Mathematical Modeling And Probability Distribution Function Analyses of Quarantine Control Strategies For Covid-19

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ABSTRACT

Currently there is no approved and recognized vaccine for the treatment of corona virus disease (COVID-19). Thus this research work investigates the development of quarantine control strategies for COVID-19 by analysis from mathematical modeling and probability distribution function. The equilibrium states and their stabilities were obtained and analyzed by the use of first order ordinary differential equations for the six human population, which is divide into six mutually exclusive compartment, namely: Susceptible Human (S_H), Susceptible Vector (S_V), Infected Human (I_H), Infected Vector (I_V), Quarantine Human (Q_H) and Recovered Human (R_H). The probability distribution function was employed to discover that the spread and pandemic of COVID-19 would be drastically reduced if the measures of social distancing and public awareness and enlightenment were increased.

Keywords: *COVID-19, quarantine, mathematical modeling, probability distribution function*

I. INTRODUCTION

Mathematicians, Statisticians and Epidemiologists have tried over the years to describe infectious diseases mathematically. Several authors, like Akinwande (2005), Brauer and Castillo-Chaver (2001), Bolarin and Adeoye (2011), Enagi (2011), Abdulrahman (2009), and Bolarin and Omatola (2016), have developed various mathematical models to study dynamics or transmission of diseases to access the spread of the diseases. More importantly, the authors used the mathematical models to understand different ways to prevent epidemics and find optimal control strategies via behavior change, vaccination, treatment, quarantine and other measures.

The past decade has seen a dramatic increase in the significance attached to infectious diseases from the public health perspective. Rankish H. (2003), Daszak P., Cunningham A.A., Hyatt A.D. (2000), Donnelly C.A., Ghani A.C., Leung G.M., Hedley A.J., Fraser C., et al. (2003), and Ogbu O., Ajuluchukwu E. and Uneke C.J. (2007) noted this trend is due in part to the emergence of new and highly pathogenic infections such as Ebola virus disease (EVD), West Nile virus, Sereve Acute Respiratory Syndrome (SARS), Middle East Respiratory Syndrome (MERS), Dengue, Marburg, Variant Creutzfeidt-Jakob disease (vCJD), Lassa fever, and Corona Virus Disease (COVID-19) also known as 2019 novel Corona Virus Disease (2019-nCOVID). There are also well-publicized concerns surrounding the deliberate introduction of pathogens as bioterrorism weapons [Gani R., Leach S. (2001) and Halloran M.E., Longini I., Nizam A., Yang Y. (2002)], and the continued persistence and resurgence of older infections, several of which now boast strains resistant to more than one drug [Keeling M.J., Gilligan C.A. (2000)]. In addition, Anderson R.M., Donnelly C.A., Ferguson N.M., Woolhouse M.E.J., Watt C.J., et al.(1996), Woolhouse M., Chase-Topping M., Haydon D., Friar J., Matthews L., et al.(2001), Cyranoski D.(2001) and Miller M.W., Wild M.A.(2004) noted that there have been a number of high-profile and economically expensive disease outbreaks in domestic livestock as well as wildlife populations.

The effective management and control of such infections is increasingly done with substantial input from mathematical models, which are used not only to provide information on the nature of the infection itself, through estimates of key parameters such as the basic reproductive ratio, but also to make predictions about the likely outcome of alternative courses of action [Heesterbeek J.A.P. (2002), Keeling M.J., Woolhouse M.E.J., Shaw D.J., Matthews L., Chase-Topping M., et al. (2001), Lipsitch M., Cohen T., Cooper B., Robins J.M., Ma S., et al. (2003), Ferguson N.M., Keeling M.J., Edmunds W.J., Gant R., Grenfell B.T., et al. (2003), Fraser C., Riley S., Anderson R.M., Ferguson N.M. (2004)].

