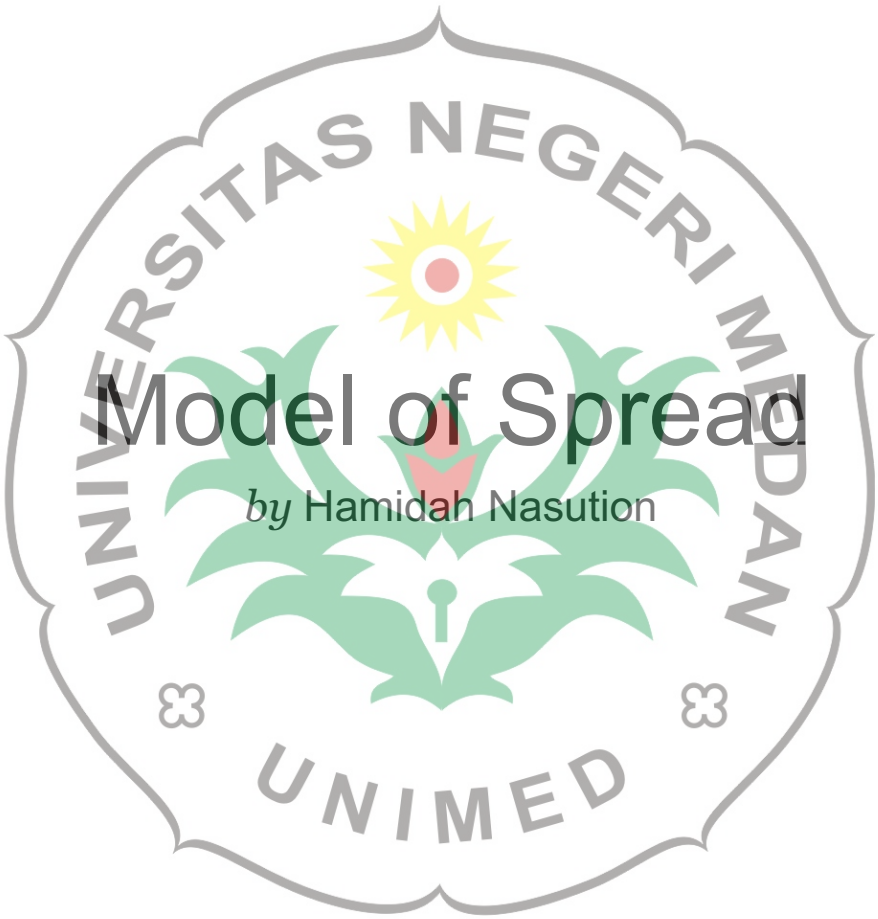

Model of Spread

by Hamidah Nasution

Submission date: 04-Jun-2020 10:14PM (UTC+0700)
Submission ID: 1337723571
File name: MODEL_OF_SPREAD_OF_INFECTIOUS_DISEASES.docx (112.26K)
Word count: 3629
Character count: 18781

MODEL OF SPREAD OF INFECTIOUS DISEASES

*Hamidah Nasution[1], Ismail Husein[2], Herlina Jusuf[3], Muqdad Irhaeem Kadhim[4], Auhtor5

[1]Department of Mathematics, Universitas Negeri Medan, North Sumatera, Indonesia
[2]Department of Mathematics, Universitas Islam Negeri Sumatera Utara, Medan, Indonesia
[3]Department of Public Health, Faculty of Sports and Health, Universitas Negeri Gorontalo
[4]College of Science, University of Al-Qadisiyah, Iraq

husein_ismail@uinsu.ac.id

Abstract. The Model of infectious diseases continues to develop along with the development of the disease. With the dynamic spread of disease, ongoing research is needed. This study developed the SIR model by taking into account the spread of disease in the presence of Reproductive Number or R0. This study proposes an epidemic model of infectious diseases in dynamic networks for SIRS types, the standard mean-field model is used as a basic framework.

Key words: Model of Spread, Infectious Disease, Modeling

1. Introduction

Indonesia as a developing country, health issues are still important to get serious attention. In particular, the problem of infectious diseases because in Indonesia alone infectious diseases that occur until the end of 2017 and are still a hot topic of discussion are diphtheria. Although it sounds like a common cold or fever, diphtheria in fact has a high mortality rate and can be transmitted quickly. Until now, the vaccination program is still believed to be the most effective way in suppressing the spread of diphtheria. One branch of modern mathematics which is important and has a wide scope of research areas is differential equations.

