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Available online at http://scik.org Commun. Math. Biol. Neurosci. 2021, 2021:3 https://doi.org/10.28919/cmbn/5216 ISSN: 2052-2541 THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES: MODELING AND ANALYSIS PUJI ANDAYANI*, LISA RISFANA SARI Universitas Internasional Semen Indonesia, Gresik City 61122, Indonesia Copyright © 2021 the author(s). This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. Abstract.

Guillain-Barre Syndrome (GBs) is an autoimmune disease that interchangeability of functions immune cells so the immune cells do not work properly. In people with GBs, immune cells destroy healthy cells, thereby reducing the growth rate of healthy cells. One of the causes of GBs is Zika virus infections. GBs in Indonesia has been around since 1859, but this incident is still rarely identi?ed because the symptoms of leg pain and rheumatism have been complained about by many people with various causes. The epidemiological mathematical model for GBs has been modi?ed, with a focus on cell-to-cell interactions, to study the behavior of the GBs transmission by involving healthy cells, infected cells, and immune cells.

The mathematical model has considered the role of immune cells in every healthy cell interaction so it can inhibit the interaction of infected cells with healthy cells. The model created is a system of non-linear differential equations with saturated incidence rates. The mathematical model obtained will be analyzed using a dynamical analysis. The stability analysis around the equilibrium point is studied by analyzing the eigenvalues of the Jacobian matrix at the equilibrium point. In the end, the numerical simulation is analyzed to ensure the analytical result.

Then the conclusion from the analysis results is described as a solution to the problem. Keywords: autoimmune; existence; Guillain-Barre syndrome (GBs); stability; Zika. 2010 AMS Subject Classi?cation: 37C25, 37N25, 39A30. *Corresponding author E-mail address: puji.andayani@uisi.ac.id Received November 16, 2020 1 2 PUJI ANDAYANI, LISA RISFANA SARI 1. INTRODUCTION Guillain-Barre syndrome (GB's) is a neurological disorder called acute poly Neurotherapy one of which is caused by Zika virus infection [1].

The Zika virus (ZIKV) emerging and trans- mitted by the Aedes mosquito is currently a challenge for health services in countries experi- encing outbreaks. ZIKV infection is mild, but in some cases, it may be mild has progressed to neurological disease such as microcephaly in infants and Guillain-Barr´ e (GBS) in adult's syn- drome. GBS is an autoimmune disorder that affects peripheral nerves. ZIKV ?rst appeared in South America for several years [1] [2]. The mildest effects of GBs varied considerably, while the most severe effects were maximum within 4 (four) weeks.

GB's disease has a variety of cases including acute in?ammatory demyelinating polyneuropathy (AIDP) which is common in the Western world. Other types of GBs are acute motor axonal neuropathy (AMAN) which often occurs in Asia and Japan, the Miller-Fisher syndrome (MFS) type, and overlap syndrome between GBs-MFs. Approximately 10% of GBs sufferers experience secondary nerve damage in the ?rst 8 (eight) weeks [2].

Based on the results of the study [2][3], about 5% of patients who were initially diagnosed with GBs, were suffering from chronic in?ammatory demyelinat- ing polyradiculoneuropathy (CIDP). Now, GB's is still one type of severe disease, even though there are treatments for GBs. Ap- proximately 25% of people with GBs need arti?cial ventilation assistance, 20% of patients are unable to walk for 6 months, and 3-10 patients die. The usual effects of GBs include pain, fatigue, or residual complaints with a period of months or years [2]. Based on data from the ministry of health [3], GBs has existed in Indonesia since 1859.

The name Guillain-Barre is taken from two French scientists who suffered from paralysis and then recovered when he re- ceived medical treatment. GB's is considered a rare disease because it only affects 1 in 100,000 people each year. The epidemiological mathematical model of Guillain-Barre Syndrome was proposed in some research. In 2007 Iwami et. al.[4] studied the GBs model to investigate three stages of the disease namely tolerance, ?are-up, and dormancy state. Iwami et. al.[5] developed the research by observing the effect of molecular mimicry on disease conditions in 2009. Another

research on GBS was conducted by Elettreby et. al. in 2019[6] and Sari et. al.[7] in 2020. Elettreby et. al.

THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES 3 observed changes in model dynamics with four different types of functional immune response. Lisa et al. examine a model by considering cross-reactive immunity and the Holling type II functional immune response. The model building assumption in this study resulted in different observations of dynamic behavior. However, in general, the molecular immune response is the main point in the study of GBs. This becomes more interesting when it is linked to the Zika virus as one of the causes of autoimmune conditions.

