DYNAMIC MODEL OF THE SPREAD OF VASCULAR STREAK DIEBACK DISEASE (VSD) BY ONCOBASIDIUM THEOBROMAEON COCOA TREES WITH TRICHODERMA SP CONTROL

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ABSTRACT

Disease Vascular Streak Dieback (VSD) is caused by the fungus Oncobasidium theobromae, which produces basidiospores and grows on infected cacao branches. Biological control of VSD disease used Trichoderma sp. The SIS model is represented the spread of this disease and provide two critical points, namely disease free critical point and the endemic critical point. Analysis stability system at the critical point used the Linearization method and the Routh-Hurwitz indicated that the system is stable and appears to have a threshold for the growth rate of Oncobasidium theobromae that must be less than 0.767. The existence and stable threshold for a spore growth rate of Oncobasidium theobromae is more remarkable than 0.767. Simulations at both critical points showed that the spore growth rate of Oncobasidium theobromae is very fluent in spreading the disease. In this case, suppressing the growth rate of Oncobasidium theobromae could be used as an excellent treatment to control the disease.

Keywords   : Mathematical Model, Oncobasidium theobromae, Trichoderma sp, VSD.

I. INTRODUCTION

Indonesia was once the world's third largest exporter of cocoa beans in 2010, with dry bean production of 550,000 tonnes[1]. For Indonesia, cocoa is the third largest foreign exchange-earning agricultural commodity after palm oil and rubber, the primary source of income for 1.7 million farming families spread across almost all provinces [2]. It is one of the reasons why it is essential to increase cocoa productivity and quality. One of Indonesia's essential diseases in cocoa plants is Vascular Streak Dieback (VSD). At the end of 2009, this pathogen was discovered in Jembrana cocoa plantations in Bali. One hundred ten thousand six hundred fourteen cocoa workers in Jembrana have experienced losses due to unproductive cocoa plants since 1980. Wood vein disease causes yield losses of around 30,000 tons annually and $ 28,000,000 annually globally [3].

Control of VSD disease can be done through several mechanisms, such as chemical control using systemic fungicides, technical culture, use of resistant clones, and biological control [4]. Trichoderma sp is a group of biocontrol agents that can inhibit the growth of several pathogenic fungi and bacteria. Apart from that, this fungus also can parasitize the hyphae of these pathogens. Trichoderma sp produces several pectinase, silanase, and chitinase cellulase enzymes, which can damage pathogen cell walls.

Based on this description, the author is interested in studying the dynamic model of the spread of Vascular Streak Dieback (VSD) disease by Oncobasidium theobromae on cocoa trees controlled by Trichoderma sp. involving a population of cocoa plants, a population of the fungus Oncobasidium theobromae, and a population of Trichoderma sp. The mathematical model was adapted from the SIS (Susceptible Infected Susceptible) model by paying attention to the interactions between populations in the system, which play a role in the spread of the disease. The SIS model is used to adjust the characteristics of VSD disease and the interactions that occur in the VSD disease spread model. Stability analysis is carried out using the Linearization method, which is analyzed at the critical points of the system that represent it.

II. METHODS

This research used secondary data, including population size, life span, natural death, migration rate, transmission rate, and growth rate. Secondary data in this research comes from several journals, articles, and books that have information about VSD disease.

Five variables in this research are divided based on the SIS mathematical model that has been constructed. The cocoa subpopulation is divided into 2, namely the vulnerable cocoa subpopulation (K_S) and the infectious cocoa subpopulation (K_I). There is a subpopulation of the fungus Oncobasidium theobromae as a pathogen that causes VSD, which is divided into two according to its life phase, namely the spore subpopulation (S_J) and a subpopulation of hyphae (H_J). There is also a sub-population of Trichoderma sp (T), a biological agent acting as a disease control agent.

The data that has been obtained is converted into parameter values that are used to analyze the data, namely determining the critical point, ensuring the existence of the critical point and analyzing the stability of that to obtain a solution to the problem regarding the dynamic model of the spread of Vascular Streak Dieback (VSD) disease by *Oncobasidium theobromae* on cocoa trees with the influence of Trichoderma sp. using the linearization method and the Routh-Hurwitz Criterion.

