Stage Progression Model for Soil-Borne Plant Disease in Oil Palm Plantation

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***Abstract*— Managing plant disease is a crucial aspect of increasing production yields and securing output quality. This research work is conducted with a view to expanding understanding of soil-borne transmission plant disease by considering the consequences of the symptom severity as a significant role in affecting this dynamic. A class of models which describes the interactions between contagious palm hosts known as dynamical system of basal stem rot (BSR) disease stage progression is formulated using a differential equations system and investigated its dynamics. A palm host disease model is developed in which the host infection is structured into *i* stages. The basic reproduction number is determined as the threshold for BSR disease transmission model. The results revealed that the population of vulnerable palm hosts and sustained palm hosts survives, while the populations of both the contagious palm hosts have vanished. Thus, an overarching objective should be to understand the dynamics of the underlying mechanisms that drive interaction between the hosts to formulate better control strategies. This research provides crucial progress in discovering the dynamics of transmission of BSR disease that will be beneficial in aiding regulate the transmission of soil-borne disease.**

***Keywords*— deterministic modelling, stage progression model, basic reproduction number, soil-borne disease transmission, basal stem rot**

I. INTRODUCTION

The oil palm or scientifically named *Elaeis guineensis Jacq* is Malaysia's golden crop. The oil palm industry is the economic backbone and serves a crucial role in the food industry, covering together the needs of a growing world population. The industry is well-established, but there are some issues that emerge recently. The oil palm industry was facing a lot of challenges including pest and disease. In this industry, the main outbreak and damaging disease are a basal stem rot (BSR) disease aggravated by a *Ganoderma boninense* fungus [1].

Ganoderma species is a soil-borne fungus. Inside the industry, there was general consensus that the primary route of BSR infection is through roots. The predominant route for infection with BSR disease appears to be through root interaction with inoculum origins in soil [2], [3]. BSR disease outbreak and transmission began when healthy tissues in the roots came in interaction with soil inoculum or diseased plant

debris left embedded in the soil [4]. Furthermore, oil palm trees that already contaminated, rooted in the soil, and multiply. The *Ganoderma boninense* fungus which contaminated the roots will propagate to the base of the tree [5]. Through contiguity of healthy palm with infected palm's roots, the infection will lead to the formation of the whole farm. Reference [4] revealed that contaminated oil palm roots can propagate the infection throughout the four rows of plants.

Effective disease management is a critical aspect of sustaining the oil palm industry as the disease is a destructive disease that has caused producers, industries, and countries losses. Lack of knowledge about diseases and pathogens can contribute to ineffective strategies for disease control. Thus, this research focused on surveillance of BSR disease transmission using a mathematical modelling approach. Mathematical models or disease transmission models are a crucial tool for understanding the propagation of infectious diseases. It can be used to identify the most influential features in the spread of the disease, predicting disease progression and propose prevention and control strategies. The development of mathematical models offers a valuable role in examining problems in different areas like health [6], [7], [8], [9], [10], technology [11], politics [12], [13] and agricultural [14], [15], [16], [17].

The main goal of this research is to formulate a mathematical model and investigate the dynamical system of BSR disease stage progression describing the interactions between multiple contagious palm hosts. Furthermore, the determining model of basic reproduction number, R_0 as the threshold for BSR disease transmission model has been implemented. Through designing such a dynamical system, an efficient and effective method for mitigating transmission of BSR disease is expected to be achieved by taking into consideration the contagious multi-host palm.

II. THE MATHEMATICAL MODEL OF BSR DISEASE TRANSMISSION

BSR disease infection can be summarized into two stages: the most obvious disease stages progressions are asymptomatic or mild stage (stage 1) and severe stage (stage 2). In mild contamination, palms hosts reveal with one or two unopened pillars and evolving yellowish leave, other symptoms show a tiny canopy, often without the fruiting bodies of Ganoderma [18], [19]. This disordered oil palm, initially symptomatic, has the emergence of necrosis on aging leaves or has decreased the leaflet size.

While in excessive phase, palms can have more than three unopened pillars and almost aging leave fractured [19], [20]. Additionally, there are yellowing leaves with a wide spectrum of necrosis, tiny canopy and the presence of fruiting bodies of *Ganoderma* [21]. Other symptoms designate that the palm host does not develop a new bunch or fruit and that the basal stem has destruction components (rotten and hole) [18], [20].