Differential equations are branches of mathematics that are quite strategic because they relate to the central parts of Algebra, Analysis, Geometry, and others that will play a major role in the introduction of concepts and problem solving relating to the real world (Waluya, 2006).

This study aims to build a model of the spread of infectious diseases in dynamic SIRS type networks for heterogeneous populations. The model will be built using the basic framework of a mathematical model to investigate a parameter known as a basic reproductive number in detail, especially if the basic assumption of the model, mixing homogeneous populations, does not apply. In the SIRS model, this parameter has a very important role as a notification of a disease outbreak. The model that will be investigated in this study is based on a standard mean-field model. The main parameters that serve as measures for controlling epidemics, known as basic reproductive numbers with the mean-field model, will be investigated in more detail in the context of developing the model. The mean-field

modification model produced essentially contains implicitly some important effects of heterogeneous mixing in contact tissue in the epidemic for vaccine allocation.

Method

SI epidemic model

The simplest mathematical model in epidemiology is known as the Ross Epidemic Model or SI, which was developed in 1911. In the SI model, the population is divided into two parts (subgroups), namely susceptible = S populations against disease transmission and infectious populations = I) against a disease. The assumptions used in this model are: that the vulnerable population remains in close contact with the infected population all the time $t \geq 0$, the number of populations is constant as N with $N = (S(t) + I(t))$, where S and I are mutually exclusive and mixing the population homogeneously so that each individual has an equal chance of infection. If $\beta \geq 0$ is the average constant (the proportion) of subgroup contact that results in a new infection the unity of time from the original state is susceptible (or also called the transmission rate constant).

SIS epidemic model

The assumptions used in this model are: that the vulnerable population remains in close contact with the infected population throughout the time ≥ 0, the number of populations is constant as N with $N = (S(t) + I(t))$, where S and I are mutually exclusive and homogeneous mixing of the population so that each individual has an equal chance of infection. However, the number or size of the infected population can decrease as the movement of infected individuals changes status to be susceptible to reuniting time with proportions σ.

SIR epidemic model

The SIR model is the basis for most of the deterministic models that are still used today. This model was first developed by Kermack and McKendrik in 1927. The SIR model has the same structure and assumptions as the SI model, the extension is that in the SIR model it is possible for the infected population / community members to recover and the total population of N to be divided into three subgroups mutually exclusive; susceptible subgroups (Susceptibles) symbolized S (t), infectious / infected subgroups I (t) and moved (Removed) subgroups symbolized R (t). R (t) represents individuals who died of illness, recovered from infection and now have permanent immunity or individuals who have been exiled from the rest of the population. So in this last subgroup, it no longer contributes to the spread of disease / epidemic. However, it is still maintained as a member of a total population of N, although there is a possibility that some of them have died.

In this model I also assume that individuals who enter R (t) cannot be re-infected. Assuming that α is a constant proportion of the condition of the infected individual subsequently is removed unity of time. Then the differential equation model that represents the rate of change of the population that is susceptible to constant unity of time as in the SI model, as in equation (3). This is because there is no direct

transfer of individuals from subgroups vulnerable to moving subgroups. However, the differential equation model of the infected subgroup needs to be modified to take into account the number of infected people and recover.

Result

Model Epidemi SI

The simplest mathematical model in epidemiology is known as the Ross'Epidemic Model or SI, which was developed in 1911. In the SI model, the population is divided into two parts (subgroups), namely susceptible (S) populations to disease transmission and infected populations (infected) infectious = I) to a disease. In Figure 1 this model is the same as SIR but without the R compartment.

The assumptions used in this model are: that the vulnerable population remains in close contact with the infected population all the time $t \geq 0$, the number of populations is constant as N with N = (S (t) + I (t), where S and I are mutually exclusive and mixing the population homogeneously so that each individual has an equal chance of infection.

If $\beta \geq 0$ is the average constant (the proportion) of subgroup contact that results in a new infection the time unity from the original state that vulnerable (or also called the transmission rate constant). Furthermore, by using the law of Mass action, the SI Model can be described as:

$$\frac{d\,S(t)}{dt} = -\beta S(t) I(t) \quad (1)$$

and

$$\frac{dI(t)}{dt} = \beta S(t) I(t) \quad (2)$$

hereafter written:

$$\frac{d\,S}{dt} = -\beta S\, I \quad (3)$$

$$\frac{dI}{dt} = \beta S\, I \quad (4)$$

with initial conditions $S(0) = S_0$ and $I(0) = I_0$.