III.   RESULTS AND DISCUSSION

This research is a mathematical review of the transition and transmission of cacao tree and pathogen subpopulations from one phase to another, represented in a dynamic model of the spread of VSD disease. The dynamic model for spreading VSD disease in cocoa plants was built based on facts and assumptions adapted to the SIS model.

Transmission of members of a subpopulation of susceptible cocoa trees (K_S) to subpopulation (K_I) with a fungal infection rate of as much as due to fungal spores (S_J) infect (K_S). Transmission of members of the infectious cocoa subpopulation (K_I) returns to the subpopulation with a transmission rate as large as that caused by controlling Trichoderma sp (T) in a subpopulation of infected cacao trees (K_I).

In fungal populations, phase transitions occur. Transition spores of Oncobasidium theobromae (S_J) develop into hyphae (H_J) with a rate of change. Spore growth rate (S_J) is the transition rate of spores that have developed into hyphae (H_J), which then go through several phases and return to a subpopulation of spores (S_J). Spore subpopulations (S_J) which develops into a subpopulation of hyphae (H_J) will go through several phases and return to a subpopulation of spores with a spore transition rate of θ.
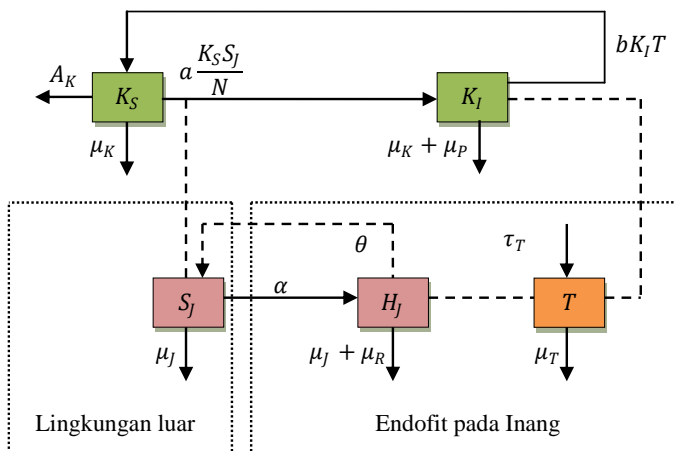

Figure 1 : The compartmental diagram of the spread of VSD disease

There is a population of Trichoderma sp (T) which suppresses the spread of VSD disease by parasitism on hyphae (H_J) Oncobasidium theobromae in infected cocoa trees (K_I). Growth of Trichoderma sp (T) is a natural growth and parasitism of pathogens with a natural growth rate of, as well as a reduction in

the subpopulation of Trichoderma sp (T) due to natural death at a rate of μ_T. Each population has a death rate. A subpopulation of vulnerable cocoa trees (K_S) decreases with a natural death rate of μ_K. The subpopulation of infected cacao trees is reduced due to infection by pathogens and natural mortality at a rapid rate $\mu_K + \mu_P$. Spore subpopulations *Oncobasidium theobromae* (S_J) decreases due to natural death with a natural death rate of μ_J. Reduced hyphal subpopulation *Oncobasidium theobromae* (H_J) due to natural mortality and parasitism *Trichoderma sp* with a death rate of $\mu_J + \mu_R$. A scheme of growth, movement, death, and interactions of each population that plays a role in the spread of VSD disease on cocoa plants can be seen in Figure 1.

From the compartment diagram in Figure 1, a system of differential equations can be built from equations that represent changes in the number of members of each subpopulation so that the dynamic model for the spread of VSD disease is as follows.

$$\frac{\partial K_S}{\partial t} = A_K + b \cdot K_I \cdot T - a \cdot \frac{K_S \cdot S_J}{N} - (\mu_K \cdot K_S) \tag{1}$$

$$\frac{\partial K_I}{\partial t} = a \cdot \frac{K_S \cdot S_J}{N} - b \cdot K_I \cdot T - (\mu_K + \mu_P) \cdot K_I \tag{2}$$

$$\frac{\partial T}{\partial t} = \tau_t \cdot H_J \cdot T - \mu_t \cdot T \tag{3}$$

$$\frac{\partial S_J}{\partial t} = \theta \cdot H_J - \alpha \cdot S_J - \mu_J \cdot S_J \tag{4}$$

$$\frac{\partial H_J}{\partial t} = \alpha \cdot S_J - \mu_J \cdot H_J - \mu_R \cdot H_J \cdot T \tag{5}$$

3.1. Critical Point T_{DFE} (Disease Free Equilibrium)

Critical point T_{DFE} describes a VSD disease-free condition expressed as follows.

$$T_{DFE} = (K_S, K_I, T, S_J, H_J) = (\frac{A_K}{\mu_K}, 0, 0, 0, 0) \tag{6}$$

Existence Critical point T_{DFE} guaranteed to exist without conditions.