Because of the high risk for the BSR disease and large number of palm hosts infected associated with *Ganoderma boninense* fungus, it is imperative to increase our understanding of the BSR disease dynamics. Mathematical models provided a valuable tool for acquiring insights into disease transmission and control. Such insights will potentially help lead us in assessing the efficacy and consequences of various protective and prevention strategies to yield quality and disease-free palm fruit.

However, most of these plant disease mathematical models have considered one infectious compartment alone. In this research, a model that includes two stages of the infectious compartment was analyzed. The model was extended by including mild contagious palm hosts, C_m and severe contagious palm hosts, C_s. The approach of [22] for modelling the zoonoses infectious disease was adapted in this research. The new model provides the investigation of the effects of dynamical changes. It also prevent both severity symptoms from continuing to spread in the occurrence of BSR disease. For that reason, understanding the dynamics of the stage transmission should be a priority in the strategy to curb the disease spread.

A. Development of PCiS Model

The developed model is an extension of the classical susceptible-infected-recovered (SIR) ordinary differential equation model. However, the compartment's name was revamped as prone, contagious and sustained (PC_iS) compartments. The contagious compartment is divided into two compartments, corresponding to contagious hosts with mild or severe disease symptoms. This model has been considered by others, for example [15], [22], [23], [24], [25] in the context of BSR disease dynamics. What is new here

is the consideration of severity stage progression. Specifically, this model is used to analyze under what circumstances, whether there is a two-stage progression of severity, investigate the dynamic population changes, and what are the evolutionary and epidemiological ramifications thereof.

In this model, a palm host population is denoted by N. The host population is segmented into prone palm hosts, denoted by P, hosts in a successive contagious class chain C_i, for $i = m,s$ which represents the various stages in disease progression, and a class of sustained palm hosts, denoted by S. The stages of the disease progression denoted by C_m and C_s . C_m represents the contagious palm hosts at asymptomatic or mild stage (stage 1) while C_s represents the contagious palm hosts at severe stage (stage 2). The total palm host population is therefore, $N = P + C_m + C_s + S$. A flow diagram of the PC_iS model is depicted in Fig. 1.

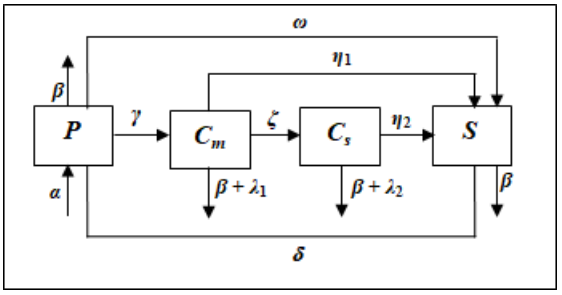

Fig. 1 A compartmental *PCiS* disease stage progression model

The descriptions of the parameters of PC_iS disease stage progression model in Fig.1 are set out in Table 1.

TABLE I
PCiS MODEL PARAMETERS DESCRIPTION

Parameters	Description
α	Recruitment of palm host
β	Rate of natural death
γ	Incidence rate of BSR disease
λ_1	Induced mortality rate at stage C_m of BSR disease
λ_2	Induced mortality rate at stage C_s of BSR disease
η_1	Recovery rate of contagious palm hosts at stage C_m
η_2	Recovery rate of contagious palm hosts at stage C_s
ζ	Transition rate of contagious compartment from C_m to C_s stage
ω	Rate of treatment
δ	Rate of transfer from sustained to prone palm hosts

The PC_iS model is expressed in mathematical terms as a system of ordinary differential equations, given as:

$$\frac{dP}{dt} = \alpha - \frac{\gamma PC_m}{N} - \beta P + \delta S - \omega P \quad (1)$$

$$\frac{dC_m}{dt} = \frac{\gamma PC_m}{N} - (\beta + \lambda_1 + \eta_1 + \zeta)C_m \quad (2)$$

$$\frac{dC_s}{dt} = \zeta C_m - (\beta + \lambda_2 + \eta_2)C_s \quad (3)$$

$$\frac{dS}{dt} = \eta_1 C_m + \eta_2 C_s - \beta S - \delta S + \omega P \quad (4)$$

with population:

$$P(t) + C_m(t) + C_s(t) + S(t) = N(t) \quad (5)$$

with initial conditions $P(0) \geq 0,\ C_m(0) \geq 0, C_s(0) \geq 0,\ S(0) \geq 0.$