The outbreak of corona-virus disease 2019 (COVID-19) was first identified in Wuhan, China, in December, 2019, with most early cases being reported in the Wuhan city, China. Most internationally exported cases reported to date have history of travel to Wuhan either by primary contacts or secondary contacts. Camacho A., Kucharski A., Aki-Sawyerr Y., et al. (2015) summarized that in the early stages of a new infectious disease outbreak, it is crucial to understand the transmission dynamics of the infection. Estimation of changes in transmission over time can provide insights into the epidemiological situation and identify whether outbreak control measures are having a measurable effect. Funk S., Ciglenecki I., Tiffany A., et al. (2017) and Riley S., Fraser C., Donnelly C.A., et al. (2003) observed that such analysis can inform predictions about potential future growth, help estimate risk to other countries, and guide the design of alternative interventions.

However, there are several challenges to such analyses, particularly in real time. There can be a delay to symptom appearance resulting from the incubation period and delay to confirmation of cases resulting from detection and testing capacity [Aylward B., Barboza P., Bawo L., et al. (2014)]. Modeling approaches can account for such delays and uncertainty by explicitly incorporating delays resulting from the natural history of infection and reporting processes. Additionally, individual data sources might be biased, incomplete, or only capture certain aspects of the outbreak dynamics. Evidence synthesis approaches, which fit to multiple data sources rather than a single dataset (or datapoint) can enable more robust estimation of the underlying dynamics of transmission from noisy data. [Nishiura H., Klinkenberg D., Roberts M., Heesterbeek J.A.P. (2009)[

II. MATHEMATICAL MODEL FORMULATION AND ANALYSIS

A. Mathematical Formulation

The human population is divided into six mutually-exclusive compartments, namely: Susceptible Human (S_H), Susceptible Vector (S_V), Infected Human (I_H), Infected Vector (I_V), Quarantine Human (Q_H) and Recovered Human (R_H)

The mathematical parameters to be included include: recruitment rate of human due to natural birth and immigration (A_H), contact rate between the infected vector and susceptible human (B_H), contact rate between the infected human and susceptible vector (B_V), natural death rate of human (μ), the rate at which quarantine human becomes susceptible (p_1), and the rate at which recovered human becomes susceptible (p_2). The rate at which infected human leave the compartment for the quarantine human (δ), removal rate from quarantine human to recovered human (ϵ), removal rate from infected human to recovered human (γ), death due to infection for infected human (μ_1), death due to infection for quarantine human (μ_2), recruitment rate of vector (A_v) and death rate of vector due to natural causes or accident (μ_v)

B. Model Assumptions

Assumption 1: The population of the susceptible human (S_H) increases through the entrance of individual at the rate of A_H as recovered human (R_H) and quarantine human (Q_H) become susceptible at the rates of p_2 and p_1 respectively. The population decreases as the susceptible human (S_H) move into the infected

human (I_H) via interaction between the susceptible human (S_H) and infected vector (I_V) at the rate of B_H and natural death at the rate of μ. This assumption is thus represented mathematically by the differential equation with respect to time t as:

$$\frac{dS_H(t)}{dt} = A_H + p_2R_H(t) + p_1Q_H(t) - (\mu + B_HI_V(t))S_H(t) \quad (1)$$

Assumption 2: The population of the susceptible vector (S_V) increases through the constant recruitment of vector into the population at the rate of A_V and the population decreases as members of the susceptible vector (S_V) move into the infected vector compartment via interaction between the susceptible vector (S_V) and infected human (I_H) at the rate of B_V and natural death at the rate of μ. This is mathematically stated as:

$$\frac{dS_V(t)}{dt} = A_V - (\mu + B_VI_H(t))S_V(t) \quad (2)$$

Assumption 3: The population of the infected human (I_H) increases as the susceptible human (S_H) move into the infected human compartment via interaction between the susceptible human (S_H) and infected vector (I_V) at the rate of B_H, and decreases as infected human (I_H) move into the recovered class due to treatment at the rate of γ, move into the quarantine class at the rate of δ, natural death rate of μ and death due to infection at the rate of μ_1. This is stated by the differential equation as:

$$\frac{dI_H(t)}{dt} = B_HI_V(t)S_H(t) - (\mu + \mu_1 + \delta + \gamma)I_H(t) \quad (3)$$