3.2. Critical Point T_{END} (Disease Endemic Equilibrium)

Critical point T_{END} describes the conditions for the spread of VSD disease which are expressed in the following matrix.

$$
\begin{bmatrix} K_S \\ K_I \\ T \\ S_J \\ H_J \end{bmatrix} = \begin{bmatrix} \dfrac{d_1}{f_1} \\[2ex] \dfrac{d_2}{f_2} \\[2ex] \dfrac{\alpha\theta - \alpha\mu_J - \mu_J^2}{(\alpha + \mu_J)\mu_R} \\[2ex] \dfrac{\theta\mu_t}{\tau_t(\alpha + \mu_J)} \\[2ex] \dfrac{\mu_t}{\tau_t} \end{bmatrix}
$$

So that T_{END} positive is given the existence condition for the critical point T_{END} namely as follows.

$$\alpha\theta - \alpha\mu_J - \mu_J^2 > 0 \tag{7}$$

Until obtained

$$\theta > \frac{\mu_J(\alpha + \mu_J)}{\alpha}. \tag{8}$$

This means that the spore growth rate has a threshold for the occurrence of endemic conditions, namely equation (8).

3.3. Critical Point Stability Analysis of T_{DFE}

Critical point $T_{DFE} = \left(\frac{A_K}{\mu_K}, 0, 0, 0, 0\right)$ is not a zero critical point so a coordinate transformation needs to be carried out. The transformation is carried out by defining a new variable (K) as follows.

$$K = K_S - \frac{A_K}{\mu_K}$$

$$\Leftrightarrow K_S = K + \frac{A_K}{\mu_K} \tag{9}$$

By substituting equation (9) into equations (1) - (5), we obtain a system of differential equations in new coordinates. Then a Jacobi matrix is formed from the system of differential equations in new variables which are then evaluated at the point (0,0,0,0,0). Critical point substitution T_{DFE} (6) into the Jacobi matrix as follows

$$
J_{DFE} = \begin{bmatrix} -\mu_K & 0 & 0 & -\dfrac{aA_K}{\mu_K N} & 0 \\[2ex] 0 & -\mu_K - \mu_P & 0 & \dfrac{aA_K}{\mu_K N} & 0 \\[2ex] 0 & 0 & -\mu_t & 0 & 0 \\[1ex] 0 & 0 & 0 & -\alpha - \mu_J & \theta \\[1ex] 0 & 0 & 0 & \alpha & -\mu_J \end{bmatrix}
$$

The eigenvalues can be obtained by determining the lambda value in the equation $\det(J - \lambda I) = 0$. The determinant of the matrix is sought using the Cofactor Expansion Method on the matrix J_{DFE} so that the characteristic polynomial is obtained

$$-\left(-\mu_K - \lambda\right)\left(-\mu_K - \mu_P - \lambda\right)\left(\mu_t + \lambda\right)\left(a_2\lambda^2 + a_1\lambda + a_0\right) = 0 \tag{10}$$

From equation (10), the following eigenvalues are obtained

$$\lambda_1 = -\mu_K$$
$$\lambda_2 = -\mu_K - \mu_P \tag{11}$$
$$\lambda_3 = -\mu_0$$

For the nonlinear part of the characteristic equation $(a_2\lambda^2 + a_1\lambda + a_0)$, the roots are tested using Criteria *Routh-Hurwitz* according to Table 1 below.