The transformations of $p = \frac{P}{N}, c_m = \frac{C_m}{N}, c_s = \frac{C_s}{N}, s = \frac{S}{N}$ system in (1-4) to simplify the differential equation, where p, c_m, c_s and s represent the fractions of the number of palm hosts in classes P, C_m, C_s and S with population N. The formulated transformed system is written as:

$$\frac{dp}{dt} = \alpha - \gamma p c_m - \beta p + \delta s - \omega p \quad (6)$$

$$\frac{dc_m}{dt} = \gamma p c_m - (\beta + \lambda_1 + \eta_1 + \zeta) c_m \quad (7)$$

$$\frac{dc_s}{dt} = \zeta c_m - (\beta + \lambda_2 + \eta_2) c_s \quad (8)$$

$$\frac{ds}{dt} = \eta_1 c_m + \eta_2 c_s - \beta s - \delta s + \omega p \quad (9)$$

which is equivalent to the system in (1-4). By substitution of this transformations, (5) is written as:

$$p + c_m + c_s + s = 1 \quad (10)$$

With manipulation of (10) to produce:

$$s = 1 - p - c_m - c_s \quad (11)$$

(11) is substituted into the transformed system in (6-9) to eliminate s and yield the simplified subsystem:

$$\frac{dp}{dt} = \alpha + \delta - \delta c_m - \delta c_s - \gamma p c_m - (\beta + \delta + \omega) p \quad (12)$$

$$\frac{dc_m}{dt} = \gamma p c_m - (\beta + \lambda_1 + \eta_1 + \zeta) c_m \quad (13)$$

$$\frac{dc_s}{dt} = \zeta c_m - (\beta + \lambda_2 + \eta_2) c_s \quad (14)$$

B. Assumptions of the model

In developing this model the following assumptions are taken into account:

TABLE III
PCiS Model Assumptions

Criteria	Assumptions
Pathogen	Palm host contaminated with the *Ganoderma boninense* fungus
Transmission type	BSR outbreak caused by transmission of soil-borne disease
Soil	All palms hosts are planted in soil that is susceptible to BSR disease
Age	All ages of infected oil palm trees

Other factors	Factors of BSR disease caused by other pathogen and environmental indicators are not considered
Population	This model was considered in a closed plant population
Treatment	All palm hosts are assumed to have been treated
Sustained hosts	Not fully healed or immune from BSR disease

III. ANALYSIS OF THE MODEL

A. *Equilibrium of the model*

The disease-free equilibrium (DFE) is denoted as E_{01} and is known as the point where the disease is absent from the population [26]. This is represented in the model as $C_m = 0$ and $C_s = 0$. The endemic equilibrium (EE) is denoted as E_{12} it is recognized as the point of equilibrium where the disease exists at a constant stage. The equilibrium steady states are determined from the transformed subsystem (12-14). In particular, the equilibrium steady states are determined by setting $\frac{dp}{dt} = 0$, $\frac{dc_m}{dt} = 0$ and $\frac{dc_s}{dt} = 0$ and solving for p, c_m and c_S in (12-14). This results in two equilibrium steady states are written as follows:

Disease-free equilibrium state:

$$E_{01} = \left(\frac{\alpha + \delta}{\beta + \delta + \omega}, 0, 0 \right)$$

Endemic equilibrium state:

$$E_{01} = \left(\frac{\beta + \lambda_1 + \eta_1 + \zeta}{\gamma}, -\frac{(\beta + \lambda_2 + \eta_2)\varepsilon}{\gamma\vartheta}, -\frac{\zeta\varepsilon}{\gamma\vartheta} \right)$$

where:

$$\varepsilon = \beta^2 + \beta\zeta - \alpha\gamma - \delta\gamma + \delta\lambda_1 + \beta\lambda_1 + \beta\eta_1 + \beta\delta + \omega\eta_1 + \delta\eta_1 + \omega\beta + \omega\lambda_1 + \omega\zeta + \delta\zeta \quad \text{and}$$

$$\vartheta = \delta\beta + \delta\lambda_2 + \beta\lambda_1 + \beta^2 + \beta\eta_1 + \beta\zeta + \beta\lambda_2 + \lambda_1\lambda_2 + \lambda_2\eta_1 + \lambda_2\zeta + \beta\eta_2 + \eta_2\lambda_1 + \eta_1\eta_2 + \eta_2\zeta + \eta_2\delta + \delta\zeta$$