In the SI model it can be said that the rate of change of contracting is positive, so the number of infected individuals will continue to increase until S (t) = 0.

The completion of this SI model, by changing equation (4) to:

$$\frac{dI}{dt} = \beta(N - I)I$$

Furthermore, with the separation of variables and integrated with a limit from 0 to t as follows: $\int_{I(0)}^{I(t)} \frac{1}{I(N-I)} dI = \int_0^t \beta dt$

was obtained: $I(t) = \frac{I(0)N}{I(0) + (N - I(0))e^{-\beta N t}}$

or written : $I(t) = \frac{I_0 N}{I_0 + (N - I_0)e^{-\beta N t}}$

observe that I (t) increases with increasing t and for $t \to \infty$, $e^{-\beta N t} \to 0$

so that $I(t) \to \frac{I_0 N}{I_0} = N$

This last model states that as time goes by, the number of infected populations will increase, eventually all populations will become infected.

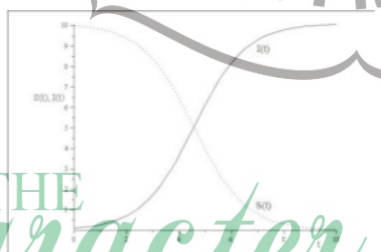

Figure 1: Model SI with β = 0,1 and initial value S(0) =10, I(0) = 0,1

Classic Model (SIR Model)

The SIR model is the basis for most of the deterministic models that are still used today. This model was first developed by Kermack and McKendrik in 1927. The SIR model has the same structure and assumptions as the SI model, the extension is that in the SIR model is possible for the infected population / community members to recover and the total population of N to be divided into three subgroups mutually exclusive; susceptible subgroups (Susceptibles) are

symbolized S (t), infectious / infected subgroups are symbolized I (t) and recovered subgroups are symbolized R (t). R (t) represents individuals who died of illness, recovered from infection and now have permanent immunity or individuals who have been exiled from the rest of the population. So in this last subgroup, they no longer contribute to the spread of disease / epidemic. However, it is still maintained as a member of a total population of N even though there is a possibility that some of them have died. In this model it is also assumed that individuals who enter R (t) cannot be re-infected. Assuming that α is a constant proportion of the condition of the infected individual subsequently is removed unity of time.

Then the differential equation model that represents the rate of change of the population that is susceptible to constant unity of time as in the SI model, as in equation (3). This is because there is no direct transfer of individuals from subgroups vulnerable to moving subgroups. However, the differential equation model of the infected subgroup needs to be modified to take into account the number of individuals infected and recovered. When the amount moved is proportional to the amount that is infected with each unit of time, then the differential equation model becomes:

$$dI/dt = \beta SI - \alpha I$$

While the rate of change in the number of removals per unit time is:

$$\frac{dR}{dt} = \alpha I$$

with initial conditions: R (0) = R0, so that the complete differential equation model which is the SIR model is:

$$\frac{DS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \alpha I \quad (5)$$

$$\frac{dR}{dt} = \alpha I \,,$$

with initial conditions: $S(0) = S_0$, $I(0) = I_0$, $R(0) = R_0$ dan $S(t) + I(t) + R(t) = N$.

The SIR model above has two parameters α and β which are determined from the results of the analysis of the observed data. The average αI cure is related to the exponential waiting time "waiting time" $e^{\alpha I}$ and $\frac{1}{\alpha}$ = average period of contracting.

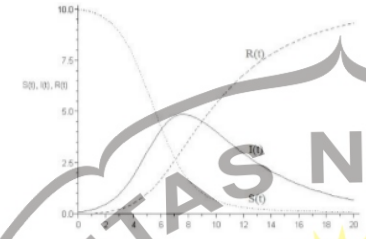

Figure 2: SIR model with $\gamma = 0{,}2$ $\beta = 0{,}1$ dan nilai awal

$S(0) = 10$, $I(0) = 0{,}1$ and $R(0) = 0$

SIRS Model

Not all diseases result in permanent immunity or death. Some diseases have a healing period and after time the recovered individual can be re-infected. Mathematically this means that a proportion of the subgroups that move the union of time ($\lambda \geq 0$) are again vulnerable. So the SIR model is modified to meodel SIRS as follows:

$$\frac{DS}{dt} = -\beta SI + \lambda R$$

$$\frac{dI}{dt} = \beta SI - \alpha I \quad (6)$$

$$\frac{dR}{dt} = \alpha I - \lambda R$$

with initial conditions: $S(0) = S_0$, $I(0) = I_0$, $R(0) = R_0$ dan $S(t) + I(t) + R(t) = N$.

