) holds, provided there exists a Lipschitz condition.

Proof

We invoke established results for existence and uniqueness of solution [8,25,26], Then, we investigate system (2) starting with the first equation and by inductive argument, same holds for others. That is, let

$$H(t,S_p)=\frac{dH}{dt}=b_p+\omega R_p-\beta_i(\hat{N})S_p-\rho_1 S_p-\mu S_p. \tag{10}$$

Applying eqn. (3) for $\beta_i(\hat{N})$ and taking the partial derivative of eqn. (10), we have

$$\frac{\partial H(t,S_p)}{\partial S_p}=\left[\sum_{i=1}^{3}\beta_i(\hat{N})c_i\right]-(\rho_1+\mu),\ \forall\ i=1,2,3. \tag{11}$$

Eqn. (11) shows that $H(t,S_p)$ and its partial derivative are defined and continuous for all (t,S_p). Then, by inductive reasoning, the right-hand side of the remaining equations of system (2) and their corresponding partial derivatives satisfy the existing conditions. That is, by existence and uniqueness theorem, there exists a set of unique solution existence and uniqueness theorem $\left\{S_p(t)+E_p(t)+I_p(t)+R_p(t)\right\}$ in some open interval with Centre at t_0. Next, we then show that this set of solution satisfies the Lipschitz condition. Now, from eqn. (10), it can be seen that

$$\left|H(t,S_{p(1)})-H(t,S_{p(2)})\right|=\begin{vmatrix} b_p+\omega R_p-\sum_{i=1}^{3}\beta_i(\hat{N})c_i S_{p(1)}-\rho_1 S_{p(1)}-\mu S_{p(1)} \\ -b_p+\omega R_p-\sum_{i=1}^{3}\beta_i(\hat{N})c_i S_{p(2)}-\rho_1 S_{p(2)}-\mu S_{p(2)} \end{vmatrix}$$

$$=\left|2\omega R_p-\left(\sum_{i=1}^{3}\beta_i(\hat{N})c_i+\rho_1+\mu\right)\left(S_{p(1)}-S_{p(2)}\right)\right|$$

$$\leq \left[2\omega R_p - \left(\sum_{i=1}^{3} \beta_i(\hat{N}) c_i + \rho_1 + \mu \right) \right] \left| \left(S_{p(1)} - S_{p(2)} \right) \right|.$$

This implies that

$$\left| H(t, S_{p(1)}) - H(t, S_{p(2)}) \right| \leq M \left| S_{p(1)} - S_{p(2)} \right|, \tag{12}$$

where $M = 2\omega R_p - \left(\sum_{i=1}^{3} \beta_i(\hat{N}) c_i + \rho_1 + \mu \right)$ is a Lipschitz constant. Then, by inductive reason, it shows that the remaining state-space satisfies the Lipschitz condition. Therefore, it is suffix to say that there exists unique solution set $\{S_p(t), E_p(t), I_p(t), R_p(t)\}$ for the system (2) for all $t > 0$. This completes the proof. □

Next, we focus next to some illustrative numerical computations as to ascertain the numerical viability of our analytic predictions.

4. Numerical Computations

In this section, we attempt to exhaust the insight to the study intense by conducting some numerical computations of the system analytic predictions as in section 3 with the consideration of its application to real-life situations. Here, we adopted certified empirical data to illustrate two sets of epidemiological examples i.e. case of off-treatment scenario and at onset-treatment scenario.

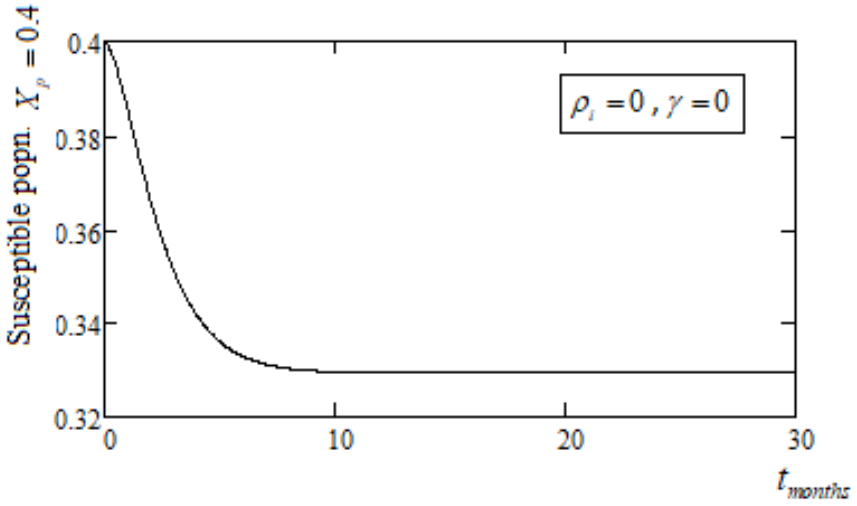
The entire simulations are accomplished using in-built Runge-Kutter of order of precision 4 in a Mathcad software. Furthermore, by extending tables 1 & 2, a corresponding compact form along with empirical data is obtained as in table 3:

Table 3: Value specifications for state-space and parameters variables for model (2)

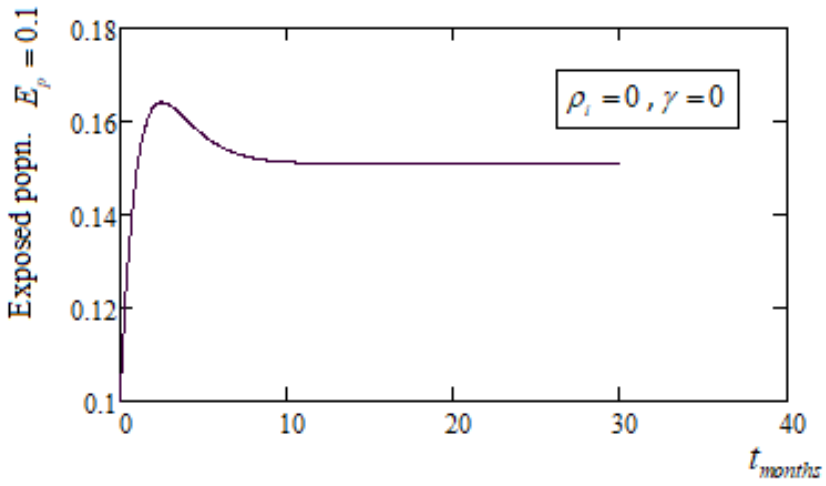
State space			Parameter variables		
Symbols	Values	Source	Symbols	Values	Source
X_p	0.4	[1, 8] (Alimi & Ayoade, 2023; Bassey & Igwe, 2022)	b_p	0.247	[19] (Alimi & Ayoade, 2023)
E_p	0.1		μ	0.31	
I_p	0.1		$\beta_{i=1,2,3}$	0.5;0.1;0.5	[26] (Bassey *et al.* 2023)
R_p	0.1		ω	0.5	[29] (Ayoade *et al.* 2019)
			σ	0,35	[1] (Alimi & Ayoade, 2023)
			$\rho_{i=1,2}$	0.4; 0.45	Estimated
			γ	0.4	Assumed
			α	0.4	[1] (Alimi & Ayoade, 2023)
			$c_{i=1,2,3}$	0.5; 0.5; 0.5	[7] (Bassey & Lebedev, 2016)

4.1. Numerical simulation under off-treatment $(\rho_i, \gamma) = 0$

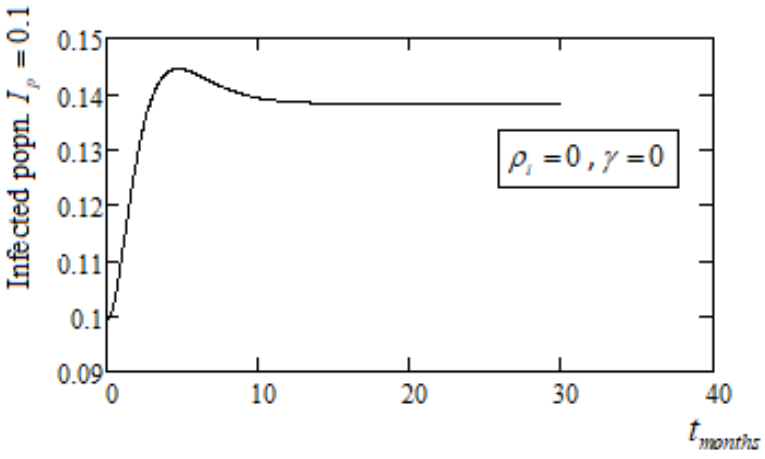
Using table 3 at $(\rho_i, \gamma) = 0$, we simulate system (2) at off-treatment scenario. For instance, fig. 2(a-d) depicts the required simulations with program algorithm denoted by **appendix A**.



