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Developing Insurance Mathematical Model to Assess Economic Burden of Dengue Outbreaks

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Abstract Dengue fever is a vector-borne viral disease that has become a worrisome health issue in tropical and subtropical countries. The seasonal trend of dengue incidence encourages outbreaks with a high risk of infection at particular periods annually that potentially resulted in a significant economic burden. The epidemiological mathematical model, the SIR-SI model, is modified by considering the time-dependent and periodic-forced infection rate parameter through sinusoidal functions to obtain well data fitting. We show the existence and the stability of the disease-free and endemic equilibria for the system and their relation to the basic reproduction number of the disease. Next, we adapt the insurance concept to develop an insurance mathematical model that accommodates the proposed dengue transmission model in calculating nominal premiums. An increase in the basic reproduction number as an important indicator of the level of disease transmission risk resulted in an increase in the nominal premium. We also introduce a reserve function that guarantees sufficient premium payments collected by insurer to cover up future expenditure due to dengue outbreaks. Through this reserve function, we obtain an adjusted premium as a minimum value of premium which ensures that the reserve function is always positive. Mathematical models combined with insurance features have the potential to become important tools for relevant authorities to gain insight into disease transmission dynamics as well as assess the economic burden induced by the occurrence of disease outbreaks.

Keywords Dengue, SIR model, insurance, economic burden, premium

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1. Introduction

Globally, dengue is an arboviral infection that is highly endemic in regions featuring tropical and subtropical climates. Over the past few decades, the worldwide dengue incidence has increased significantly in terms of the frequency of epidemics, and severe dengue disease is the notable cause of mortality and morbidity [1]. Dengue has a huge impact on human health and an estimated half of the world's population in more than 128 countries is now at risk with Asia bearing 70% of the global burden [2–4]. According to an estimate, annually, 100 million people are affected by dengue infections, and 500,000 are hospitalized with more than 25,000 reported deaths globally [5]. More than 20% of the annual dengue cases evolve into a high level of dengue disease severity, dengue hemorrhagic fever (DHF) or dengue shock

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syndrome (DSS), which requires intensive medical treatment, whereas the rest of the cases remain in the milder dengue fever (DF) form. Dengue is considered the fastest-growing vector-borne viral disease in the past 30 years, and the number of incidences has increased fourfold [6]. In the 1970s, dengue epidemics were reported in less than 10 countries. However, nowadays, more than 150 countries all over the world were affected by dengue infection [7]. WHO recorded around 5.2 million dengue cases globally in 2019 which increased dramatically compared to 2.4 million cases in 2010 and half a million cases in 2000 [8]. Moreover, within 2000-2019, age-related dengue mortality are higher in younger age group and showed an increase from 960 to 4,032 deaths in a year [9].

The complexities of the mechanism underlying dengue re-emergence are not yet fully understood, which implies that the occurrence of the outbreaks is mostly unpredictable. Various researches have been conducted to examine the possible variables that influence the emergence of dengue outbreaks around the world. Climate change, globalization process, unplanned urbanization, and environmental degradation allegedly play a critical role in the increase and expansion of dengue cases. In general, meteorological factors have been recognized as the major determinant in driving dengue epidemic. Climatic and weather conditions greatly influence the development, ecology, behavior, and survival of the *Aedes* mosquito as the primary vector that transmits dengue virus [10]. The risk of dengue infection in humans is strongly associated to the number of mosquito larvae [11]. The number of mosquito larvae in most tropical and subtropical regions is higher in the rainy season than in other seasons [12–14]. The expansion of egg-laying areas due to high rainfall and a slum environment that allows many water-filled containers has led to an abundance of vector populations. Weather in each region has a trend of repeating each year depending on climatic conditions which in turn encourages the recurrence of dengue outbreaks with a high number of cases at certain periods in a year [15]. The seasonal pattern of dengue cases coincides mostly with the rainy season.

Dengue is a significant economic burden of infectious disease in many dengue affected areas, especially in developing countries. Shepard and others [16] have conducted a research about the economic burden related to dengue in some Southeast Asia countries, and over the decade of 2001-2010, it was estimated that the annual average economic burden was US$950 million (US$610 million - US$1,384 million, with 95% certainty levels). In Sri Lanka, the total cost of dengue case management in hospitals were approximately US$3.45 million or US$1.50 per capita), whereas the total expenditure for dengue prevention programs exceeded US$1.27 million in 2012 [17]. In 2010, economic cost related to dengue in the Americas averaged US$2.1 billion per year, with substantial year-to-year variation ranging from US$1 billion to US$4 billion [18]. Considering all these previous studies, it can be seen that, in recent years, the dengue outbreaks have become a burden on the country's economy and have had a significant effect on the worldwide socio-economic sector. The economic cost induced by dengue epidemic can be generally categorized as direct and indirect cost. Expenses incurred for the prevention program, surveillance and reporting, and medical treatment are included in direct costs [19]. The cost of the prevention program accommodates activities to prevent dengue, such as controlling vector population and awareness campaigns. Surveillance and reporting cost is linked to the efforts of governments and related authorities in observing and disseminating any dengue information to the public. Medical expenses include the costs of diagnosis, hospitalization, and outpatient care [20]. In addition, indirect

costs due to dengue are related to lost productivity and lost human hours due to sickness and untimely death [21].

Although it is difficult to assess the economic burden caused by dengue outbreaks, it is crucial to estimate the costs of infectious disease mitigation by health authorities and the government as policymakers. Information on the economic burden caused by dengue outbreaks is required to set health policy priorities, allocate the funds needed to control the disease, inform decision-makers about the implementation of existing dengue fever management programs, and propose new strategies that possibly increase the effectiveness of the disease control. The effectiveness of the healthcare system is also influenced by the level of affordability of medical expenses for each individual. Developing a conceptual framework for estimating future direct medical costs is an important task in reducing the burden effects of epidemics and promoting the development of more efficient control strategies.