Figure 3: SIRS model with $\alpha = 0{,}2$, $\beta = 0{,}1$, $\lambda = 0{,}2$ and initial value $S(0) = 10$, $I(0) = 0{,}1$ and $R(0) = 0$

CONSTRUCTION R0

Basic Reproduction Number (R0)

R0 which is usually called the Basic Reproduction Number is the average number of secondary infections produced when an infected individual is entered into the host population where each individual is in a susceptible condition. In most deterministic models, an infection begins fully if and only if R0>1, and otherwise if R0<1 then the number of infections will decrease and eventually become extinct. So the basic reproduction number is often seen as a threshold quantity that determines when an infection can attack and survive in a new host population.

If it is assumed that all pairs of individuals have contact at the same time so as to produce a new infected individual ie □,

The average rate of infected individuals has contact with susceptible individuals and then susceptible individuals become infected with time unity ie α, $\alpha \geq 0$.

R0 construction in the SIRS model, i.e.:

$$\frac{dI}{dt} = \beta SI - \alpha I$$

Growth of infection will take place if $\beta SI - \alpha I > 0$ or $\beta SI > \alpha I$

$\beta S > \alpha$ with S(0) = N so (βN/ α) > 1. Then thus $R_0 = \beta N/\alpha$

Logistics Equations in Epidemiology

Logistics equations are most often discussed when we study population dynamics with densities dependent on birth and death.

5.1 Conclusion

This study proposes an epidemic model of infectious diseases in dynamic networks for SIRS types, the standard mean-field model is used as a basic framework. In this SIRS epidemic model, a very basic parameter in discussing a disease epidemic is R0 (basic reproductive number). R0 has the main role as a threshold of an outbreak, on the relevance of testing control measures.

THE Character Building UNIVERSITY

References

[1] Albert, R., Jeong, H. & Baraba´si, A.-L. 1999 Diameter of the world-wide web. Nature 401, 130-131.

[2] Albert, R., Jeong, H. & Baraba´si, A.-L. 2000 Error and attack tolerance of complex networks. Nature 406, 378-381.

[3] Bailey, N. T. J. 1957 The mathematical theory of epidemics. London: Griffin.

[4] Bak, P., Chen, K. & Tang, C. 1990 A forest-fire model and some thoughts on turbulence. Phys. Lett. A 147, 297-300.

[5] Baraba´si, A. L. & Albert, R. 1999 Emergence of scaling in random networks. Science 286, 509-512.

[6] Barbour, A. & Mollison, D. 1990 Epidemics and random graphs. In Stochastic processes in epidemic theory (ed. J.-P. Gabriel, C. Lefe`vre & P. Picard), pp. 86-89. New York: Springer.

[7] Bearman, P. S., Moody, J. & Stovel, K. 2004 Chains of affection: the structure of adolescent romantic and sexual networks. Am. J. Sociol. 110, 44-91.

[8] Bolloba´s, B. 1979 Graph theory. New York: Springer. Bolloba´s, B. 1985 Random graphs. London: Academic Press. Boots, M. & Sasaki, A. 1999 'Small worlds' and the evolution of virulence: infection occurs locally and at a distance. Proc. R. Soc. B 266, 1933-1938. (doi:10.1098/rspb.1999. 0869.)

[9] Diekmann, O., Heesterbeek, J. A. P. & Metz, J. A. J. 1998 A deterministic epidemic model taking account of repeated contacts between the same individuals. J. Appl. Prob. 35, 462-468.

[10] Dietz, K. & Hadeler, K. P. 1988 Epidemiological models for sexually transmitted diseases. J. Math. Biol. 26, 1-25.