In this case, Kumar and Delogu's [8] study describes the immune response mechanism that results in autoimmune conditions. In the case of disease epidemiology, there are several interpretation results obtained when the assumptions are different. In this study, the authors chose the problem formulation of how the dynamic behavior of the epidemiology of GBs, when analyzed analytically and numerically, assumed the rate of occurrence was saturated. This study was conducted to develop an epi- demiological model of Guillain-Barre Syndrome with a saturated incidence rate. So that it can enrich the epidemiological model, especially the epidemiological model of GBs.

A modi?ed model for Guillain-Barre Syndrome are studied by identify the interaction between between healthy cell healthy cell (H), infected cell (F), and immune cell (C) [3] [4]. We assume the birth rate of a healthy cell is dynamically growing with parameter a, the interac- tion between healthy cells and infected cells rates with parameter ß1, and interactions between healthy cells and immune cells with parameter ß2. The parameter ? is the rate of immune cell interaction when facing the presence of healthy cells, with parameter v as a speed inhibiting parameter due to the role of immune cells in the systems.

Meanwhile, the death rate of the cell population is shown respectively by the parameters µ1, mu2 and mu3 represents the rate of dam- age rate of healthy cells, the damage rate to infected cells, and the rate of damage to immune cells. Then a modi?ed model of Guillain-Barre Syndrome is shows in the following system. (1) d H (t) d t = a H (t)- ß1 H (t) F (t) 1 + vC (t) - ß2 H (t) C (t) 1 + vC (t) - µ1 H (t); d F (t) d t = ß1 H (t) F (t) 1 + vC (t) - ? F (t) C (t)- µ2 F (t); d C (t) d t = ß2 H (t) C (t) + ? F (t) C (t)- µ3 C (t) . Model (1) is a nonlinear differential equation that does not have an explicit time-dependent solution. Hence, we study these models over a long period of time.

By changing the right-hand 4 PUJI ANDAYANI, LISA RISFANA SARI side with zero, then the equilibrium point of model (1) are at the following. E P 0 = (0 ,0 ,0); E P 1 = _ µ2 ß1 ,

?1 ß1 ,0 _ ; E P 2 = _ µ3 ß2 ,0 , ?1 ß1 + v ?1 _ ;(2) E P 3 = _ 0 , µ3 ? ,- µ2 ? _ ; and E P 4 = (F 4 , H4 , C4) . (3) Where, ?1 = a- µ1, ?2 = 1 + vC4; F 4 = µ3 ?1 ?2 (µ2 ß2 + ? ?1) ?2 + ? ?1 ; and H4 = µ3 (µ2 + ? C4) ?2 (µ2 ß2 + ? ?1) ?2 + ? ß2 vC42 . 2.

MATHEMATICAL ANALYSIS In biological problems, the positivity of the system needs to be analyzed to ensure that the system solution is positive. The following lemma proved that system 1 has positive solutions at in?nite times. Lemma 2.1. Suppose the model 1 have positive initial values H (0)= 0 , F (0)= 0 , C (0)= 0 , then the solution (H (t) , F (t) , C (t)) are positives for all time t= 0 . Proof. To prove the positive solution, it is suf?cient to show all trajectories of system 1 are nonnegative for time t > 0. The ?rst equation of system (1), which is health cells over time t is given by the following inequality.

(4) d H (t) d t =- _ ß1 F (t) 1 + vC (t) + ß2 C (t) 1 + vC (t) + µ1 _ H (t); By solving the inequality, and taking limit tß 8, we have (5) H (t)= H0 ex p _ - _ µ1 t + Z ß1 F (t) + ß2 C (t) + µ1 vC (t) 1 + vC (t) _ d t _ (6) liminf t? 8 H (t)= 0 . Similarly, in the same way, the positivity of equation F (t) and C (t) when t goes to in?nity are proved. _ 2.1. Existence of Equilibrium. The existence of an equilibrium point needs to be analyzed to ensure that the observed equilibrium point is in the invariant area.

The conditions for the existence of the equilibrium point model 1 can be shown in the following table: THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES 5 TABLE 1. Existence and Local Stability Condition of the Equilibria Eq. Point Existence Conditions E P 0 Always Exists - E P 1 Exist ?1 > 0 E P 2 Exist ?1 > 0 E P 3 Not Exist - E P 4 - C4= 0 To show the conditions for the existence of point E P 4, it is necessary to analyze the positivity of C4 as follows. C4 is positive root of (7) P (C4) = ? ß2 vC42- ß1 C4- ? µ3 (?1 ?2- ß2 C4)(µ2 + ? C4)(µ2 ß2 + ? ?1) ?2 .