This paper deals with the development of an insurance-based model to determine the direct costs of medical treatment related to dengue outbreak. The parameter of infection rate in the SIR-SI model is considered time-dependent and periodic to capture the seasonal pattern of dengue case data. The calculation of insurance premiums is redefined referring to the proposed dengue transmission model from an insurance perspective. The approach of using mathematical models integrated with insurance concept is expected to provide new insights and usefulness in both explaining the dynamic of dengue transmission and assesing the economic burden induced by the outbreak. In addition to being useful for health authorities and the government, estimating the economic burden and overall costs of the epidemic can assist the insurance industry to develop new breakthroughs in health insurance.

2. Methods

2.1. Mathematical model

To obtain the description of the dynamics of dengue within host and vector populations, we modified a classical SIR model for infectious disease. The SIR model type is the basic model in epidemiology that is widely used to explain and describe disease transmission [22]. From an epidemiological viewpoint, host populations can be subdivided into three compartments with respect to the dominant circulating strain of dengue virus, and vector populations can be subdivided into two compartments. The host is divided into susceptibles (S_h), i.e., individuals who do not have immune defenses against viral infection; infected (I_h), i.e., individuals that are infected and infectious; and recovered (R_h), i.e., individuals that are recovered from dengue infection. The vector is divided into susceptibles (S_v), i.e., mosquitoes that free from the virus, and infected (I_v), i.e., mosquitoes that carry the virus and can transmit it to the host through bites. For the sake of simplicity, we assume that the vectors carry the virus until death and no recovery compartment for the vector.

The dengue transmission model is shown in the following system of non-linear

differential equations.

$$\begin{aligned}
\frac{dS_h}{dt} &= \mu_h(N_h - S_h) - \beta_h S_h \frac{I_v}{N_v}, \\
\frac{dI_h}{dt} &= \beta_h S_h \frac{I_v}{N_v} - (\mu_h + \gamma_h) I_h, \\
\frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= \mu_v(N_v - S_v) - \beta_v S_v \frac{I_h}{N_h}, \\
\frac{dI_v}{dt} &= \beta_v S_v \frac{I_h}{N_h} - \mu_v I_v.
\end{aligned} \tag{2.1}$$

The parameters β_h and β_v represent the rate of infection from vector-to-host and host-to-vector, respectively. The host natural birth and mortality rate is denoted by μ_h, while μ_v denotes the reproduction rate of vector. The parameter γ_h indicates the recovery rate of host from dengue infection.

Considering that there are two compartments in the mosquito population which contain parameters whose values are difficult to observe and we do not have reliable data about mosquito populations, we decide to reduce the vector compartments in its coexistence equlibrium. The coexistence equilibrium is considered as the best representation for vector population because vector dynamics occur on a faster time scale than host dynamics. The coexistence equilibria are given as follows.

$$S_v^* = \frac{\beta_v \mu_v N_v N_h}{\beta_v I_h + \mu_v N_h}, \qquad I_v^* = \frac{\beta_v N_v I_h}{\beta_v I_h + \mu_v N_h}. \tag{2.2}$$

By using the equlibrium value I_v^* for the virus carrier vector and the fact that $S_h = N_h - (I_h + R_h)$, we obtain a reduced equations system given by:

$$\begin{aligned}
\frac{dI_h}{dt} &= \beta_h(N_h - (I_h + R_h))\frac{\beta_v I_h}{\beta_v I_h + \mu_v N_h} - (\mu_h + \gamma_h) I_h, \\
\frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h.
\end{aligned} \tag{2.3}$$

We have two possible equilibria of the differential equation system, i.e. the disease-free equilibrium (DFE), $(I_h^\circ, R_h^\circ) = (0, 0)$, and the endemic equilibrium (EE), $(I_h^*, R_h^*) = (\mu_h \eta, \gamma_h \eta)$, where

$$\eta = \frac{\beta_h \beta_v - \mu_v(\mu_h + \gamma_h)}{\beta_v(\beta_h + \mu_h)(\gamma_h + \mu_h)} N_h.$$

To determine a basic reproduction number, R_0, we use Next Generation Matrix (NGM) to obtain a Jacobian matrix by considering the infected subsystem and linearizing at disease-free equilibrium. The basic reproduction number is derived from the dominant eigenvalue of the Jacobian matrix, which is shown as:

$$R_0^2 = \frac{\beta_h \beta_v}{\mu_v(\mu_h + \gamma_h)}. \tag{2.4}$$

The existence of the disease-free equilibrium (DFE) is guaranteed. Meanwhile, we require the condition $R_0 > 1$ for the existence of endemic equilibrium (EE) by considering the value of I_h^* and the positivity of $(\mu_h, \gamma_h, \beta_h, \beta_v)$. Theorem 2.1 and Theorem 2.2 show the stability analysis at DFE and EE, respectively.