Assumption 4: The population of the infected vector (I_V) increases as the susceptible vector move into the infected vector (I_V) compartment via interaction between the susceptible vector (S_V) and infected human (I_H) at the rate of B_V and decreases due to the natural death of the vector at the rate of μ_V. Mathematically,

$$\frac{dI_V(t)}{dt} = B_VI_H(t)S_V(t) - \mu_VI_V(t) \quad (4)$$

Assumption 5: The population of the quarantine human (Q_H) increases as the infected human (I_H) leave the compartment for quarantine compartment at the rate of δ and the population decreases as people move into the recovered human (R_H) and susceptible human (S_H) classes due to treatment at the rates of ϵ and $p_1$respectively, natural death rate of μ and death due to infection for the quarantine compartment at the rate of μ_2. Mathematically, this is stated as:

$$\frac{dQ_H(t)}{dt} = \delta I_H(t) - (\mu + \mu_2 + p_1 + \epsilon)Q_H(t) \quad (5)$$

Assumption 6: The population of the recovered human (R_H) increases as the infected human (I_H) and quarantine human (Q_H) move into the recovered class due to treatment at the rate of γ and ϵ respectively. The population decreases as the recovered human (R_H) become susceptible at the rate of p_2 and natural death occurs at the rate of μ. This is mathematically stated by the differential equation as:

$$\frac{dR_H(t)}{dt} = \gamma I_H(t) + \epsilon Q_H(t) - (\mu + p_2)R_H(t) \quad (6)$$

C. Mathematical Analysis Of The Models At Equilibrium Points

At equilibrium, $\frac{dS_H(t)}{dt} = \frac{dS_V(t)}{dt} = \frac{dI_H(t)}{dt} = \frac{dI_V(t)}{dt} = \frac{dQ_H(t)}{dt} = \frac{dR_H(t)}{dt} = 0$ (7)

Let, $S_H = x_1,\ S_V = x_2,\ I_H = x_3,\ I_V = x_4,\ Q_H = x_5,\ R_H = x_6$

Thus,

$$A_H + p_2x_6 + p_1x_5 - (\mu + B_Hx_4)x_1 = 0 \quad (8)$$

$$A_V - (\mu + B_Vx_3)x_2 = 0 \quad (9)$$

$B_H x_4 x_1 - (\mu + \mu_1 + \delta + \gamma)x_3 = 0$ (10)
$B_V x_3 x_2 - \mu_V x_4 = 0$ (11)
$\delta x_3 - (\mu + \mu_2 + p_1 + \epsilon)x_5 = 0$ (12)
$\gamma x_3 + \epsilon x_5 - (\mu + p_2)x_6 = 0$ (13)

From equation (11); $B_V x_3 x_2 = \mu_V x_4$

$$x_4 = \frac{B_V x_3 x_2}{\mu_V} \quad (14)$$

Substitute equation (14) into equation (10), it gives:

$$B_H \frac{B_V x_3 x_2}{\mu_V} x_1 - (\mu + \mu_1 + \delta + \gamma)x_3 = 0$$

$$\left[B_H \frac{B_V x_2}{\mu_V} x_1 - (\mu + \mu_1 + \delta + \gamma)\right] x_3 = 0$$

Thus,

$$\left[B_H \frac{B_V x_2}{\mu_V} x_1 - (\mu + \mu_1 + \delta + \gamma)\right] = 0, \quad or \quad x_3 = 0 \quad (15)$$

Suppose $x_3 = 0$, substitute $x_3 = 0$ into equations (9), (10), (11), (12) and (13), we have:

$A_V - \mu x_2 = 0$ (16)
$B_H x_4 x_1 = 0$ (17)
$-\mu_V x_4 = 0$ (18)
$-(\mu + \mu_2 + p_1 + \epsilon)x_5 = 0$ (19)
$\epsilon x_5 - (\mu + p_2)x_6 = 0$ (20)