B. *Reproductive number*

An important concern is whether the infectious disease can invade the population or whether it will die out. To determine this, we computed the basic reproduction number, R_0 which is the number of secondary outbreaks in an otherwise entirely susceptible population developed from one infected case [27]. It is a threshold parameter, such that if $R_0 < 1$ the disease will die out, but if $R_0 > 1$ the disease can invade the population. The system possesses a disease-free equilibrium (DFE) such that if $R_0 < 1$, the DFE is locally asymptotically stable. The next generation matrix approach [27] is utilized to determine the basic number of reproduction. Let F_i be the rate of new infections in compartment i and let V_i be the rate of remaining transfer terms-of compartment i by any means other than new infection. In the system there are two infected compartments, C_m and C_s, so $i = 2$ and F_i and V_i are given by the following two equations:

$$F_i = \begin{pmatrix} \gamma P C_m \\ \zeta C_m \end{pmatrix}$$

$$V_i = \begin{pmatrix} \beta C_m + \lambda_1 C_m + \eta_1 C_m + \zeta C_m \\ \beta C_s + \lambda_2 C_s + \eta_2 C_s \end{pmatrix}$$

Now, let F and V denote the linearization of F_i and V_i at the DFE ie. $E_{01} = \left(\frac{\alpha + \delta}{\beta + \delta + \omega}, 0, 0 \right)$ by calculating the Jacobian. After linearizing the system, it turns to the following:

$$F = J_{F_i} = \begin{pmatrix} \gamma p & 0 \\ \zeta & 0 \end{pmatrix}$$

$$V = J_{V_i} = \begin{pmatrix} \beta+\lambda_1+\eta_1+\zeta & 0 \\ 0 & \beta+\lambda_2+\eta_2 \end{pmatrix}$$

The basic reproduction number is given by the spectral radius of the next generation matrix, denoted $R_0 = \rho\left(FV^{-1}\right)$. Hence,

$$R_0 = \frac{\gamma(\alpha+\delta)}{\varphi}$$

where $\vartheta = \beta^2 + \beta\lambda_1 + \beta\eta_1 + \beta\zeta + \beta\delta + \delta\lambda_1 + \delta\eta_1 + \delta\zeta + \omega\beta + \omega\lambda_1 + \omega\eta_1 + \omega\zeta$

C. Local asymptotical stability

Once the equilibrium states are established, the stability of the equilibrium states is examined. In an attempt to achieve this, the model population behavior in the vicinity of the equilibrium states is examined. We now need to determine the stability of E_{01} and E_{12}. The local stability of each equilibrium point is defined by the sign of all eigenvalues. The eigenvalues, L are solutions of the characteristic equation.

The Jacobian matrix at E_{01} is shown as below:

$$J(E_{01}) = \begin{bmatrix} -\beta-\delta-\omega & -\delta-\frac{\gamma(\alpha+\delta)}{\beta+\delta+\omega} & -\delta \\ 0 & \frac{\gamma(\alpha+\delta)}{\beta+\delta+\omega}-E & 0 \\ 0 & \zeta & -F \end{bmatrix}$$

where E and F:

$$E = \beta+\lambda_1+\eta_1+\zeta$$

$$F = \beta+\lambda_2+\eta_2$$

The characteristic equation of the above Jacobian matrix is:

$$L^3 + \frac{1}{\beta+\delta+\omega}\Big(F\beta + F\delta + F\omega - \gamma\alpha - \gamma\delta + E\beta + E\delta + E\omega + \beta^2 + 2\delta\beta + 2\beta\omega + \delta^2 + 2\delta\omega + \omega^2\ L^2\Big)$$
$$+ \Big(F\beta^2 + F\delta^2 + F\omega^2 - \beta\delta^2 + E\beta^2 + E\delta^2 + E\omega^2 - F\gamma\alpha - F\gamma\delta + FE\beta + FE\delta + FE\omega + 2F\delta\beta$$
$$+ 2F\beta\omega + 2F\delta\varpi - \gamma\alpha\beta - \gamma\alpha\delta - \gamma\alpha\omega - \gamma\delta\omega + 2E\beta\delta + 2E\beta\omega + 2E\delta\omega)L\Big)$$
$$+ F\left(-\gamma\alpha - \gamma\delta + E\beta + E\delta + E\omega\right)$$

where the eigenvalues, ω are:

$$L_1 = -\beta-\delta-\omega, \quad L_2 = -\left(\beta+\lambda_2+\eta_2\right) \text{ and } L_3 = \frac{-\gamma\alpha-\gamma\delta+E\beta+E\delta+E\omega}{\beta+\delta+\omega}$$