Theorem 2.1. *If $R_0 < 1$, then DFE is locally asymptotically stable.*

Proof. At the disease-free equilibrium, the Jacobian matrix is given as follows:

$$J^\circ = \begin{bmatrix} \frac{\beta_h \beta_v}{\mu_v} - (\mu_h + \gamma_h) & 0 \\ \gamma_h & -\mu_h \end{bmatrix}. \tag{2.5}$$

Matrix J° has a characteristic polynomial $P(\lambda) = \lambda^2 + a_1\lambda + a_2$ where

$$a_1 = \mu_h - (\gamma_h + \mu_h)(R_0^2 - 1),$$
$$a_2 = -\mu_h(\mu_h + \gamma_h)(R_0^2 - 1).$$

Since all parameters are positive, then $R_0 < 1$ implies $a_1, a_2 > 0$. Based on *Routh-Hurwitz*'s stability criterion, the polynomial has roots with a negative real part. Therefore, the disease-free equilibrium (DFE) is locally asymptotically stable. □

Theorem 2.2. *If $R_0 > 1$, then EE is locally asymptotically stable.*

Proof. At the endemic equilibrium, the Jacobian matrix is given as follows:

$$J^* = \begin{bmatrix} \frac{\delta(1+\alpha+\rho)}{\rho} & \delta \\ \alpha & -1 \end{bmatrix} \tag{2.6}$$

with

$$\alpha = \frac{\gamma_h}{\mu_h}, \qquad \rho = \frac{\beta_h}{\mu_h}, \qquad \delta = \frac{\mu_v(\alpha+1) - \beta_v\rho}{\mu_v(\alpha+1) + \beta_v}.$$

Matrix J^* has a characteristic polynomial $P(\lambda) = \lambda^2 + a_1\lambda + a_2$ where

$$a_1 = 1 - \frac{\delta(1+\alpha+\rho)}{\rho},$$
$$a_2 = -\delta\left(\alpha + \frac{1+\alpha+\rho}{\rho}\right).$$

Considering the *Routh-Hurwitz*'s stability criterion, the second-degree polynomial $P(\lambda)$ has both roots with negative real part if and only if the coefficients are positive. Hence, the condition for asymptotic stability of EE is $a_1, a_2 > 0$. Since μ_h, β_h, γ_h are positive, then we have $\alpha > 0$ and $\rho > 0$. Consequently, $a_1 > 0$ and $a_2 > 0$ are satisfied when $\delta < 0$ or equivalently:

$$\frac{\mu_v(\alpha+1) - \beta_v\rho}{\mu_v(\alpha+1) + \beta_v} = -\frac{\mu_v(\mu_h + \gamma_h)}{\mu_v(\mu_h + \gamma_h) + \mu_h\beta_v}\left(R_0^2 - 1\right) < 0. \tag{2.7}$$

The condition $R_0 > 1$ implies that the Jacobian matrix J^* has eigenvalues with negative real parts and the endemic equilibrium (EE) is locally asymptotically stable. □

2.2. Parameter estimation

To obtain a more realistic description, we considered the infection rate parameter, β_h, to be time-dependent, whose value changes over time. The obtained mathematical model will be fitted to dengue data recorded in Semarang, Indonesia. Semarang

is both the largest and the capital city of Central Java province. It is considered as one of the dengue-endemic cities in Indonesia, where high-frequency of incidences are reported annually. Semarang is precisely located in the middle of the north coast of the densely populated island of Java. It covers an area of 373.78 square kilometers (144.32 square miles) and, at the 2020 census, the population of the city was 1,653,524 which makes it the ninth most populous city in Indonesia. Semarang features high temperatures throughout the year of around 30°C (87°F) to 32°C (90°F). The rainy season mostly occurs at the beginning (January-April) and the end (November-December) of a year, in which January tops the wettest month list with 371mm (14.6in) of rainfall. Overall, Semarang features a typical tropical climate that supports vector population growth and dengue virus transmission.