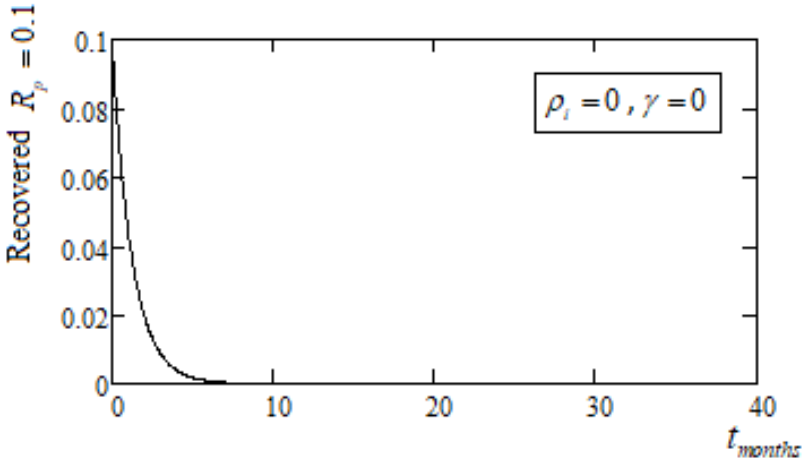
a) Susceptible popn. under off-treatment, $b_p = 0.247$



b) Exposed popn. under off-treatment, $\beta_1 = 0.5 , c_1 = 0.5$



c) Infected popn. under off-treatment, $\beta_2 = 0.1 , c_2 = 0.5$



d) Recovered under off-treatment, $\beta_3 = 0.5 , c_3 = 0.5$

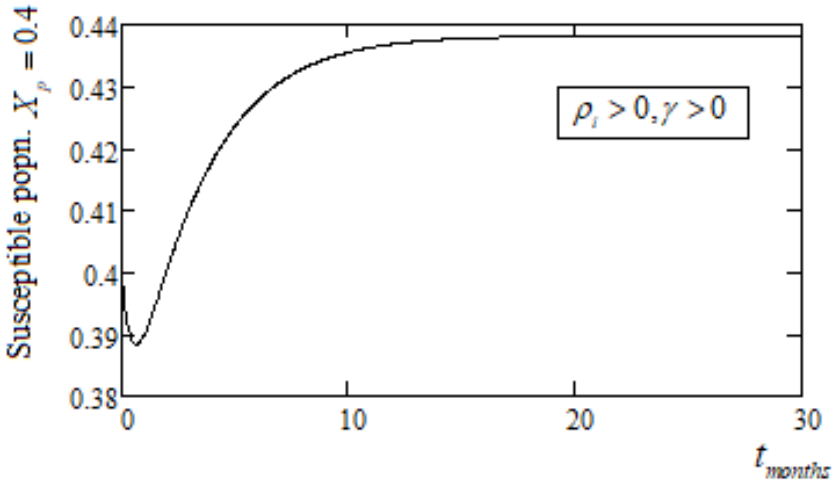
Fig. 2 (a-d): SEIR infectious dynamic model under off-treatment scenario

Fig. 2(a-d) depicts simulation of basic model (2) in off-treatment scenario. For instance, form fig. 2(a), we observed that the susceptible under off-treatment scenario exhibited rapid concave –like declination at $t_f \leq 8$ months with $0.329 \leq S_p(t) \leq 0.4$ and attained steady declined stability at $10 \leq t_f \leq 30$ months. For fig. 2(b), where the exposed subpopulation experienced off-treatment environment, the compartment exhibited rapid asymptomatic symptoms of infection at $t_f \leq 3$ months with $0.1 \leq E_p(t) \leq 0.164$ and the assumed steady state of $E_p(t) \leq 0.57$ for all $10 \leq t_f \leq 30$ months.