Table 1 : Routh-Hurwitz Stabilit T_{DFE}

λ^2	a_2	a_0
λ^1	a_1	0
λ^0	$b_1 = a_0$	0

Disease-free critical point stability is met with conditions

$$b_1 = a_1 = -\theta + \mu_J(\alpha + \mu_J) > 0$$
$$\frac{\mu_J(\alpha + \mu_J)}{\alpha} > \theta \tag{12}$$

3.4. Critical Point Stabilty of Analysis T_{END}

Critical point $T_{END} = \left(K_S, K_I, T, S_J, H_J\right)$ is not a zero critical point so it is necessary to carry out a coordinate transformation so that T_{DFE} in the new coordinates is the zero critical point. The transformation is carried out by defining a new variable, namely L, M, N, O, and P for the variables in the matrix. then a system of differential equations in new coordinates is obtained, then a Jacobi matrix is formed which is evaluated at the point (0,0,0,0,0) and substituting the critical point T_{END} into the Jacobi matrix (J_{END}). The determinant of the matrix is sought using the Cofactor Expansion Method on the matrix J_{END} so that the characteristic polynomial is obtained

$$(a_2\lambda^2 + a_1\lambda = a_0)(z_2\lambda^2 + z_1\lambda + z_0) = 0 \tag{13}$$

The stability of the system is determined by examining the coefficients on the characteristic polynomial. From the first factor of equation (13) the characteristic equation is obtained $P_1(\lambda) = a_2\lambda^2 + a_1\lambda + a_0$ with:

$$a_2 = N\alpha\mu_R\tau_t + N\mu_J\mu_R\tau_t$$

$$a_1 = N\left(2\left(\mu_K + \frac{1}{2}\mu_P\right)(\alpha + \mu_J)\mu_R + b\left(\alpha\theta - \alpha\mu_J - \mu_J^2\right)\right)\tau_t + a\theta\mu_R\mu_t$$

$$a_0 = N\tau_t\mu_R\left(\alpha + \mu_J\right)\mu_K^2 + \left(\left(\mu_P\left(\alpha + \mu_J\right)\mu_R + b\left(\alpha\theta - \alpha\mu_J - \mu_J^2\right)\right)N\tau_t + \mu_t a\theta\mu_R\right)\mu_K + a\theta\mu_P\mu_R\mu_t$$

For the characteristic equation $P_1(\lambda)$, the roots are tested using Criteria *Routh-Hurwitz.*

Table 2 Routh-Hurwitz Stability of First Factor $P_1(\lambda)$ T_{END}

λ^2	a_2	a_0
λ^1	a_1	0
λ^0	$b_1 = a_0$	0

So that the critical point T_{END} stable, then all terms in the second column of Table 2 must have the same sign. Because $a_2 > 0$, polynomial coefficients a_1 and a_0 guaranteed to be positive with the existence condition of T_{END} that is $> \frac{\mu_J(\alpha+\mu_J)}{\alpha}$.

For the second factor $P_2(\lambda)$ that is $(z_2\lambda^2 + z_1\lambda + z_0)$ in eqution (13) the roots are tested using the Criterion *Routh-Hurwitz* according to Table 3 below.

Table 3 : Routh-Hurwitz Stability of the Second Factor $P_2(\lambda)$ T_{END}

λ^3	z_3	z_1
λ^2	z_2	z_0
λ^1	b_1	0
λ^0	$c_1 = z_0$	0

with:

$$z_3 = \alpha + \mu_J$$
$$z_2 = \alpha^2 + (\theta + 2\mu_J)\alpha + \mu_J^2$$
$$z_1 = (\alpha\theta - \alpha\mu_J - \mu_J^2)\mu_t$$
$$z_0 = c_1 = (\alpha\theta - \alpha\mu_J - \mu_J^2)(\alpha + \mu_J)\mu_t$$
$$b_1 = \frac{\alpha\theta\mu_t(\alpha\theta - \alpha\mu_J - \mu_J^2)}{\alpha^2 + \alpha\theta + 2\alpha\mu_J + \mu_J^2}$$

Polynomial coefficients $P_2(\lambda)$ that is z_0, z_1 dan b_1 guaranteed to be positive with the existence condition of T_{END} is $\theta > \frac{\mu_J(\alpha+\mu_J)}{\alpha}$. Critical point stability T_{END} determined by the eigenvalues of the characteristic equation (13). Based on stability analysis *Routh-Hurwitz* in the second column of Table 2 and Table 3 there is no change in the if sign $\theta > \frac{\mu_J(\alpha+\mu_J)}{\alpha}$ so that all the roots of the characteristic equation $P_1(\lambda)$ yaitu λ_1, λ_2 and P_2 are $\lambda_3, \lambda_4, \lambda_5$ has negative roots and with these conditions fulfilled, it can be concluded that the system is stable at the endemic critical point T_{END}.