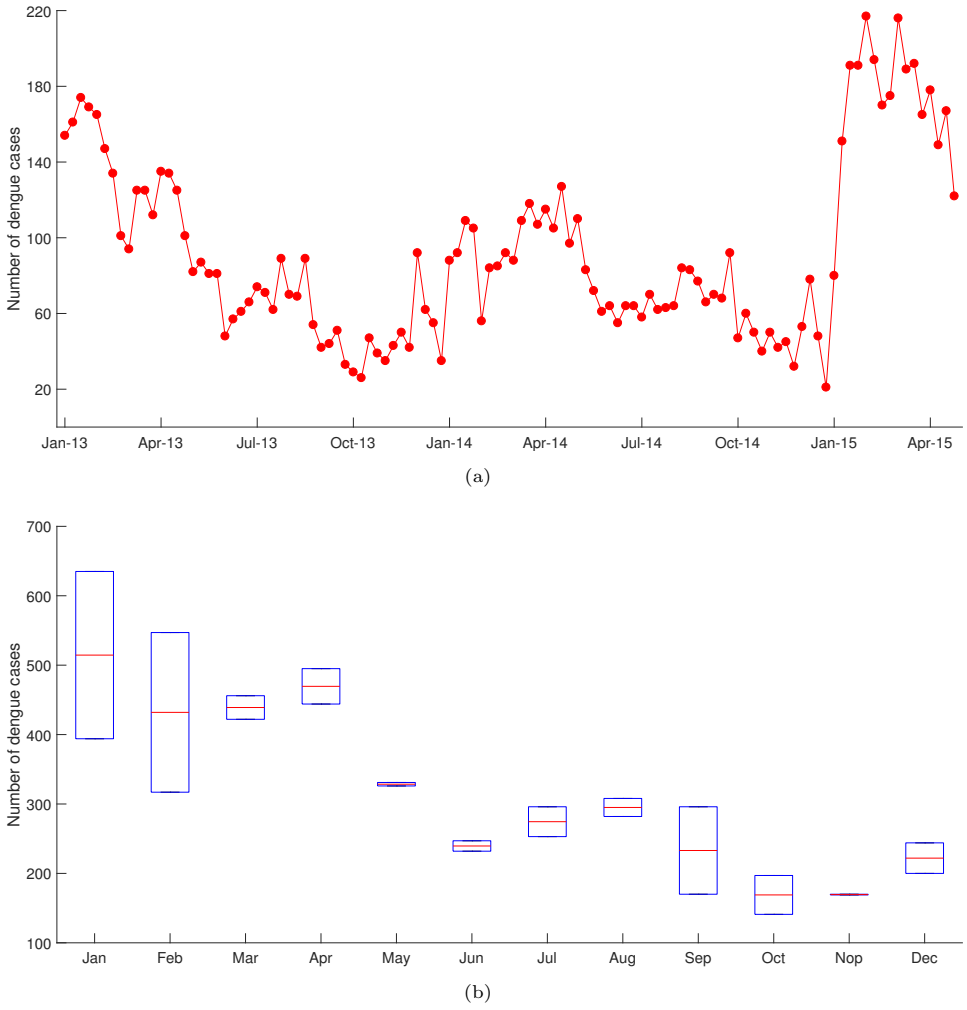


Figure 1. (a) The number of dengue cases; and (b) the box plot of dengue cases each month in Semarang.

Dengue data is obtained and recorded by the Semarang City Health Office on a weekly basis. The total number of hospitalized dengue cases is an accumulation of Dengue Fever (DF), Dengue Hemorrhagic Fever (DHF), and Dengue Shock

Syndrome (DSS), with no specific classification for each level. Due to lack of data availability, we use dengue data for 112 weeks starting from the first week of January 2013 to the last week of April 2015. Figure 1(a) shows the number of dengue cases throughout the observation period and Figure 1(b) presents a boxplot which indicates that most of the dengue incidences in Semarang occur at the beginning of the year, January-April, when it is the wettest period with high rainfall intensity.

Further, seasonal variation in terms of dengue incidences is generally found in countries featuring either tropical or subtropical climates. An abundance of vector populations in a specific season leads to the occurrence of seasonality in dengue outbreaks. The repeated pattern of climate variability drives the annual variation of dengue transmission and results in seasonal dengue cases [23]. It can be observed in Figure 1(a) that the number of dengue incidences in Semarang also indicates the existence of seasonality. The high incidences mostly occurred during the rainy season and, as entering the dry season, the number of dengue incidences started to decrease. To capture the seasonality pattern in the dengue dynamics, we accommodated it into the model through the infection rate with periodic forced. We used a sinusoidal function inspired by empirical observations of vector dynamic that shows seasonal patterns in reality [24]. The infection rate parameter is approximated as a periodic function to accommodate seasonality seasonality and is given as follows:

$$\beta_h(t) = \beta_0 + \sum_{k=1}^{n} \left[a_k \cos(k\omega t + \phi_k) + b_k \sin(k\omega t + \psi_k) \right], \tag{2.8}$$

where β_0 denotes the baseline, a_k and b_k are the cosine and sine amplitude, ϕ_k and ψ_k are the cosine and sine phase, ω is the period of seasonality, and n is the number of modulo. By substituting Eq. 2.8 into our Model 2.1, we obtain a periodically forced non-linear system. Although there are many complex functions that can represent seasonality, we consider that a simple sinusoidal function is sufficient to describe the seasonal incidence induced by seasonal climate cycles.

The values of each parameter in the periodic infection rate, $\beta_h(t)$, were estimated by minimizing the error between dengue data and model output. We performed Spiral Dynamics Optimization (SDO) method introduced by Tamura and Yasuda [25] to obtain the minimum root-mean-square error (RMSE) between model output (I_h) and the data of infected people. The fixed values of the remaining parameters obtained from the literature and the references were summarized in Table 1.

Table 1. Fixed parameter values used in the numerical simulation.

Parameter	Description	Value	Unit	References
μ_h	Host natural birth and mortality rate	$1/(65 \times 52)$	1/week	[26–29]
μ_v	Vector mortality rate	$7/10$	1/week	[26,27]
β_v	Infection rate of host to vector	$2\mu_v$	1/week	[27]
γ_h	Host recovery rate	$7/6$	1/week	[28,29]
N_h	Total population	1.5×10^6	people	-
$S_h(0)$	Initial value of susceptible human	$0.569 \times N_h(0)$	people	[28,29]

The initial value of the total population, $N_h(0)$, approximates the total population in Semarang to be 1.5 million people. The initial value of infected host is obtained from data i.e. the number of infected individuals recorded in the first week of January 2013, $I_h(0) = 154$. Based on the researches conducted by Andraud [28] and Ooi [29], we assume that the initial proportion of susceptible individuals in a population is 56.9%. Considering the constant human population, the initial value of recovered host is determined by $R_h(0) = N_h(0) - (S_h(0) + I_h(0))$.

2.3. Insurance model

Insurance coverage can be interpreted as an agreement about financial protection loss and is represented by a policy. As a form of risk management, insurance is particularly used to guard against the potential risk of an uncertain future loss [30]. The main idea of developing a mathematical model of insurance is to estimate how much we should allocate today to cover possible financial losses in the future. Basically, the model of insurance can be considered as a process of developing a specific cooperation between two parties in which, in exchange for a fee, a party agrees to compensate another party in the event of a particular loss, damage, or injury [31]. The party that underwrites the insurance risk and undertakes to pay compensation for financial losses is called the insurer. The insured, on the other hand, is the party covered under the insurance policy who will receive compensation for financial loss. Insurance companies charge premiums in exchange for providing insurance coverage to any person or company for availing of an insurance policy. The concept of insurance can be used to measure economic burden by estimating potential future financial losses due to certain risk events listed in insurance policy. Referring to this insurance concept, we are trying to develop a new approach to calculating insurance premiums through a dengue transmission model and introducing a reserve function that guarantees sufficient premium payments collected by the insurer to cover future claims as an effort to assess the economic burden due to dengue outbreaks.