From equation (16); $A_V = \mu x_2$

$$x_2 = \frac{A_V}{\mu} \quad (21)$$

From equation (18); $-\mu_V x_4 = 0$

$$x_4 = \frac{0}{-\mu_V}$$

$$x_4 = 0 \quad (22)$$

From equation (19): $-(\mu + \mu_2 + p_1 + \epsilon)x_5 = 0$

$$x_5 = \frac{0}{-(\mu + \mu_2 + p_1 + \epsilon)}$$

$$x_5 = 0 \quad (23)$$

Substitute equation (23) into equation (20); $-(\mu + p_2)x_6 = 0$

$$x_6 = \frac{0}{-(\mu + p_2)}$$

$$x_6 = 0 \quad (24)$$

Substitute equations (21), (22) and (24) into equation (8);

$A_H - \mu x_1 = 0$

$$x_1 = \frac{A_H}{\mu} \quad (25)$$

D. Stability Analysis of Disease Free Equilibrium

It should be recalled that the system of the equation of the model at equilibrium is:

$$A_H + p_2x_6 + p_1x_5 - (\mu + B_Hx_4)x_1 = 0 \quad (26)$$
$$A_V - (\mu + B_Vx_3)x_2 = 0 \quad (27)$$
$$B_Hx_4x_1 - (\mu + \mu_1 + \delta + \gamma)x_3 = 0 \quad (28)$$
$$B_Vx_3x_2 - \mu_Vx_4 = 0 \quad (29)$$
$$\delta x_3 - (\mu + \mu_2 + p_1 + \epsilon)x_5 = 0 \quad (30)$$
$$\gamma x_3 + \epsilon x_5 - (\mu + p_2)x_6 = 0 \quad (31)$$

The Jacobian matrix at disease free equilibrium is given by

$$J\left(\frac{A_H}{\mu}, \frac{A_V}{\mu}, 0, 0, 0, 0\right) = \begin{bmatrix} -\mu & 0 & 0 & -A_HB_H/\mu & p_1 & p_2 \\ 0 & -\mu & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\mu + \mu_1 + \delta + \gamma) & A_HB_H/\mu & 0 & 0 \\ 0 & A_HB_H/\mu & 0 & -\mu_V & 0 & 0 \\ 0 & 0 & \delta & 0 & -(\mu + \mu_2 + p_1 + \epsilon) & 0 \\ 0 & 0 & \gamma & 0 & \epsilon & -(\mu + p_2) \end{bmatrix} \quad (32)$$

The characteristics equation becomes

$$\begin{bmatrix} -\alpha_1 - \lambda & 0 & p_3 & p_{11} & 0 & -\alpha_2 \\ \alpha_3 & -\alpha_7 - \lambda & 0 & 0 & 0 & \alpha_2 \\ 0 & \gamma & -\alpha_8 - \lambda & \varepsilon & 0 & 0 \\ 0 & \delta & 0 & -\alpha_9 - \lambda & 0 & 0 \\ 0 & -\alpha_4 & 0 & 0 & -\alpha_6 - \lambda & 0 \\ 0 & \alpha_4 & 0 & 0 & \alpha_6 & -\mu_v - \lambda \end{bmatrix} \quad (33)$$

The determinant is:

$$(-\mu - \lambda_1)(-(\mu + \mu_1 + \delta + \gamma) - \lambda_2)(-(\mu + p_2) - \lambda_3)(-(\mu + \mu_2 + p_1 + \epsilon) - \lambda_4)(-\mu - \lambda_5)(-\mu_v - \lambda_6) = 0 \quad (34)$$

This implies

$$\lambda = -\mu, \lambda_2 = -(\mu + \mu_1 + \delta + \gamma), \lambda_3 = -(\mu + p_2), \lambda_4 = -(\mu + \mu_2 + p_1 + \epsilon), \lambda_5 = -\mu, \lambda_6 = -\mu_v \quad (35)$$

Since all the eigen-vaues are all negatives, this means the disease free equilibrium state is stable.