3.5. Simulation

Simulations are also carried out by providing initial values for all variables at each critical point which are used to describe the curves in the simulation. The initial variable values and parameter values that will be used in the simulation are in Table 4 and Table 5 as follows.

Table 4 : Initial values of research variables

Variable	Information	Value	Dimention
K_S	Sub population of susceptible cacao tree	60	Population
K_I	Sub population of infection cacao tree	60	Population

T	Trichoderma sp	40	Population
S_J	Phase spore of Oncobasidium theobromae	30	Population
H_J	Phase hyphae of Oncobasidium theobromae	20	Population

Table 5 : Research parameter values

Parameter	Information	Value	Dimension	Source
A_K	Recruitment rate of cacao trees	100	Population	Assumption
μ_K	Natural death rate of cacao trees	0.00361	Day	Saputro dan Octaviana, 2020
a	Transmission rate of susceptible cacao trees become infection	0.0013699	Day	Fauziyah,dkk. 2018
b	Transmission rate of cacao trees infection back to susceptible	0.01	Day	Fauziyah,dkk. 2018
μ_P	Death rate of cacao trees caused Oncobasidium theobromae	0.001	Day	Fauziyah,dkk. 2018
μ_J	Death rate of Oncobasidium theobromae	0.0005	Day	Fauziyah,dkk. 2018
θ	Growth rate of Oncobasidium theobromae spore	0.865	Day	Syarat eksis
α	Transition rate pf spore become hyphae	0.0067	Day	Fauziyah,dkk. 2018
τ_T	Natural growth rate of Trichoderma sp	0.68	Day	Thamrin dan Atria Martina, 2020.
μ_R	Death rate of Oncobasidium theobromae caused parasitisme	0.9	Day	Herman, dkk. 2014.
μ_T	Natural death rate of Trichoderma sp	0.868	Day	Hapsari, 2003.

Solution curves with initial values and predetermined parameter values are displayed at 12-month time intervals.

3.5.1. Simulation of Disease Free Critical Point (T_{DFE})
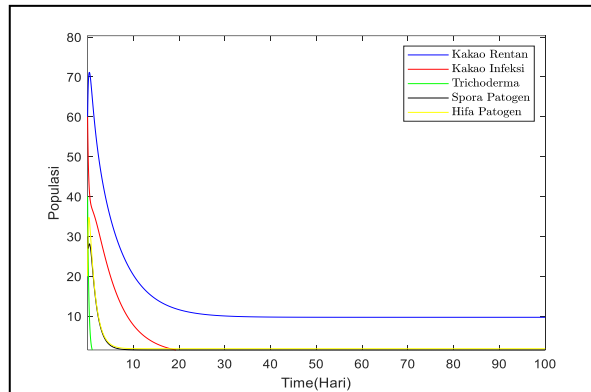

Figure 2 : Disease-free critical point simulation curve T_{DFE}

Simulation curve for critical point T_{DFE} figure 2 interprets VSD disease-free conditions where in this condition, the growth of the susceptible cocoa subpopulation experiences a drastic increase in a short time and again experiences a decrease in the number of subpopulations as time increases until it reaches a stable condition within 25 days. The decline in the number of vulnerable cacao subpopulations is due to natural mortality. The infected cacao subpopulation experienced a decline and continued to decline until it reached zero in less than 20 days. The decrease in the number of infected cocoa subpopulations was due to the significant decrease in the subpopulation of disease-causing pathogenic hyphae and spores as well as the natural death of the infected cocoa subpopulation.