The roots of polynomials 7 are C4 = - ?1 v ?1- ß2 < 0; or C4 =- µ2 ? < 0; or the roots of Pol i (C4) = a C42 + bC4 + c = 0 , To see the positive root of Pol i (C4) we need to identify the value of- b / a and c / a at the following. C41 + C42 =- ? ?1 + µ2 ß2 ? ß2 < 0 and C41 . C42 =- ? ?1 + µ2 ß2- µ3 ß1 v ? ß2 . Then, the polynomials P (C4) have one positive root. 2.2. Local Stability. The stability of each point identi?ed by analyzing each equilibrium in the following Jacobian matrix: [5] [6] (8) J (H , F , C) = ? ? ? ? ? ? ? (a- µ1)- ß1 F 1 + vC - ß1 H 1 + vC (ß1 F + ß2 C) vH (1 + vC)2 - ß2 H 1 + vC ß1 F 1 + vC ß1 H 1 + vC- ? C- µ2 - ß1 vH F (1 + vC)2- ? F ß2 C ? C ß2 H- ? F- µ3 ? ? ? ? ? ? ? Furthermore, the stability of each equilibrium point is analyzed by computing the eigenvalues of each Jacobi matrix at each equilibrium point.

The local stability at each equilibrium point is described by proving the following

Lemmas. Lemma 2.2. The equilibrium E P 0 is locally asymptotically stable when a < µ1 . 6 PUJI ANDAYANI, LISA RISFANA SARI Proof. The stability of E P 0 are analyzed by identi?ed the Jacobian of E P 0. (9) J (E P 0) = ? ? ? ? ? a- µ1 0 0 0 - µ2 0 0 0 - µ3 ? ? ? ? ? The eigen value of J (E P 0) are a- µ1 ,- µ2 ,- µ3, then equilibrium E P 0 is stable when a= µ1. _ Lemma 2.3. The equilibrium E P 1 is locally stable (center) when a = µ1 . Proof.

The Jacobian matrix of E P 1 is (10) J (E P 1) = ? ? ? ? ? ? ? ? 0 - µ2 - µ2 (ß2 + v (a- µ1)) ß1 a- µ1 0 - (a- µ1)(µ2 v + ?) ß1 0 0 µ2 ß2 + ? (a- µ1) ß1 - µ3 ? ? ? ? ? ? ? ? Then the eigen values of J (E P 1) are ?1 =- µ3 ß1- µ2 ß2- ? (a- µ1) ß1 , ?2 = (µ2 ?1) i, ?3 =- (µ2 ?1) i. According to dynamical analysis theory [6], the equilibrium point E P 1 is stable if a = µ1. _ Lemma 2.4. The equilibrium E P 2 is locally stable (center) if 0= (a- µ1)= ß2 v and ß2 µ2- µ3 ß1= 0 . Proof. The Jacobian matrix of E P 2 is J (E P 2).

The eigen values of J (E P 2) are ?1 = 1 ß 2 2 (ß2- v (a- µ1)) ((a- µ1)(- µ3 ß1 v (2 ß2- v (a- µ1)))- ß 2 2 (? + µ2 v)- ß 2 2 (µ2 ß2- µ3 ß1))- 2 v a µ3 ß1 ß2 < 0 , ?2 =- µ3 (a- µ1)(ß2- v (a- µ1)) i , ?3 = µ3 (a- µ1)(ß2- v (a- µ1)) i. Hence, the E P 2 is locally stable if 0= (a- µ1)= ß2 v and ß2 µ2- µ3 ß1= 0 .. _ The stability of E P 3 are doesn't identi?ed, because the equilibrum E P 3 is doesn't exist. Then the stability of E P 4 are describe at the following. THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES 7 Lemma 2.5. The equilibrium E P 4 is locally asymptotically stable if ß2 µ2- µ3 ß1 < 0 and a- µ1 > 0 . Proof.