III. PROBABILISTIC MODEL FOR CONTROL

Some questions are pertinent to be answered while controlling the spread of Corona Virus Disease (COVID-19), the questions are:

i. "At any time instance t, what fraction of the total population is infected?"
ii. "Allowing the disease to run its full course, what fraction of the total population will become infected?"
iii. "Will person p (or group of people g) become infected within t time units of the onset of the disease?"
iv. What effects will being quarantined or self-isolation have in controlling the spread of COVID-19?
v. What effects do social distancing have in controlling the spread of COVID-19?

If the second question above is considered, it might seem strange at first, because in both the common **SIQR** (**S**usceptible-**I**nfected-**Q**uarantined-**R**ecovered) model and contact networks, it is quite possible that the entire population will become infected (and then recovered) exactly once. However, this brings the issue of disease prevention. However, the introduction of such things as vaccinations and public enlightenment/education to the model would result in individuals being initialized to be in the "recovered" state. Of course, some models (including formulations of contact networks) are probabilistic and will not always give the same answer for deterministic questions of this sort. Then one can relax the queries somewhat by rephrasing them with a probabilistic flavor:

i. "At any time instance t, what is the likelihood that the fraction of the total population is infected is at least c?"
ii. "Allowing the disease to run its full course, what is the likelihood that the fraction of the total population becomes infected is at least c?"
iii. "What is the likelihood that person p (or group of people g) becomes infected within t time units of the onset of the disease?"
iv. "What is the likelihood that person p (or group of people g) becomes infected and get quarantined within t time units of the onset of the disease and become recovered?"
v. "What is the likelihood that person p (or group of people g) who are susceptible practice social distancing within t time units of the onset of the disease and so not get infected with the disease?"

The realm of model checking allows one to pose questions of the sort above, both probabilistic and non-probabilistic.

A. Definition of the Probability Distribution for Corona Virus Disease Control

Like existing probability distributions are developed among which are Bernoulli probability distribution, Poisson probability distribution, Gamma probability distribution, Weibull probability distribution, Nakagami probability distribution, Gamma probability distribution, Beta probability distribution and many others, from the analysis of previous research works a new probability distribution function is just being formulated and can be used in controlling the spread the control of the disease.

Definition: A random variable x is having a probability density function (p.d.f.) with two parameters (α and β) given as:

$$f(x/\alpha,\ \beta) = \frac{1}{2^{\alpha}\pi^{\beta}}(x-\beta)^{\alpha-1}e^{-(x-\beta)^{\alpha}} \quad \begin{matrix} 0 \le x \le \infty \\ 0 \le \alpha \le \infty \\ 0 \le \beta \le \infty \\ x > \beta \end{matrix} \tag{36}$$

and cumulative distribution function given as:

$$Pr(X = x/\alpha,\ \beta) = e^{-(x-\beta)^{\alpha}} \tag{37}$$

It should be noted that α is denoted as shape parameter and β is denoted as location parameter.

B. Estimation Of The k-th Raw Moment

By the method of raw moments, the k-th raw moment of the probability distribution function given in equation (36) above is obtained as:

$$E(x^k) = \int_{-\infty}^{\infty} x^k f(x)dx \quad k = 1,2,3,\ldots \tag{38}$$

$$= \int_{0}^{\infty} x^k \frac{1}{2^{\alpha}\pi^{\beta}}(x-\beta)^{\alpha-1}e^{-(x-\beta)^{\alpha}}dx$$

$$= \frac{1}{2^{\alpha}\pi^{\beta}}\int_{0}^{\infty} x^k\,(x-\beta)^{\alpha-1}e^{-(x-\beta)^{\alpha}}dx$$

If $t = (x-\beta)^{\alpha} \Rightarrow t^{1/\alpha} = x-\beta \Longrightarrow x = t^{1/\alpha}+\beta$ and $dx = \frac{1}{\alpha}t^{-(1-1/\alpha)}dt$