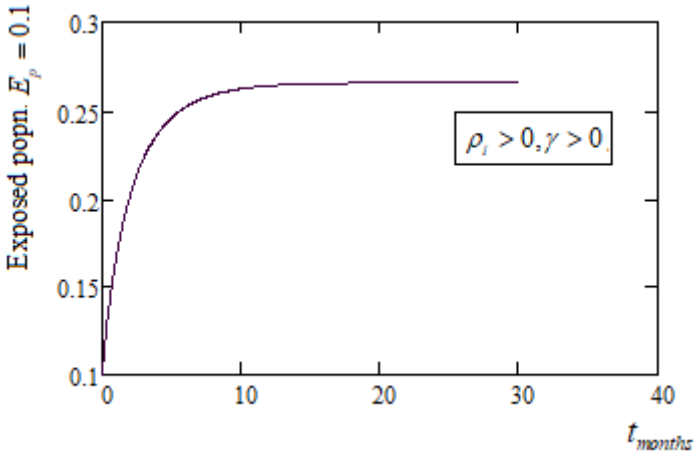
Furthermore, at off-treatment condition, fig. 2(c), which represent the infectious compartment exhibits similar epidemiological behaviour as in fig.2(b). Here, inclined infection rate stood at $0.1 \leq I_p(t) \leq 0.144$ and remain steady for all $10 \leq t_f \leq 30$ months. Under no control functions, the recovery compartment as depicted by fig. 2(d), shows rapid population extinction due to lack of any control functions i.e. $2.797 \times 10^{-12} \leq R_p(t) \leq 0.1$ at the earliest interval of $t_f \leq 8$.

4.2. Numerical simulation under onset-treatment $(\rho_i, \gamma) > 0$

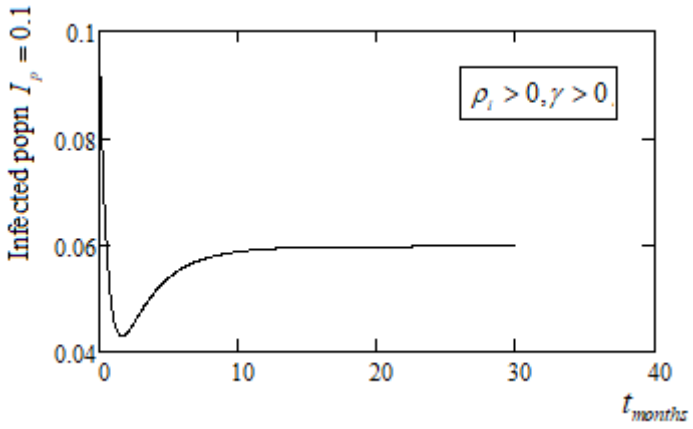
With the inducement of designated bilinear control functions in the form of chemotherapy and vaccine, we illustrate the input of treatment on system (2) as depicted in fig. 3(a-d). we denote the program algorithm by **appendix B.**



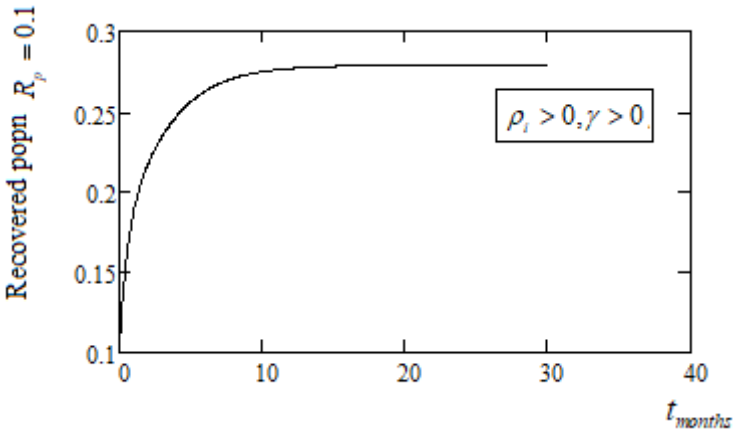
a) Susceptible popn. under onset-treatment, $b_p = 0.347$



b) Exposed popn. under off-treatment, $\beta_1 = 0.5$, $c_1 = 0.5$



c) Infected popn. under off-treatment, $\beta_2 = 0.1$, $c_2 = 0.5$



d) Recovered under off-treatment, $\beta_3 = 0.5$, $c_3 = 0.5$

Fig. 3 (a-d): SEIR infectious dynamic model under off-treatment scenario

Here, fig. 3(a-d) depicts system simulations under onset control functions i.e. introduction of bilinear controls if the form of chemotherapy and designated vaccine. For instance, fig. 3(a) which represent the susceptible under onset-treatment exhibited rapid convex-like curve with $0.389 \le S_p(t) \le 0.438$ for all months. In fig. 3(b), which depicts the exposed subpopulation, we see the compartment exhibiting some slight inclination of asymptomatic infection stage with $0.1 \le E_p(t) \le 0.266$ for all $10 \le t_f \le 30$ months.