We present the Routh-Hurwitz necessary and sufficient conditions to have negative parts for all roots of the characteristic polynomial to specify the nature of the eigenvalues. Thus, this signifies asymptotic stability as implemented by [28], [29]. Clearly, L_1 and L_2 have negative values. From evaluating all eigenvalues, the real parts of all eigenvalues have negative signs when R_0<1, hence the equilibrium point E_{01} of the model is locally asymptotically stable.

In order to determine the stability of the endemic equilibrium point E_{12}, Jacobian matrix eigenvalues values at E_{12} are calculated as:

$$J(E_{12}) = \begin{bmatrix} \frac{(\beta+\lambda_2+\eta_2)\varepsilon}{\vartheta} - \beta - \delta - \omega & -\delta-\beta-\lambda_1-\eta_1-\zeta & -\delta \\ -\frac{(\beta+\lambda_2+\eta_2)\varepsilon}{\vartheta} & \beta+\lambda_1+\eta_1+\zeta-E & 0 \\ 0 & \zeta & -F \end{bmatrix}$$

The corresponding characteristic equation is given by:

$$L^3 + AL^2 + BL + C = 0$$

where ;

$$A = \frac{F\vartheta - \lambda_1\vartheta - \eta_1\vartheta - \zeta\vartheta + E\vartheta - \varepsilon\beta - \varepsilon\lambda_2 - \varepsilon\eta_2 + \delta\vartheta + \omega\vartheta}{\vartheta},$$

$$B = \frac{1}{\vartheta}\left(-\varepsilon\beta E - \varepsilon\lambda_2 E - \varepsilon\eta_2 E - \beta\vartheta\lambda_1 - \beta\eta_1\vartheta - \beta\vartheta\zeta + \beta\vartheta E - \delta\vartheta\beta - \delta\vartheta\lambda_1 - \delta\eta_1\vartheta - \delta\vartheta\zeta\right.$$
$$+ \delta\vartheta E - \omega\vartheta\beta - \omega\lambda_1\vartheta - \omega\eta_1\vartheta - \omega\zeta\vartheta + \omega E\vartheta - \varepsilon\beta\delta - \varepsilon\lambda_2\delta - \varepsilon\eta_2\delta - \beta^2\vartheta$$
$$\left. - F\lambda_1\vartheta - F\eta_1\vartheta - F\zeta\vartheta + FE\vartheta - F\varepsilon\beta - F\varepsilon\lambda_2 - F\varepsilon\eta_2 + F\vartheta\delta + F\vartheta\omega\right)$$

and

$$C = -\frac{1}{\vartheta}\left(F\varepsilon\beta E + F\varepsilon\lambda_2 E + F\varepsilon\eta_2 E + F\beta\vartheta\lambda_1 + F\beta\vartheta\eta_1 + F\beta\vartheta\zeta - F\vartheta\beta E + F\delta\vartheta\beta\right.$$
$$+ F\delta\vartheta\lambda_1 + F\delta\vartheta\eta_1 + F\delta\vartheta\zeta - F\delta\vartheta E + F\omega\vartheta\beta + F\omega\lambda_1\vartheta + F\omega\eta_1\vartheta + F\omega\zeta\vartheta - F\omega E\vartheta$$
$$\left. + \varepsilon\beta\zeta\delta + \varepsilon\beta F\delta + \varepsilon\lambda_2\zeta\delta + \varepsilon\lambda_2 F\delta + \varepsilon\eta_2\zeta\delta + \varepsilon\eta_2 F\delta + F\beta^2\vartheta\right)$$

Regarding the analysis by [30], the equilibrium E_{12} is locally asymptotically stable if all roots of the characteristic model equation have negative real parts. This requirement can be accomplished by complying with the Routh Hurwitz criteria [31]. All roots of this characteristic equation have negative real parts if and only if its coefficients are positive (A>0, C >0 and AB-C >0). Thereby, all of the eigenvalues of the characteristic equation are negative real parts. Consequently, the endemic equilibrium E_{12} of the PC_iS model is locally asymptotically stable. From evaluation, all eigenvalues have negative real parts for R_0 >1.