[11] Doherty, I. A., Padian, N. S., Marlow, C. & Aral, S. O. 2005 Determinants and consequences of sexual networks as they affect the spread of sexually transmitted infections. J. Infect. Dis. 191, S42-S54.

[12] Eames, K. T. D. & Keeling, M. J. 2002 Modeling dynamic and network

[13] heterogeneities in the spread of sexually transmitted diseases. Proc. Natl Acad. Sci. USA 99, 13330-13335.

[14] Eames, K. T. D. & Keeling, M. J. 2003 Contact tracing and disease control. Proc.

[15] R. Soc. B 270, 2565-2571. (doi:10.1098/rspb.2003.2554.)

[16] Eames, K. T. D. & Keeling, M. J. 2004 Monogamous networks and the spread of sexually transmitted diseases. Math. Biosci. 189, 115-130.

[17] Eichner, M. 2003 Case isolation and contact tracing can prevent the spread of

[18] smallpox. Am. J. Epidemiol. 158, 118-128.

[19] Eubank, S., Guclu, H., Kumar, V. S. A., Marathe, M. V., Srinivasan, A., Toroczkai,

[20] Z. & Wang, N. 2004 Modelling disease outbreaks in realistic urban social networks. Nature 429, 180-184.

[21] Ferguson, N. M. & Garnett, G. P. 2000 More realistic models of sexually transmitted disease transmission dynamics: sexual partnership networks, pair models, and moment closure. Sex. Transm. Dis. 27, 600-609.

[22] Ferguson, N. M., Donnelly, C. A. & Anderson, R. M. 2001 The foot-and-mouth epidemic in Great Britain: pattern of spread and impact of interventions. Science 292, 1155-1160.

[23] Frank, O. & Strauss, D. 1986 Markov Graphs. J. Am. Stat. Soc. 81, 832-842.

[24] Fraser, C., Riley, S., Anderson, R. M. & Ferguson, N. M. 2004 Factors that make an infectious disease outbreak controllable. Proc. Natl Acad. Sci. USA 101, 6146-6151.
[25] Garnett, G. P. & Anderson, R. M. 1996 Sexually transmitted diseases and sexual behavior: insights from mathematical models. J. Infect. Dis. 174, S150-S161.
[26] Ghani, A. C. & Garnett, G. P. 1998 Measuring sexual partner networks for transmission of sexually transmitted diseases. J. R. Stat. Soc. A 161, 227-238.
[27] Ghani, A. C. & Garnett, G. P. 2000 Risks of acquiring and transmitting sexually transmitted diseases in sexual partner networks. Sex. Transm. Dis. 27, 579-587.
[28] Ghani, A. C., Swinton, J. & Garnett, G. P. 1997 The role of sexual partnership networks in the epidemiology of gonorrhea. Sex. Transm. Dis. 24, 45-56.
[29] Gilbert, M., Mitchell, A., Bourn, D., Mawdsley, J., Clifton-Hadley, R. & Wint, W. 2005 Cattle movements and bovine tuberculosis in Great Britain. Nature 435, 491-496.
[30] Grassberger, P. 1983 On the critical behaviour of the general epidemic process and dynamical percolation. Math. Biosci. 63, 157-172.
[31] Grenfell, B. T. 1992 Chance and chaos in measles dynamics. J. R. Stat. Soc. B 54, 383-398.
[32] Grenfell, B. T., Bjornstad, O. N. & Kappey, J. 2001 Travelling waves and spatial hierarchies in measles epidemics. Nature 414, 716-723.
[33] Grimmett, G. 1989 Percolation. Berlin: Springer.
[34] Halloran, M. E., Longini Jr. I. M., Nizam, A. & Yang, Y. 2002 Containing bioterrorist smallpox. Science 298, 1428-1432.
[35] Handcock, M. S. & Jones, J. H. 2004 Likelihood-based inference for stochastic models of sexual network formation. Theor. Popul. Biol. 65, 413-422. Harary, F. 1969 Graph theory. Reading, MA: Addison-Wesley.
[36] Harris, T. E. 1974 Contact interactions on a lattice. Ann. Probab. 2, 969-988. Haydon, D. T., Chase-Topping, M., Shaw, D. J., Matthews, L., Friar, J. K., Wilesmith, J. & Woolhouse, M. E. J. 2003 The construction and analysis of epidemic trees with reference to the 2001 UK foot-and-mouth outbreak. Proc. R. Soc. B 270, 121-127. (doi:10.1098/rspb.2002.2191.)
[37] Hethcote, H. W. & Yorke, J. A. 1984 Gonorrhea transmission dynamics and control. Springer Lecture Notes in Biomathematics. Berlin: Springer.
[38] Husein, Ismail H Mawengkang, S Suwilo "Modeling the Transmission of Infectious Disease in a Dynamic Network" Journal of Physics: Conference Series 1255 (1), 012052, 2019.
[39] Husein, Ismail, Herman Mawengkang, Saib Suwilo, and Mardiningsih. "Modelling Infectious Disease in Dynamic Networks Considering Vaccine." Systematic Reviews in Pharmacy 11.2, pp. 261-266, 2020.
[40] Husein, Ismail, YD Prasetyo, S Suwilo "Upper generalized exponents of two-colored primitive extremal ministrong digraphs"AIP Conference Proceedings 1635 (1), 430-439, 2014
[41] S Sitepu, H Mawengkang, I Husein "Optimization model for capacity management and bed scheduling for hospital" IOP Conference Series: Materials Science and Engineering 300 (1), 01,2016.
[42] Jeong, H., Tombar, B., Albert, R., Oltvai, Z. N. & Baraba´si, A.-L. 2000 The large-scale organization of metabolic networks. Nature 407, 651-654.
[43] Jolly, A. M. & Wylie, J. L. 2002 Gonorrhoea and Chlamydia core groups and sexual networks in Manitoba. Sex. Transm. Infect. 78, i45-i51.