The infectious compartment under bilinear control functions as in fig.3(c), clearly vindicated the inducement of control functions as we observed rapid declined viral load in the earliest interval of $t_f \le 3$ months with $0.043 I_p(t) \le 0.1$. Infection is then seen to rise slightly to $I_p(t) \le 0.06$ at $10 \le t_f \le 30$ months due to consistent exposure. Finally, from fig. 3(d), where recovery due to presence of control functions, we observed rapid inclined parabolic-like smooth curve at $t_f \le 10$ months with $0.1 \le R_p(t) \le 0.279$ and then sustained steady state at that region for all $10 \le t_f \le 30$ months.

5. Discussion of Results

So far in this research, we have formulated as an improvement of existing result [1], a simplified generalized SEIR mathematical model that seek to addressed the insight to the mathematical properties of a well-posed infectious model as well as determined the consequential impact of the application of induced bilinear control functions (in the form of treatment and/or chemotherapy and vaccine). The materials and methods explored a set of 4-Dimensional mathematical subpopulations investigated using bilinear control functions. Analytic predictions of model well-posedness was conducted using fundamental theory of differential equations in conjunction with Lipschitz condition.

Following derived model, the study was numerically computed in two illustrative examples, using in-built Gunge-Kutter of order of precision 4 in a Mathcad surface. Firstly, the case of off-treatment scenario was considered, followed by the application of onset-treatment – the assumed designated bilinear control functions. Remarkably, under off-treatment it was observed that the susceptible exhibited rapid population declined due to lack of any control functions as in fig. 2(a) with fig. 2(d) characterized by population extinction within months intervals of $t_f \le 8$. Both figures were in consonant with exiting results under similar conditions [1,7]. Furthermore, the consequential high clearance rate of the susceptible is a clear vindication of the rapid spread of the infection for both the exposed and infected compartments, see figs 2(b & c). A similar case study can be found in the investigation of HIV transmission dynamics [22].

Resourcefully, following the introduction of designated control functions, we saw an enhanced rejuvenated susceptible (fig. 3(a)) as well as rate of rapid recovered compartment – fig. 3(d). Moreso, unlike in the absent of control functions, where the exposed and infected compartments exhibited rapidly inclined infection rates on a contact average rate of 0.5 per day, at the onset-treatment system, we observed tremendous infection declination (see figs 3(b & c)). Clinically, these results are in affirmation of existing results where control function were cogently administered [23,26].

6. Conclusion and Recommendations

In this investigation, a 4 – Dimensional deterministic SEIR mathematical model have been formulated. The entire research has been conceived to addressed the challenging needs for a simplified generalized SEIR mathematical model that account for the treatment and control of infectious diseases dynamics. The model seeks and presented an insight to a simplified generalized mathematical model for the smooth understanding of the treated dynamics of infectious diseases epidemics. The investigation was conducted in the presence of bilinear control functions considered as designated treatment and/or chemotherapy with induced vaccine.

As a leverage, a control model of off-treatment scenario was as well conducted together with the onset-treatment scenario. Results indicated that at off-treatment scenario, subpopulations at varying compartments exhibited near population extinction. On the contrary, with the introduction of bilinear control functions, both the susceptible and recovered compartments exhibited tremendous population rejuvenations. These was evidence by the rapid contraction of the infectious compartments, a situation, which can be attributed to inducement of control functions. Moreso, the exposed compartment served as an indicator to the dynamics of infection proportion in view of the off-and-onset treatment analysis. The study therefore, recommends the SEIR modified generalized mathematical model for routine

intense application to real-life control measures for infectious diseases, provided there exits coherent adherence to appropriate medical specification.

Declaration of competing interest

The author otherwise known as the corresponding author on behalf of the authors declares that there exists no conflict of interest in this manuscript and that the entire content remains the original recipes of the authors with no part/whole submitted for consideration to any journal.