Considering the SIR compartment model described in Eq. 2.1, we redefine it as introduced by [32] to obtain different points of view from the insurance perspective. Susceptible people can contribute to the insurance system by joining health insurance, being charged insurance premiums, and paying a number of insurance funds to benefit medical expense claims when they are turning to be dengue-infected people. Meanwhile, during a dengue outbreak, infected people who participate in paying the premium are the party who gets the financial loss and will receive payment claims from the insurance company for medical treatment costs while being hospitalized or other compensation benefits for the paid premium.

Let variable $S_h(t)$ and $I_h(t)$ denote the probability of an individual being susceptible and infected at time t, respectively. In terms of income from insurance, the total expected present value of premium payments in the period t is $P(t)$. On the other hand, from the expenditure side of the investment plan, the total expected present value for benefits payments (claims) in period t is denoted by $C(t)$. The values of $P(t)$ and $C(t)$ are given by the following equations.

$$\begin{aligned} P(t) &= \int_0^t \exp(-\sigma t) S_h(t) dt, \\ C(t) &= \int_0^t \exp(-\sigma t) I_h(t) dt, \end{aligned} \tag{2.9}$$

where $\sigma > 0$ denotes the force of interest. Considering the expected total value of premium payments, $P(t)$, and claim payments, $C(t)$, the possible future loss by investors, is defined as follows:

$$L(t) = C(t) - \kappa P(t), \tag{2.10}$$

where κ represents the amount of premium payment. According to the equivalence principle, an insurance investment requires that the expected loss value is zero. Considering this equivalence principle, the amount of premium for each payment can be calculated as follows:

$$\kappa = \frac{C(t)}{P(t)} = \frac{\int_0^t \exp(-\sigma t) I_h(t) dt}{\int_0^t \exp(-\sigma t) S_h(t) dt}. \tag{2.11}$$

Thus, it can be observed that the amount of premium payment, κ, depends on the value of basic reproduction number of disease (R_0) and the force of interest.

The basic reproduction number (R_0) is generally known as the average number of secondary infections and it becomes an important indicator to determine if the disease will vanish or persist. Therefore, the dynamic of the infected individual (I_h) is strongly associated with the value of R_0, and the dynamic of I_h significantly influences the amount of premium defined by Eq. 2.11. In fact, an increase in the R_0 value indicates an increased risk of the disease spreading and an accelerated outbreak progress. The large impact of the outbreak has the potential to induce even greater economic losses that need higher premium payments to cover there losses.

In addition, the premium payment computed by proposed mathematical model does not guarantee that the investment income is sufficient to cover up future expenditure, especially when the dengue infection transmit quickly and widely. To verify this, we introduce a reserve function, $F(t)$, and perform the actuarial analysis. We define the reserve function as the difference between accumulated claim payment and accumulated premium payment. As we mentioned before, $C(t)$ and $P(t)$ are the total sum of claim received and the total sum of premium paid, respectively, up to time t. The rate of change in the total claim can be denoted as the sum of the rate of change of claim payment and the rate of change of interest obtained from the current total claim payment. In the total premium income, the rate of change can be defined in a similar way. The rate of change in claim payment and premium income are presented as the following differential equations.

$$\begin{aligned} \frac{dC}{dt} &= I_h + \sigma C, \\ \frac{dP}{dt} &= \kappa S_h + \sigma P, \end{aligned} \tag{2.12}$$

with $C(0) = I_h(0) > 0$ and $P(0) = \kappa S_h(0) > 0$. Since the value of reserve function is obtained from the difference between accumulated premium payments and accumulated claims, the reserve function has the rate of change as follows:

$$\frac{dF}{dt} = \frac{dP}{dt} - \frac{dC}{dt} = \kappa S_h + \sigma P - (I_h + \sigma C) = \kappa S_h - I_h + \sigma(P - C). \tag{2.13}$$

Using our assumption $F = P - C$, we obtain:

$$\frac{dF}{dt} = \kappa S_h - I_h + \sigma F, \tag{2.14}$$

with the initial value $F(0) = \kappa S_h(0) - I_h(0) > 0$.

The premium computed by the equivalence principle does not ensure premium sufficiency to pay claims and is possible to result in undesirable negative reserves during the observation period. This may occur due to the insurer not being able to collect enough premiums from investors. Therefore, it is necessary to determine the minimum amount of the premium to ensure that benefit reserves are always positive. We modify the premium calculation in such a way that the premium income collected by the insurer can guarantee the adequacy of claim payments submitted by the insured in the future. The proposed new premium is adjusted by setting the value of the reserve function to be positive during the policy term.

Theorem 2.3. *If* $\kappa' = \frac{I_h(0)+\int_0^t \exp(-\sigma t) I_h(t)dt}{S_h(0)+\int_0^t \exp(-\sigma t) S_h(t)dt}$ *then* $F(t) > 0$ *for all time* t.