[44] Karlberg, M. 1997 Testing transitivity in graphs. Soc. Networks 19, 325-343. Keeling, M. J. 1997 Modelling the persistence of measles. Trends Microbiol. 5, 513-518.
[45] Keeling, M. J. 1999 The effects of local spatial structure on epidemiological invasions. Proc. R. Soc. B 266, 859-867. (doi:10.1098/rspb.1999.0716.)
[46] Keeling, M. J. 2005 Implications of network structure for epidemic dynamics. Theor. Popul. Biol. 67, 1-8.
[47] Keeling, M. J., Rohani, P. & Grenfell, B. T. 2001 Seasonallyforced disease dynamics explored as switching between attractors. Physica D 148, 317-335.
[48] Kermack, W. O. & McKendrick, A. G. 1927 A contribution to the mathematical theory of epidemics. Proc. R. Soc. A 115, 700-721.
[49] Klovdahl, A. S. 1985 Social networks and the spread of infectious diseases: the AIDS example. Soc. Sci. Med. 21, 1203-1216.
[50] Klovdahl, A. S. 2001 Networks and pathogens. Sex. Transm. Dis. 28, 25-28.
[51] Klovdahl, A. S., Dhofier, Z., Oddy, G., O'Hara, J., Stoutjesdijk, S. & Whish, A.1977 Social networks in an urban area: first Canberra study Aust. N. Z. J. Sociol. 13, 169-172.
[52] Kretzschmar, M., van Duynhoven, Y. T. H. P. & Severijnen, A. J. 1996 Modelingprevention strategies for gonorrhea and chlamydia using stochastic network simulations. Am. J. Epidem. 144, 306-317.
[53] Kuperman, M. & Abramson, G. 2001 Small world effects in an epidemiological model. Phys. Rev. Lett. 86, 2909-2912.
[54] Leinhardt, S. (ed.) 1977 Social networks: a developing paradigm. New York; 2002 Thomas Parran Award Lecture. Sex. Transm. Dis. 30, 478-482.
[55] Rothenberg, R. B., Potterat, J. J., Woodhouse, D. E., Muth, S. Q., Darrow, W. W. & Klovdahl, A. S. 1998 Social network dynamics and HIV transmission. AIDS 12, 1529-1536.
[56] Rozenfeld, A. F., Cohen, R., ben-Avraham, D. & Havlin, S.2002 Scale-free networks on lattices. Phys. Rev. Lett. 89, 218701.
[57] Syah Rahmad, M K M Nasution, Ismail Husein, Marischa Elveny, "Optimization Tree Based Inference to Customer Behaviors in Dynamic Control System", International Journal of Advanced Science and Technology, pp. 1102 – 1109,2020.
[58] Husein Ismail, Rahmad Syah, "Model of Increasing Experiences Mathematics Learning with Group Method Project", International Journal of Advanced Science and Technology, pp. 1133-1138, 2020.
[59] Syah Rahmad, Mahyuddin K.M Nasution, Ismail Husein, "Dynamic Control Financial Supervision (OJK) for Growth Customer Behavior using KYC System", International Journal of Advanced Science and Technology, pp. 1110 – 1119, 2020.
[60] Schwartz, I. B. 1985 Multiple recurrent outbreaks and predictability in seasonally forced nonlinear epidemic models. J. Math.Biol.18, 233-253.
[61] Scott, J. 1991 Social network analysis: a handbook. London: SAGE Publications. Snijders, T. A. B. 2001 The statistical evaluation of social network dynamics. Sociol. Methodol. 31, 361-395.
[62] Szendro˝i, B. & Csa´nyi, G. 2004 Polynomial epidemics and clustering in contact networks. Proc. R. Soc. B 271, S364-S366. (doi:10.1098/rsbl.2004.0188.) Travers, J. & Milgram, S. 1969 An experimental study of the small world problem. Sociometry 32, 425-443.