IV. NUMERICAL ANALYSIS

In this article, we present the empirical results from the proposed model. Firstly, this model is achieved with Maple software using a fourth-order Runge–Kutta scheme. The parameters used in the numerical simulations are based on empirical research conducted by [32], [33], [34], [35], [36], [37].

In which case, the result of R_0 equals 0.13687 with 0.14 in average. When R_0 is less than 1, on average each infectious palm host infects less than one other palm host, and the pathogen will die out in the population. From numerical results, we conclude that if $R_0 < 1$, the oil palm plantation will free from BSR disease.

In this section, the numerical analysis of the model in Eq. 1-4 is presented to show the dynamical changes of each population with the transmission of BSR disease in oil palm plantations. Below are the diagrams that resulted from the numerical simulation. The dynamics of the models are shown. Fig. 2 – Fig. 5 demonstrate the prone palm hosts, mild contagious palm hosts, severe contagious palm hosts and sustained palm hosts, respectively.

In Fig. 2, it can be observed that the prone palm hosts population, P decreases strongly and at about after 5 years it increases and stabilize at a certain steady value. While in Fig. 3, it can be observed that the mild

contagious palm hosts, C_m will be decreasing and disappear after 3 years. Likewise with severe contagious palm hosts, C_s. Nevertheless, it initially spikes sharply and achieves a certain maximum, thus plunging and approaching an equilibrium quantity. In the meantime, in Fig. 5, we observe that the population of sustained palm hosts declines at a slower rate and eventually survive at a certain steady value.