Proof. Consider the rate of change of the reserve function

$$\frac{dF}{dt} = \kappa S_h - I_h + \sigma F,$$
$$\frac{d}{dt}\left(\exp(-\sigma t)F\right) = \left(\exp(-\sigma t)\right)\left(\kappa S_h - I_h\right),$$
$$\int_0^t \frac{d}{dt}\left(\exp(-\sigma t)F\right) dt = \int_0^t \left(\exp(-\sigma t)\right)\left(\kappa S_h - I_h\right) dt,$$
$$\exp(-\sigma t)F = F(0) + \int_0^t \left(\exp(-\sigma t)\right)\left(\kappa S_h - I_h\right) dt,$$
$$\exp(-\sigma t)F = \kappa\left(S_h(0) + \int_0^t \left(\exp(-\sigma t)\right) S_h dt\right) - \left(I_h(0) + \int_0^t \left(\exp(-\sigma t)\right) I_h dt\right).$$

Hence, to obtain $F(t) > 0$ for all time t, we require

$$\kappa\left(S_h(0) + \int_0^t \left(\exp(-\sigma t)\right) S_h dt\right) - \left(I_h(0) + \int_0^t \left(\exp(-\sigma t)\right) I_h dt > 0 \qquad (2.15)$$

or equivalently

$$\kappa > \frac{I_h(0) + \int_0^t \exp(-\sigma t) I_h(t)dt}{S_h(0) + \int_0^t \exp(-\sigma t) S_h(t)dt}. \qquad (2.16)$$

Thus, the minimum of κ showed in Eq. 2.16 can be set as the proposed new premium, κ'.

$$\kappa' = \frac{I_h(0) + \int_0^t \exp(-\sigma t) I_h(t)dt}{S_h(0) + \int_0^t \exp(-\sigma t) S_h(t)dt}. \qquad (2.17)$$

□

3. Result and simulation

In this section, the results of numerical simulation are presented considering the estimated parameter values of seasonal infection rate $\beta_h(t)$ that minimize error between dengue data and the model's output. Dengue data is the weekly number of dengue-infected people reported and recorded by Semarang City Health Office from January 2013 to April 2015. The mathematical model's output represents the result of numerical simulation that indicates the number of infected people, I_h, in a week.

We simulate the calculation of premium through the simulation of the insurance mathematical model. We also consider the variations of basic reproduction number R_0 and force of interest separately, and interpret the simulation's result.

Table 2. Parameter values for seasonal infection rate β_h.

Parameter	Description	95% Confidence Interval		
		Estimated Value	Lower Bound	Upper Bound
β_0	Baseline	0.7513	0.6522	0.8503
a_1	Cosine amplitude for mod 1	−0.0603	−0.0638	−0.0568
a_2	Cosine amplitude for mod 2	−0.4683	−0.5248	−0.4118
a_3	Cosine amplitude for mod 3	−0.0579	−0.0623	−0.0535
ϕ_1	Cosine phase for mod 1	3.5983	3.3261	3.8704
ϕ_2	Cosine phase for mod 2	−3.5535	−3.8172	−3.2898
ϕ_3	Cosine phase for mod 3	−6.4968	−6.8232	−6.1704
b_1	Sine amplitude for mod 1	−0.0108	−0.0114	−0.0102
b_2	Sine amplitude for mod 2	0.4352	0.3818	0.4886
b_3	Sine amplitude for mod 3	−0.0809	−0.0878	−0.0740
ψ_1	Sine phase for mod 1	9.9600	9.1993	10.7207
ψ_2	Sine phase for mod 2	4.1839	3.9507	4.4171
ψ_3	Sine phase for mod 3	5.6060	5.2227	5.9893

3.1. Data fitting with seasonal infection rates

First, we determined the period of seasonality, ω, in our proposed sinusoidal function. The period of seasonality is $\omega = 2\pi T$ with T stands for the period of seasonality. We implemented Fast Fourier Transform (FFT) on dengue data and we obtained the dominant frequency of Fourier spectrum is $f = 0.02$ or equivalent to period $T = 50$ weeks, approximately corresponding to an annual pattern. Based on this fact that dengue cases recur annually, we afterward determined the number of modulo, n, which represents the number of local peaks in the annual dengue data. Applying the Gaussian filter, we obtained $n = 3$ which indicates the existence of three local peaks in a year. Next, in order to obtain the value of the remaining parameters in the periodic-forced infection rate, $(\beta_0, a_k, b_k, \phi_k, \psi_k)$, we used Spiral Dynamic Optimization. This optimization method determines the optimal value of a set of parameters that has a minimum error between the actual dengue data and the number of infected individuals (I_h) resulted from the mathematical model. The coefficient of the seasonal infection rate generated by the optimization is shown in Table 2. The bootstrap realization was implemented 100 times to obtain a 95% confidence interval.