Thus,

$$E(x^k) = \frac{1}{2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^k\, t^{(1-1/\alpha)}e^{-t}\frac{1}{\alpha}t^{-(1-1/\alpha)}dt$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^k\, e^{-t}dt \quad (39)$$

when k=1 in equation (39),

$$E(x) = \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^1\, e^{-t}dt$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left\{\int_0^{\infty}t^{1/\alpha}e^{-t}dt + \int_0^{\infty}\beta e^{-t}dt\right\}$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{1}{\alpha}\right)+\beta\right]$$
$$= \frac{\beta+\Gamma\left(1+\frac{1}{\alpha}\right)}{\alpha 2^{\alpha}\pi^{\beta}} \quad (40)$$

This is the **mean** of the distribution.

when k=2 in equation (39),

$$E(x^2) = \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^2\, e^{-t}dt$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\int_0^{\infty}t^{2/\alpha}e^{-t}dt + 2\beta\int_0^{\infty}t^{1/\alpha}e^{-t}dt + \beta^2\int_0^{\infty}e^{-t}dt\right]$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{2}{\alpha}\right)+ 2\beta\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^2\right] \quad (41)$$

when k=3 in equation (39),

$$E(x^3) = \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^3\, e^{-t}dt$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\int_0^{\infty}t^{3/\alpha}e^{-t}dt + 3\beta\int_0^{\infty}t^{2/\alpha}e^{-t}dt + 3\beta^2\int_0^{\infty}t^{1/\alpha}e^{-t}dt + \beta^3\int_0^{\infty}e^{-t}dt\right]$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{3}{\alpha}\right)+ 3\beta\Gamma\left(1+\frac{2}{\alpha}\right)+3\beta^2\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^3\right] \quad (42)$$

when k=4 in equation (39),

$$E(x^4) = \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\int_0^{\infty}(t^{1/\alpha}+\beta)^4\, e^{-t}dt$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\int_0^{\infty}t^{4/\alpha}e^{-t}dt + 4\beta\int_0^{\infty}t^{3/\alpha}e^{-t}dt + 6\beta^2\int_0^{\infty}t^{2/\alpha}e^{-t}dt + 4\beta^3\int_0^{\infty}t^{1/\alpha}e^{-t}dt + \beta^4\int_0^{\infty}e^{-t}dt\right]$$

$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{4}{\alpha}\right)+ 4\beta\Gamma\left(1+\frac{3}{\alpha}\right)+6\beta^{2}\Gamma\left(1+\frac{2}{\alpha}\right)+4\beta^{3}\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{4}\right] \quad (43)$$

and so on for other values of k.

a) Estimation Of-The Variance Of The Probability Distribution

$$Var\,(x) = E(x^{2}) - [E(x)]^{2}$$
$$= \frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{2}{\alpha}\right)+ 2\beta\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{2}\right] - \left[\frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{1}{\alpha}\right)+ \beta\right]\right]^{2}$$
$$= \frac{\alpha 2^{\alpha}\pi^{\beta}\Gamma\left(1+\frac{2}{\alpha}\right)-\left(\Gamma\left(1+\frac{1}{\alpha}\right)\right)^{2}+\beta\left(2\Gamma\left(1+\frac{1}{\alpha}\right)+\beta\right)(\alpha 2^{\alpha}\pi^{\beta}-1)}{\alpha^{2}2^{2\alpha}\pi^{2\beta}} \quad (44)$$

b) Estimation Of-The Skewness Of The Probability Distribution

$$skewness = \frac{E(x^{3})}{[\sqrt{E(x^{2})}]^{3}}$$
$$= \frac{\frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{3}{\alpha}\right)+ 3\beta\Gamma\left(1+\frac{2}{\alpha}\right)+3\beta^{2}\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{3}\right]}{\left[\frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{1}{\alpha}\right)+ \beta\right]\right]^{3/2}}$$
$$= \alpha^{1/2}2^{\alpha/2}\pi^{\beta/2}\frac{\left[\Gamma\left(1+\frac{3}{\alpha}\right)+ 3\beta\Gamma\left(1+\frac{2}{\alpha}\right)+3\beta^{2}\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{3}\right]}{\left[\left[\Gamma\left(1+\frac{1}{\alpha}\right)+ \beta\right]\right]^{3/2}} \quad (45)$$