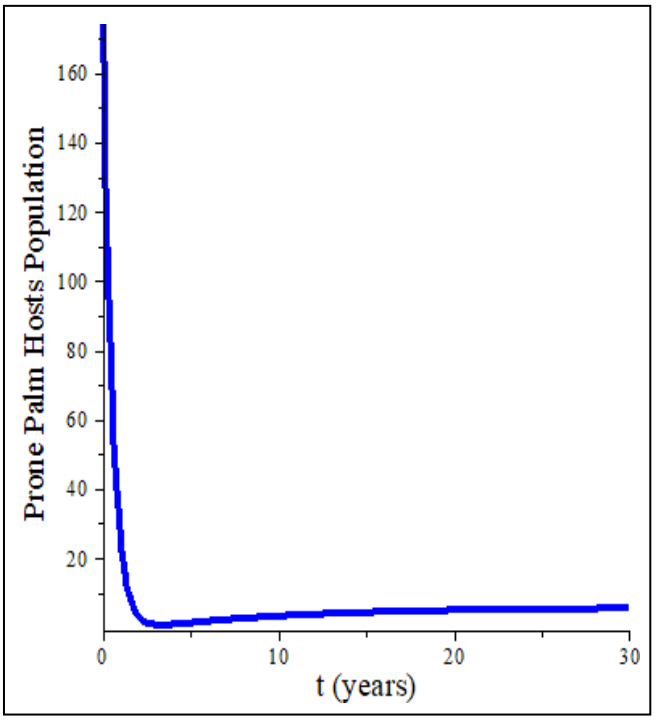

Fig. 2 Graphical simulations of prone palm hosts against time

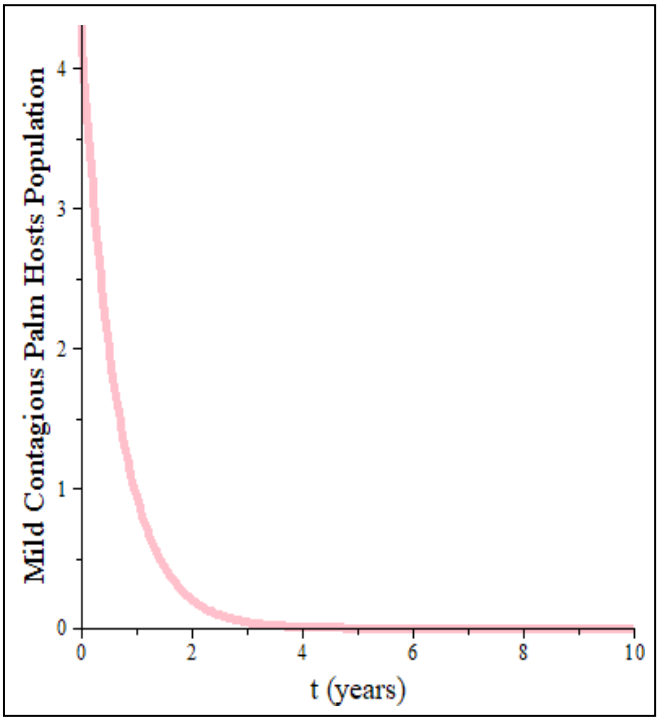

Fig. 3 Graphical simulations of mild contagious palm hosts against time

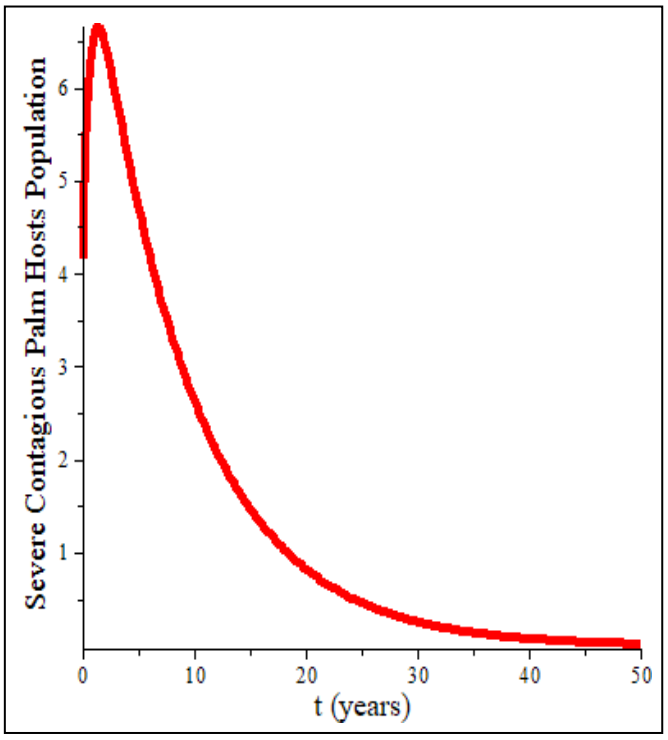

Fig. 4 Graphical simulations of severe contagious palm hosts against time

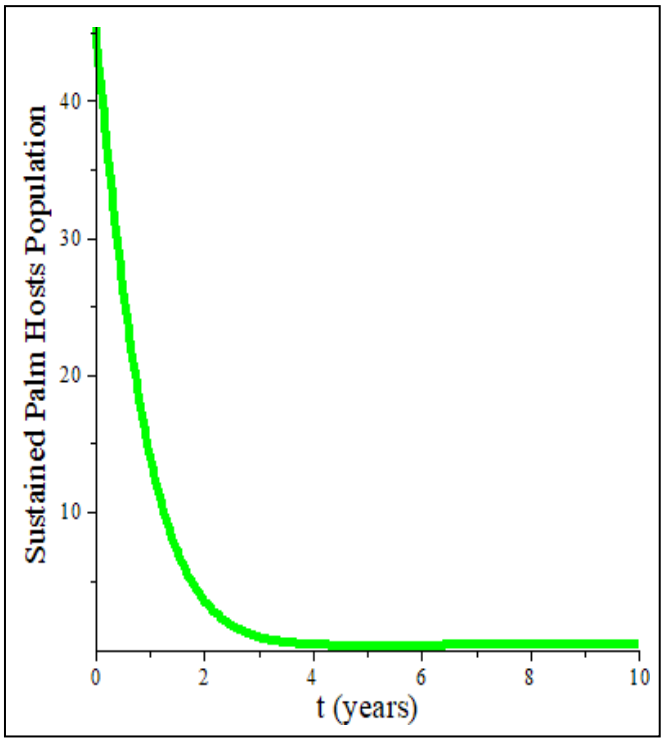

Fig. 5 Graphical simulations of sustained palm hosts against time

Meanwhile, Fig. 6 represents the dynamical changes for the entire palm hosts population. This figure shows the dynamics of BSR disease in oil palm plantation in Malaysia. From this figure, a limited population of prone palm hosts and sustained palm hosts remains, as both populations of the contagious palm hosts die off. In this case, the condition found in Malaysia is considered to be congenial.