The stability of E P 4 identi?ed by analyze the characteristic polynomial of Jacobian matrix J (E P 4) as follow (11) P (E P 4) = k3 ? 3 + k2 ? 2 + k1 ? + k0 . With, (12) ?3 = ?2 (ß2 µ2 + ? (a- µ1)) + ? ß2 vC2 4; ?4 = µ3 ß1 + µ2 ß2; and ?5 = µ2 + ? C4 . Where, k3 = ?2 ?2 3; k2 = ?3 (?6 (?2 ?4 + ß2 vC2 4) + ?1 ?2 2 ? (?6- ?1)- ?1 ?2 ?4 + ß ? C4 (1- v2 C2 4) + µ2 v (1 + ? C4)2 + ß2 C4 (1 + µ2 v (1 + 2 ? C4) + v ?2 C2 4)); and k0 =- µ3 C (?2 1 ?2 ? (?2 ? (?1 ? + µ2 ß2) + µ3 ß1 (µ2 v- ?) + ?3 ß2 C4 (v3 C3 4- 3 vC4- 2)) + ?1 ?2 (µ3 ß1 ß2 (?2 C4 ?4 + µ2 v ?5) + ?1 ?5 ? v2 C2 4) + ?1 ?2 (ß2 ? (?4 C4- ß2 µ2 2- 5 ß2 ? µ2 C4 - 2 µ2 ß2 C2 4 ? v ?6)) + ß 2 2 (µ2 ? C4 (2 µ3 ß1- a µ2 ß2) + µ2 ?4 (µ2- vC4)- v ? C4 (3 µ2- 2 ? C2 4))).

By analyzing the positivity of each coef?cient on the polynomial P (E P 4), and involving the Routh Hurwitz criterion [7], it can be concluded that the equilibrium point E P 4 is locally stable when ß2 µ2- µ3 ß1 , a- µ1 > 0, because it satisfy the condition k0 > 0; k3 > 0; and k1 k2 > k3 k0. _ 3. NUMERICAL RESULT AND DISCUSSION The results of the mathematical analysis in the previous chapter need to de?ne numerically involving some parameters. We can relate these parameters to real conditions. For examples, a death rate of cell problems, the death rate of healthy cells is caused by many things such as necrosis, which can be activated by components of the immune system.

In the problem of this mathematical model of GB′ s disease, we divide the conditions of distribution into several state conditions, according to the Lemma in the mathematical analysis. In the ?rst case we assume the condition a- µ1= 0, the stable condition for E P 0 by take some parameter value as follows. Case 1 : a = 0 .5; ß1 = 0 .1; ß2 = 0 .1; v = 0 .003; ? = 0 .5; µ1 = 0 .6; µ2 = 0 .01; µ3 = 0 .1. 8 PUJI ANDAYANI, LISA RISFANA SARI (A) Case a- µ1 < 0 (B) Case a- µ1 = 0 FIGURE 1. Numerical simulation under condition a- µ1= 0 In Figure 1 we can see two kinds of condition, when a- µ1 < 0 and a- µ1 = 0.

In the ?rst simulation, 4 starting points were selected to inform us the trajectory orbit in several initial value. We choose initial value 1 is (2 ,2 ,2) to assume the initial condition before cell inter- actions, means the probability of healthy cell, infected cell and immune cells is balance. The second initial value I v2 : (4 ,0 ,2) is describe the cell interaction if the initial infected cell with- out infected cells clearly shows that the cell population is tends to E P 0 = (0 ,0 ,0).

Meanwhile, the third initial value describes cell interactions if there are no healthy cells as the initial value, so that the interaction only occurs between infected cells and immune cells, which interact and tends to E P 0. In all conditions, it can be seen if the system trajectory will go to point E P 0. Thus, it can be said that the conditions that have been mentioned cause the system to stabilize to point E P 0. Then for case 2 we take some parameter value as follows a = 0 .8; ß1 = 0 .1; ß2 = 0 .1; v = 0 .003; ? = 0 .5; µ1 = 0 .01; µ2 = 0 .01; µ2 = 0 .1. THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES 9 FIGURE 2.

Numerical simulation under condition a- µ1 > 0 In second case, we try to modify the parameter with the condition a- µ1 = 0 .79. By taking some initial values which close to E P 0 and E P 1, it can be seen that the trajectory is away from point E P 0 = (0 ,1;7 ,9;0), forming a trapping areas in the F (t) and C (t) planes. For two initial value which are initial value 3 : (0 ,0 ,10) and initial value 4: (0 ,6 ,0) for t = 1000, then the trajectory is tends to E P 0 = (0;0;0). Figure 3 shows the numerical simulation results in case 3.