c) Estimation Of The Kurtosis Of The Probability Distribution

$$kurtosis = \frac{E(x^{4})}{[E(x^{2})]^{2}}$$
$$= \frac{\frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{4}{\alpha}\right)+ 4\beta\Gamma\left(1+\frac{3}{\alpha}\right)+6\beta^{2}\Gamma\left(1+\frac{2}{\alpha}\right)+4\beta^{3}\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{4}\right]}{\left[\frac{1}{\alpha 2^{\alpha}\pi^{\beta}}\left[\Gamma\left(1+\frac{2}{\alpha}\right)+ 2\beta\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{2}\right]\right]^{2}}$$
$$= \alpha 2^{\alpha}\pi^{\beta}\frac{\left[\Gamma\left(1+\frac{4}{\alpha}\right)+ 4\beta\Gamma\left(1+\frac{3}{\alpha}\right)+6\beta^{2}\Gamma\left(1+\frac{2}{\alpha}\right)+4\beta^{3}\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{4}\right]}{\left[\left[\Gamma\left(1+\frac{2}{\alpha}\right)+ 2\beta\Gamma\left(1+\frac{1}{\alpha}\right)+\beta^{2}\right]\right]^{2}} \quad (46)$$

d) Graphical Representation Of The Probability Distribution Function

The graph below shows the curve of the probability distribution function (pdf) in equation (36) of the two parameters (α *and* β) at several values of the two parameters (α *and* β) as shown below:

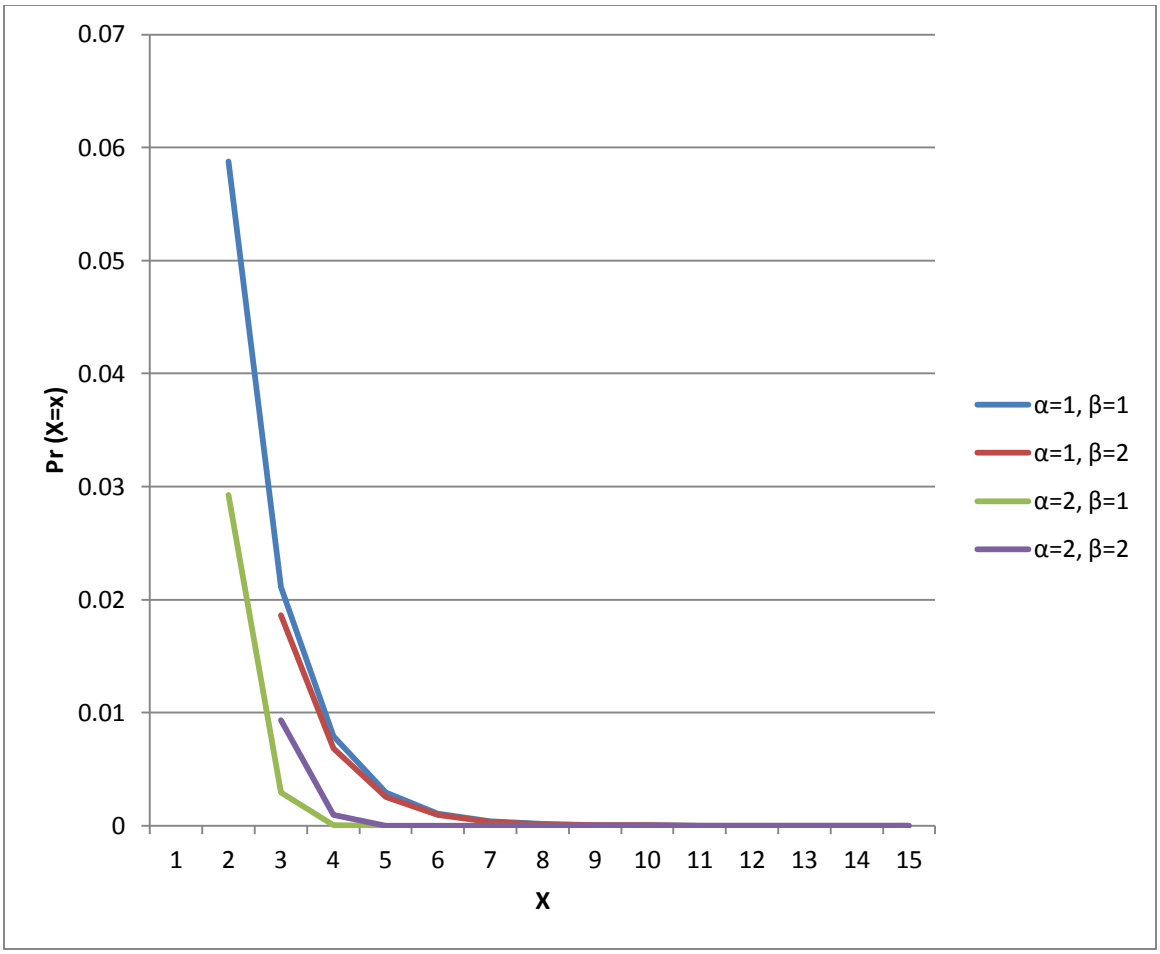

Figure 1: Graphical representation of the probability density function curve under different values of $(\alpha \; and \; \beta)$

B. Application Of The Probability Distribution To The Corona Virus Disease Control

If the two parameters in the probability distribution are explained and termed in the terms of sociological factors for the controlling of corona virus disease among human population, like making α to be the number of occurrence or outbreak of the disease in any geographical location and β to be the number or rate of awareness or publicity or enlightenment about the disease in the geographical environment, or making α to be the duration of outbreak the disease and β to be the length of social distancing maintained between individuals in the population. One will observe that the possibility of having the disease will drastically reduce among the human population as the values of the two parameters $(\alpha \; and \; \beta)$ are increased to reduce, control and eliminate the spread of the disease.

Generally most infectious diseases are preventable to a greater or lesser degree. In the case of diseases resulting from environmental exposures, prevention is a matter of eliminating or sharply reducing the factors responsible for in the environment. However, the infectious diseases may be prevented in one of the two general ways: by preventing contact and transmission of infection between the susceptible host and the source of infection, and by rendering the host unsusceptible, either by selective breeding or by induction of an effective artificial immunity.

As emphasized by Centre for Disease Control (CDC) and World Health Organization (WHO), over the past few decades, raising awareness through media campaigns have been used in an attempt to affect

various health behaviours in human populations. Typical campaigns have placed messages in media that reach large audiences, most frequently via television or radio or social media, but also outdoor media, such as billboards and posters, and print media, such as magazines and newspapers. Exposure to such messages is generally passive, resulting from an incidental effect of routine use of media. Some campaigns incorporate new technologies like the internet, mobile phones and personal digital assistants. (Kotler P. and Lee N. R., 2008)

Media campaigns can be of short duration or may extend over long periods. They may stand alone or be linked to other organized programme components, such as clinical or institutional outreach and easy access to newly available or existing products or services, or may complement policy changes. Multiple methods of dissemination might be used like peer-to-peer campaigns, campaigns at religious centres, market places, recreation centres, and many others.

IV. CONCLUSION

This research work developed a mathematical model for the control of corona virus disease using quarantine control strategy. The Disease Free Equilibrium (DFE) state was found to be stable. From the analysis of probability distribution function, the spread of the disease was also seen to be decreasing among the human population as control measures (like public awareness/awareness, social distancing, length/duration of the disease outbreak) are increased.

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