On the basis of the numerical analysis of the population as a whole, it was found that the eruption of BSR disease does not disseminate and that mild contagious palm hosts would die out in less than five years. This is attributed to numerous measures of prevention and control in combating the oil palm plantation epidemic. Nevertheless, the severe contagious palm hosts should be taken into consideration and given more priority because they still exist the oil palm plantation (within 50 years) and take much longer time to die out. Treatment needs to be elevated so that BSR disease can be restored and sustained to produce yield. It needs to be enhanced, as well as surveillance so that the contagious palm hosts do not propagate and infect other plants.

This numerical analysis shows that beyond a certain period of time both contagious palms hosts inevitably stabilize to zero. It relates to this model since it takes into consideration the assumptions that all of these palm hosts were treated. It also coincides with the R_0 determination value, i.e 0.14 which is if R_0 <1, then the outbreak of the disease will not spread. Continuous prevention and treatment should, however, be undertaken to regulate the spread of BSR disease because susceptible palm hosts do not collapse to extinction. From numerical analysis, the number of prone palm hosts will be stable at 5.6571626 while the number of sustained palm hosts will be remained at 0.6629357. It means that 5,657,163 palm hosts may be prone to BSR disease while a total of 662,936 palm hosts will be uninterrupted but it may be re-infected and other trees may be infected. This is a relatively large amount and has the ability to cause BSR disease. Thus, preventive measures and treatments should be routinely carried out in Malaysia's oil palm plantation.

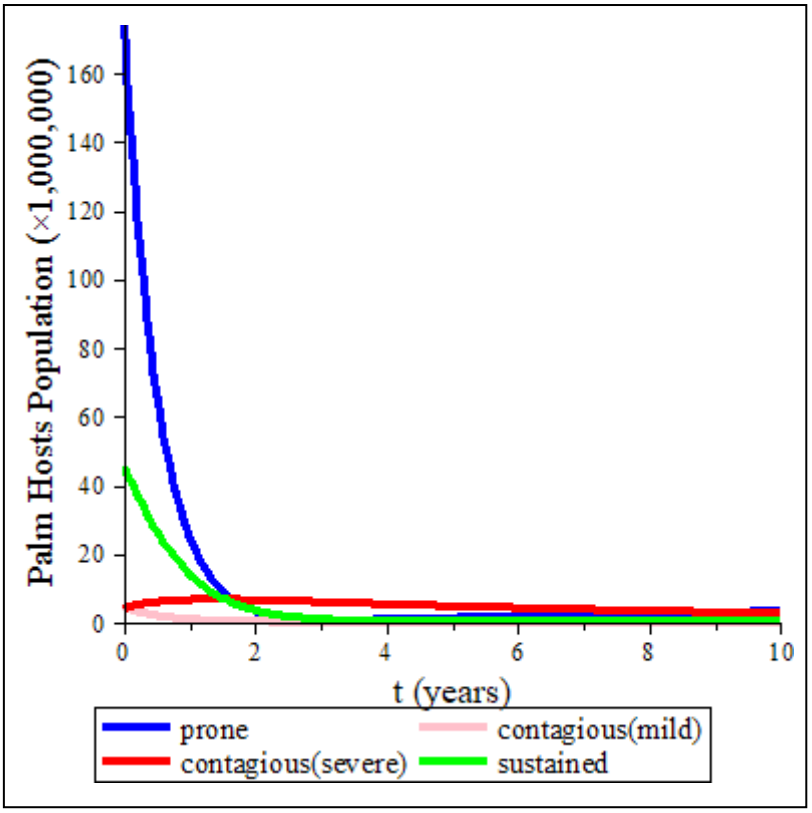

Fig. 6 Dynamical changes of palm hosts population against time

V. CONCLUSION

In this article the dynamical stage transmission on the spread of the BSR disease infection was examined. A new mathematical model has been proposed, which takes into consideration the existence of mild and extreme infectious palm hosts. The advantage of this model is that the outcome can be used to assess the most crucial stage of disease progression and requires further attention. The theoretical studies of multi-stage progression model and their numerical simulations have been highlighted in this article. The findings

of this model indicate that the severe contagious palm hosts, C_s, still decimate and persist in the oil palm plantation and take a much longer period to vanish. It is particularly detrimental to the oil palm population because it can transmit BSR disease and actions should be taken to eradicate contagious palm hosts, even if it eventually fades to extinction. Field management must better understand and enhance strategy for the early detection and control of the spread of *Ganoderma boninense* fungi.

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