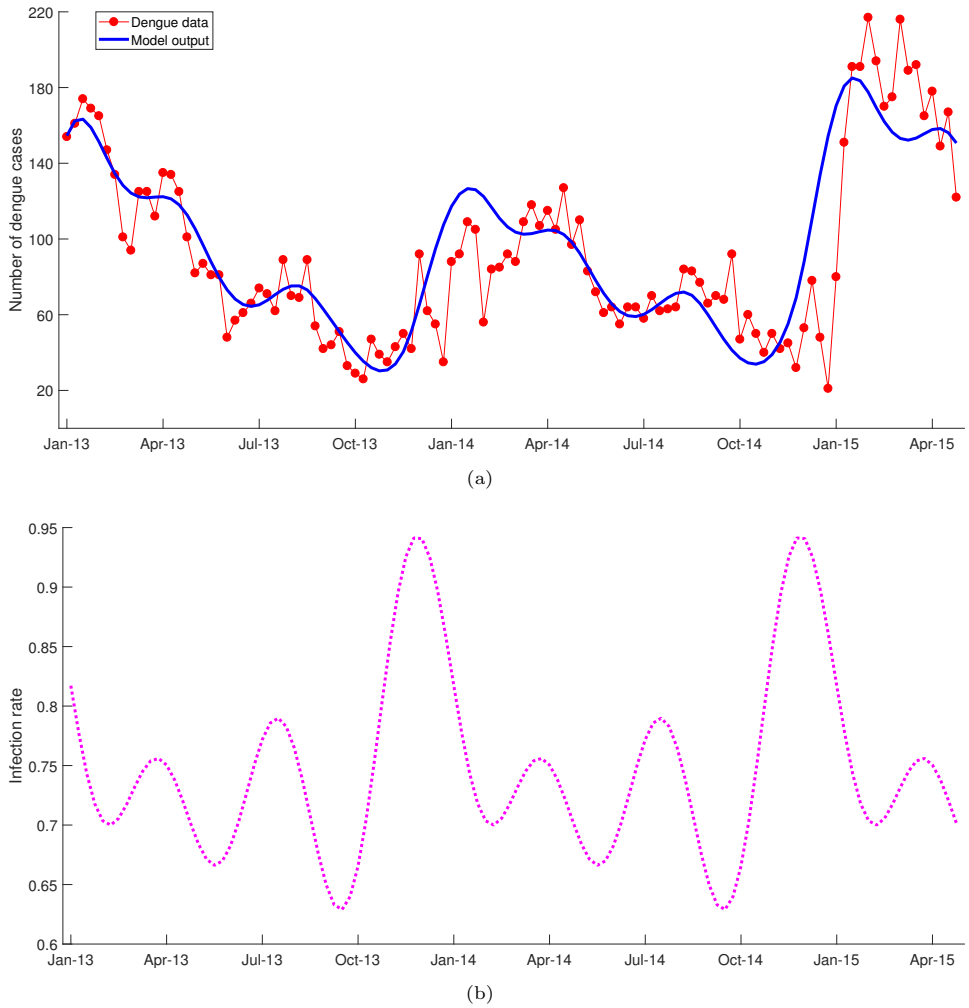


Figure 2. (a) The result of numerical simulation of data fitting by using seasonal infection rate $\beta_h(t)$; and (b) the seasonal infection rate $\beta_h(t)$.

In Figure 2(a), we present the simulation results that integrate the seasonal infection rate into the model. The simulations produce good data fitting with well-captured annual patterns. To measure the goodness of fit, the Pearson correlation coefficient is evaluated between the model's output and the dengue data. Using a significance level of $p < 0.05$, the coefficient of Pearson correlation is $r = 0.8307$ which indicates a firm positive connection between the simulation and the data. Figure 2(b) shows the time-dependent infection rate β_h indicating seasonality.

The high incidence of dengue in Semarang mostly occurs from January to April each year along with the wet period. Entering the dry season in early June, dengue cases have decreased until October. This repeat pattern occurs every year and seasonal infection rates explain this phenomenon well. The increase in dengue cases along with the rainy season can be explained by the expansion of the egg-laying area for mosquitoes in stagnant water generated from trapped rainwater. High rainfall and rain intensity that is not accompanied by a good sanitation system will

support mosquitoes to grow and breed, particularly in slums and densely populated areas. The expansion of the breeding site encourages an abundance of mosquito populations and automatically increases the risk of dengue fever transmission.

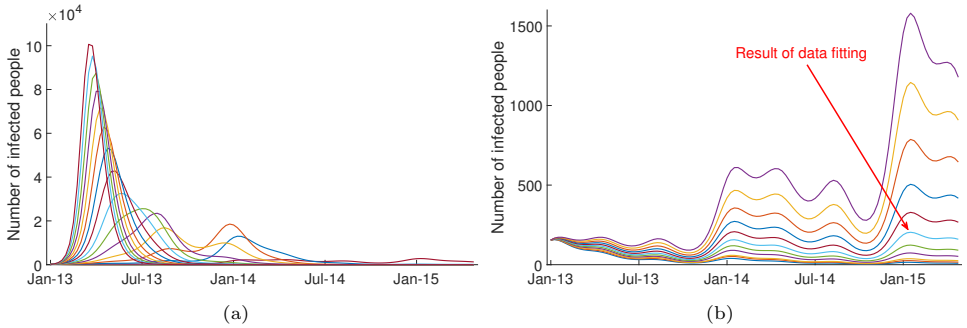


Figure 3. (a) Variations of the basic reproduction number, $0.8659 \leq R_0 \leq 2.5708$, on the number of infected people; and (b) variations of R_0 close to the result of data fitting, $1.2425 \leq R_0 \leq 1.3282$, on the number of infected people and the simulation result of $R_0 \approx 1.2876$ is indicated by the red arrow.

The basic reproduction number, R_0, is intended to be an indicator of transmissibility of infectious agents and parasites [33]. This parameter is often used as a reference to determine the severity of dengue outbreaks and is a consideration for designing control strategies [34]. As can be seen in Eq. 2.4, the value of R_0 is affected by the value of the rate of dengue infection. Working with a time-dependent infection rate parameter, the calculation of R_0 is carried out using the average seasonal infection rate indicated by the baseline β_0 in the sinusoidal function (see Eq. 2.8). Therefore, the value of R_0 of the model generated by data fitting is $R_0 \approx 1.2876$. Figure 3(a) shows the number of infected people respect to the variations of basic reproduction number, and Figure 3(b) shows the same result as a narrower R_0 and is close to the value of data fitting. We can observe that an increase in R_0 induces an earlier dengue outbreak peak. On the other hand, a decrease in basic reproduction number R_0 implies a lower number of dengue cases.