By choosing the following parame- ter values which are a = 0 .6; ß1 = 0 .8; ß2 = 0 .8; v = 0 .001; ? = 0 .5; µ1 = 0 .2; µ2 = 0 .4; µ3 = 0 .1, we can see the behavior of the system around the equilibrium points E P 0 and E P 2. Based on the simulation in Figure 3 we can see that when the initial value I v2 = (1 ,1 ,1), the trajectory will ?uctuate around the equilibrium point of E P 2. When we select the initial value in the H- C plane is I v3 = (2 ,0 ,1), the trajectory forms a repeating graph, circling around the equilibrium point E P 2, but not leading to the equilibrium point.

This condition is in accordance with the analytical calculation, where the Jacobian eigenvalues are both complexes. Biologically, this condition states that in conditions there are only healthy cell populations and immune cells are not fully achieved, only close. 10 PUJI ANDAYANI, LISA RISFANA SARI FIGURE 3. Numerical simulation under condition 0= (a- µ1)= ß2 v and ß2= µ3 ß1 µ2 In the next case, the orbital behavior will be shown when the parameter values meet the stability requirements of the endemic equilibrium point.

Analytically, the endemic conditions can be ful?lled under certain conditions, then we will show the numerical orbital behavior. By selecting the parameter values a = 1; ß1 = 0 .5; ß2 = 0 .2; v = 0 .001; ? = 0 .5; µ1 = 0 .3; µ2 = 0 .01; µ3 = 0 and the value of C4 = 6 we have the following. (A) (B) FIGURE 4. Numerical simulation under condition in Lemma 2.5 THE CELL-TO-CELL TRANSMISSION OF GUILLAIN-BARRE SYNDROMES 11 In Figure 4a we can see the orbital behavior of model 1 with the initial value (10 ,2 ,6).

The orbital behavior will be stable center around the bounder equilibrium point in the H- C plane, the F- H plane, and the F- C plane. By changing the change from the initial value to (2 ,0 ,6) at the same parameter value, we will obtain Figure 4(b). System 1 will approach the equilibrium point in the H- C plane but not asymptotic, it means that under these conditions, the extinct of infected cells cannot happen. The satis?ed conditions for the stability of endemic equilibrium occur and are found analytically. Numerically, the persistence condition for all cell populations is rare to ?nd. It happened only at some initial values. 4.

CONCLUSION Guillain-Barre Syndrome (GBs) is an immune disorder that is quite dangerous because it can have an impact on death. There is no de?nite cause of these GBs, but one of them GBs is the impact of the Zika virus which attacks adult humans other than pregnant women. A mathematical model that studies cell-to-cell interactions on the spread of GBs has been constructed in the system (1) involving a population of healthy cells, infected cells, and immune cells.

The model formed is a non-linear differential equation with a saturated occurrence rate. The invariant condition has been proven analytically by ?nding the boundary of the system. The conditions of existence at each equilibrium point are studied in order to show what conditions cause the equilibrium point to be studied. In this case, the immune response is assumed to be nonlinear so that the baseline reproduction rate is not found. In model (1), the trivial equilibrium point is not always stable. The trivial equilibrium point will be locally stable if the rate of birth of healthy cells is less than the rate of damage to healthy cells.

Other conditions change when the opposite symptoms occur, namely when the birth rate of healthy cells is greater than the death rate. There will be several conditions in these conditions followed by changes in other parameters. Meanwhile, the birth rate which is equal to the death rate will cause a stable condition around the equilibrium point in the presence of healthy and infected cells. The stability condition of the third equilibrium point (the point of equilibrium without in- fected cells) is obtained analytically and numerically on the condition of certain parameter values.

This condition is never precisely acquired or not asymptotically stable, but the orbital 12 PUJI ANDAYANI, LISA RISFANA SARI behavior conditions approach the point of stability without infected cells. In the opposite con- dition, when the growth rate of the cell is greater than the damage rate, followed by other conditions, then analytically and numerically endemic or persistent conditions of all cell popu- lations can be achieved. However, endemic precise conditions are quite rare, because it depends on their initial values. This behavior means that the initial conditions play a role in the fate of the orbital behavior of the system.

In other words, eradicating infected cells depends on the initial conditions. Our results suggest that early detection of infection provides a greater chance of cure. ACKNOWLEDGEMENT The author would like to thank DRPM RISTEKBRIN for the ?nancial support of the research. Our gratitude also goes to LPPM UISI for its technical and administrative support. CONFLICT OF INTERESTS The author(s) declare that there is no con?ict of interests. REFERENCES [1] L. Barbi, A. V. Coelho, L. C. Alencar and S.

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