Credit authorship contribution statement

The authors' contributions to this manuscript were thus - **Bassey Echeng Bassey:** conceptualization, methodology, data collections, writing – original draft, writing – reviews and editing, algorithm and software programming, analysis and writing of final version. **Iwge O. Ewona:** Methodology, supervision, formal analysis, editing and valuations. Adagba O. Henry: Overall review in-depth analysis and partial funding.

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Appendices

Appendix A: **Program for Untreated basic SEIR model**

ORIGIN := 1 **system parameters**

$H := (0.4 \;\; 0.1 \;\; 0.1 \;\; 0.1)^T$

$F(t, H) :=$
$\beta i \leftarrow \beta 1 \cdot c1 + \beta 2 \cdot c2 + \beta 3 \cdot c3$
$F_1 \leftarrow bp + \omega \cdot H_4 - (\beta i \cdot H_1) - \rho 1 \cdot H_1 - \mu \cdot H_1$
$F_2 \leftarrow (\beta i \cdot H_1) - (\sigma \cdot H_2) - \mu \cdot H_2$
$F_3 \leftarrow (\sigma \cdot H_2) - (\gamma + \rho 2) \cdot H_3 - (\mu + \alpha) \cdot H_3$
$F_4 \leftarrow (\gamma + \rho 2) \cdot H_3 + \rho 1 \cdot H_1 - (\omega + \mu) \cdot H_4$
F

Result

$J := rkfixed(H, 0, T, n, F) =$

	1	2	3	4	5
1	0	0.4	0.1	0.1	0.1
2	0.03	0.4	0.102	0.1	0.098
3	0.06	0.4	0.105	0.1	0.095
4	0.09	0.4	0.107	0.1	0.093
5	0.12	0.399	0.109	0.1	0.091
6	0.15	0.399	0.111	0.1	0.089
7	0.18	0.399	0.113	0.1	0.086
8	0.21	0.399	0.115	0.1	0.084
9	0.24	0.398	0.117	0.1	0.082
10	0.27	0.398	0.119	0.1	0.08
11	0.3	0.398	0.121	0.1	0.078
12	0.33	0.397	0.122	0.101	0.077
13	0.36	0.397	0.124	0.101	0.075
14	0.39	0.396	0.126	0.101	0.073
15	0.42	0.396	0.127	0.101	0.071
16	0.45	0.396	0.129	0.102	...

Appendix B: **Program for Treated basic SEIR model**

ORIGIN := 1 **system parameters**

$H := (0.4 \;\; 0.1 \;\; 0.1 \;\; 0.1)^T$

$F(t, H) :=$
$\beta i \leftarrow \beta 1 \cdot c1 + \beta 2 \cdot c2 + \beta 3 \cdot c3$
$F_1 \leftarrow bp + \omega \cdot H_4 - (\beta i \cdot H_1) - \rho 1 \cdot H_1 - \mu \cdot H_1$
$F_2 \leftarrow (\beta i \cdot H_1) - (\sigma \cdot H_2) - \mu \cdot H_2$
$F_3 \leftarrow (\sigma \cdot H_2) - (\gamma + \rho 2) \cdot H_3 - (\mu + \alpha) \cdot H_3$
$F_4 \leftarrow (\gamma + \rho 2) \cdot H_3 + \rho 1 \cdot H_1 - (\omega + \mu) \cdot H_4$
F

Result

$J := rkfixed(H, 0, T, n, F) =$

	1	2	3	4	5
1	0	0.4	0.1	0.1	0.1
2	0.03	0.399	0.103	0.096	0.105
3	0.06	0.397	0.105	0.093	0.109
4	0.09	0.396	0.108	0.09	0.114
5	0.12	0.395	0.111	0.087	0.118
6	0.15	0.394	0.113	0.084	0.122
7	0.18	0.393	0.116	0.082	0.126
8	0.21	0.393	0.118	0.079	0.129
9	0.24	0.392	0.12	0.077	0.133
10	0.27	0.391	0.123	0.074	0.136
11	0.3	0.391	0.125	0.072	0.139
12	0.33	0.39	0.127	0.07	0.143
13	0.36	0.39	0.129	0.068	0.145
14	0.39	0.39	0.131	0.067	0.148
15	0.42	0.389	0.133	0.065	0.151
16	0.45	0.389	0.135	0.063	...

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