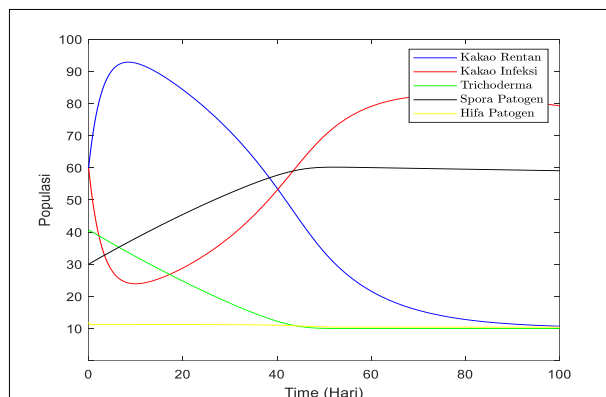### 3.5.2. Simulation of Endemic Critical Point (T_{END})

Figure 3 : Endemic critical point simulation curve T_{END}

Endemic condition simulation curve for T_{END} Figure 4.3 shows the number of vulnerable cocoa subpopulations which have increased and continued to decrease in less than 20 days. This condition is inversely proportional to the infected cocoa subpopulation, which experienced

a decline in numbers reaching less than 30 populations and then increased again at the same time as the number of susceptible cocoa subpopulations decreased. The decrease in the susceptible cacao subpopulation and the increase in the infectious cacao subpopulation were due to a decrease in the subpopulation *Trichoderma sp* which controls the spread of VSD disease.

The subpopulation of pathogenic fungal spores experienced an increase in the number of subpopulations from an initial number of 30 populations that could double to 60 populations in less than 50 days.

Increase in the development of subpopulations of spores and hyphae *Oncobasidium theobromae* meaning low effectiveness *Trichoderma sp* as a disease control fungus where *Trichoderma sp* experienced a decrease in the number of sub-populations, which initially was 40 sub-populations, which continued to decline until the number was almost equivalent to the number of sub-populations of pathogenic hyphae and also experienced a constant condition. Condition of the number of sub-populations *Trichoderma sp* constant can be an existential consideration *Trichoderma sp* as controlling the spread of disease. Apart from that, a growth model can be designed *Trichoderma sp* which is able to overcome persistent endemic conditions.

IV. CONCLUSION

1. The dynamic model for the spread of VSD disease can be arranged into a system of differential equations as follows.

$$\frac{\partial K_S}{\partial t} = A_K + b \cdot K_I \cdot T - a \cdot \frac{K_S \cdot S_J}{N} - (\mu_K \cdot K_S)$$

$$\frac{\partial K_I}{\partial t} = a \cdot \frac{K_S \cdot S_J}{N} - b \cdot K_I \cdot T - (\mu_K + \mu_P) \cdot K_I$$

$$\frac{\partial T}{\partial t} = \tau_t \cdot H_J \cdot T - \mu_t \cdot T$$

$$\frac{\partial S_J}{\partial t} = \theta \cdot H_J - \alpha \cdot S_J - \mu_J \cdot S_J$$

$$\frac{\partial H_J}{\partial t} = \alpha \cdot S_J - \mu_J \cdot H_J - \mu_R \cdot H_J \cdot T$$

2. Two critical points were obtained from the dynamic model of the spread of VSD disease, namely T_{DFE} and T_{END}. Critical Point which describes a disease-free condition T_{DFE} is as follows.

$$T_{DFE} = (K_S, K_I, T, S_J, H_J) = (\frac{A_K}{\mu_K}, 0, 0, 0, 0)$$

T_{DFE} exists unconditionally and is stable with conditions $\frac{\mu_J(\alpha + \mu_J)}{\alpha} > \theta$. The critical points that describe endemic disease conditions are as follows.

$$\begin{bmatrix} K_S \\ K_I \\ T \\ S_J \\ H_J \end{bmatrix} = \begin{bmatrix} \dfrac{d_1}{f_1} \\[2ex] \dfrac{d_2}{f_2} \\[2ex] \dfrac{\alpha\theta - \alpha\mu_J - \mu_J^2}{(\alpha + \mu_J)\mu_R} \\[2ex] \dfrac{\theta\mu_t}{\tau_t(\alpha + \mu_J)} \\[2ex] \dfrac{\mu_t}{\tau_t} \end{bmatrix}$$

T_{END} will exist and be stable with conditions $\theta > \frac{\mu_J(\alpha + \mu_J)}{\alpha}$. Fulfillment of the value of the critical point stability requirements T_{DEF} will cause a tipping point T_{END} becomes non-existent and unstable.

3. The simulation results show that subpopulations of pathogenic fungal spores greatly influence disease-free and endemic conditions. Controlling the growth rate of Oncobasidium theobromae (θ) spores can be done to maintain disease-free conditions.

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