[63] Wallinga, J., Edmunds, W. J. & Kretzschmar, M. 1999 Perspective: human contact patterns and the spread of airborne infectious diseases. Trends Microbiol. 7, 372-377.
[64] Warren, C. P., Sander, L. M.&Sokolov, I. M. 2002 Geography in a scale-free network model. Phys. Rev. E 66, 056105.
[65] Wasserman, S. & Faust, K. 1994 Social network analysis. Cambridge: Cambridge University Press.
[66] Watts, D. J. 1999 Small worlds: the dynamics of networks between order and randomness. Princeton: Princeton University

Model of Spread

ORIGINALITY REPORT

17%	9%	11%	8%
SIMILARITY INDEX	INTERNET SOURCES	PUBLICATIONS	STUDENT PAPERS

PRIMARY SOURCES

1	repository.usu.ac.id Internet Source	3%
2	www.ims.nus.edu.sg Internet Source	2%
3	www.tandfonline.com Internet Source	1%
4	www.biomedcentral.com Internet Source	1%
5	rsif.royalsocietypublishing.org Internet Source	1%
6	M. MORRIS. "Epidemiology and Social Networks:: Modeling Structured Diffusion", Sociological Methods & Research, 08/01/1993 Publication	1%
7	Submitted to Trinity Grammar School Student Paper	1%
8	"Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory", Springer Science and	1%

	Business Media LLC, 2002 Publication	
9	Hyman, J.M.. "Modeling the impact of random screening and contact tracing in reducing the spread of HIV", Mathematical Biosciences, 200301 Publication	1%
10	Submitted to UWC-Maastricht Student Paper	1%
11	Tailei Zhang, Zhidong Teng, Shujing Gao. "Threshold conditions for a non-autonomous epidemic model with vaccination", Applicable Analysis, 2008 Publication	<1%
12	Submitted to University of Durham Student Paper	<1%
13	David M Vickers. "Current crisis or artifact of surveillance: insights into rebound chlamydia rates from dynamic modelling", BMC Infectious Diseases, 2010 Publication	<1%
14	www.suagm.edu Internet Source	<1%
15	KEELING, M.J.. "Individual-based Perspectives on R"0", Journal of Theoretical Biology, 20000307	<1%

	Publication	
16	Submitted to University of Leeds Student Paper	<1%
17	faculty.ksu.edu.sa Internet Source	<1%
18	Submitted to Associatie K.U.Leuven Student Paper	<1%
19	SAMUEL BOWONG, JEAN JULES TEWA, JURGEN KURTHS. "DYNAMICS OF THE SPREAD OF TUBERCULOSIS IN HETEROGENEOUS COMPLEX METAPOPULATIONS", International Journal of Bifurcation and Chaos, 2013 Publication	<1%
20	Banks, Natalie Clare, Dean Ronald Paini, Kirsty Louise Bayliss, and Michael Hodda. "The role of global trade and transport network topology in the human-mediated dispersal of alien species", Ecology Letters, 2014. Publication	<1%
21	Submitted to University of Portsmouth Student Paper	<1%
22	Hisashi Inaba. "Age-Structured Population Dynamics in Demography and Epidemiology", Springer Science and Business Media LLC, 2017	<1%

Publication

Exclude quotes	Off	Exclude matches	Off
Exclude bibliography	On		