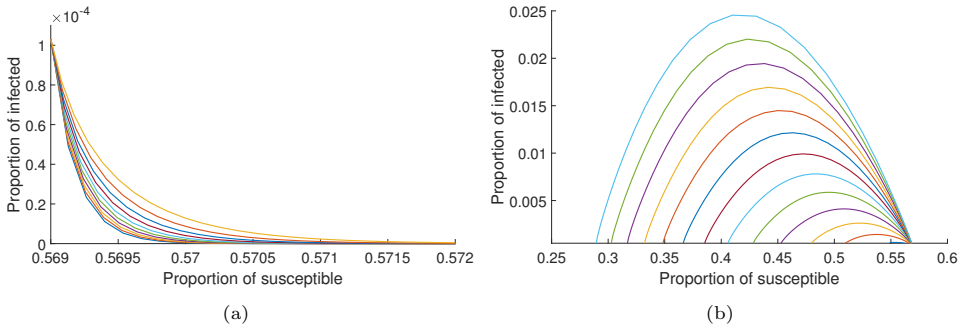


Figure 4. The solution trajectory projected onto susceptible and infected compartment, (S_h, I_h), with $R_0 < 1$ for disease free case (a); and with $R_0 > 1$ for endemic case (b).

Figures 4(a) and 4(b) present the solution trajectories of our proposed mathematical model system in the (S_h, I_h) plane for disease-free and endemic cases,

respectively. With initial value $(S_h(0), I_h(0))$, the simulation for $R_0 < 1$ results in the disappearance of the disease as shown by the proportion of infected tending to zero. For endemic case, $R_0 > 1$ encourages the occurrence of dengue outbreak.

3.2. Insurance model simulation

The basic reproduction number of an epidemiological model is generally known as the average number of secondary infections produced by an infected individual in the population of susceptible. The value R_0 is an important indicator to determine whether the disease will vanish or persist. All references in mathematical epidemiology have mentioned that $R_0 < 1$ indicates that the number of infected individuals decreases over time and the disease vanishes. The human population converges to a state with zero infected individuals, or it is called a disease-free equilibrium. Otherwise, when $R_0 > 1$, the number of infected people increases, attains a peak, and declines to an endemic equilibrium for endemic models. Hence, the value of basic reproduction number, R_0, has a significant influence on the dynamics of infected (I_h) and susceptible (S_h) individuals in our mathematical model which will automatically affect the calculation of the amount of the premium payment defined in Eq. 2.11. Working with a time-dependent infection rate parameter, the value of R_0 presented in Figure 5(a) is calculated using the average of seasonal infection rate, indicated by the baseline β_0 in the sinusoidal function as can be seen in Eq. 2.8.

We can observe in Figure 5(a) that the changes in the value of basic reproduction number (R_0) affect the variations in the amount of insurance premium. A higher value of R_0 results in a higher premium value. An increase in the R_0 value indicates an increased risk of the disease spreading and an accelerated outbreak progress. The large and widespread impact of the outbreak has the potential to induce even greater future losses [35]. Thus, a greater risk of spreading disease will have an impact on increasing premium payments. In contrast, the smaller the R_0 value, the lower the risk of an outbreak occurring and the disease will disappear over time. In this condition, the potential for future losses is low, so the premium is small. Further, based on our simulation, the premium value starts to increase significantly when $R_0 > 1.3$. On the other hand, $R_0 < 1.3$ indicates that the payment value of insurance premium is close to zero. The results of our data fitting shown in Figure 2(a) use the average basic reproduction number $R_0 \approx 1.28$, which has a premium calculation value approximately 0.003 (see Figure 5(a)).

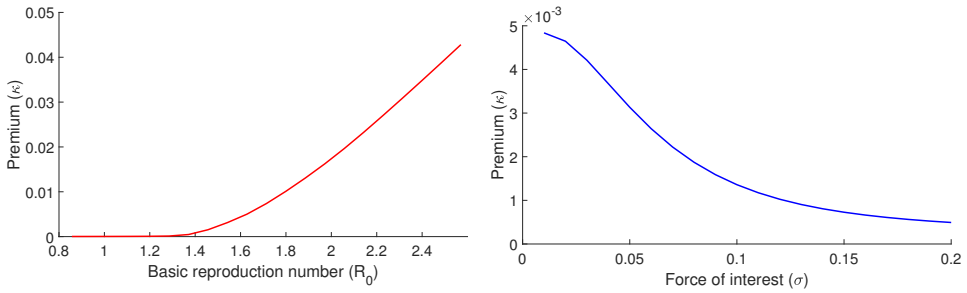


Figure 5. (a) Variations of the basic reproduction number (R_0) on the amount of premium payment; and (b) variations of the force of interest (σ) on the the amount of premium payment.

Figure 5(b) presents the variations of premium value affected by the change of force of interest (σ). The force of interest represents the uncertainty of future expenditures due to external factors or can be interpreted as a discounting factor. It can be seen that $\sigma = 1\%$ corresponds to the premium $\kappa = 4.8 \times 10^{-3}$ and the premium value continues to decrease as the value of force of interest increases, with $\sigma = 20\%$ resulting premium $\kappa = 4.9 \times 10^{-4}$. This can be explained that a large discounting factor (σ) results in a decrease in the present value of the future sum of the premium payment received by the insurer from the investors.

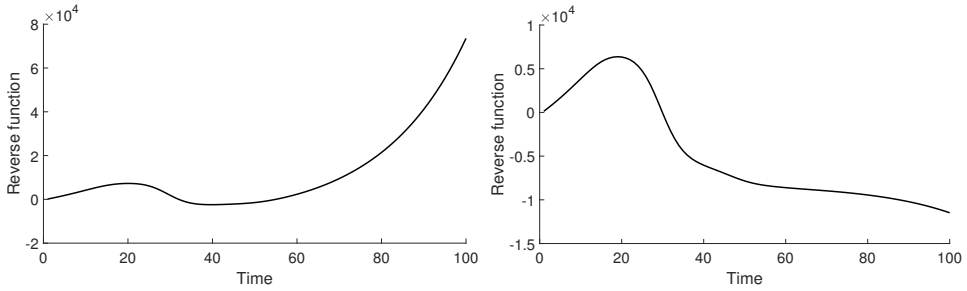
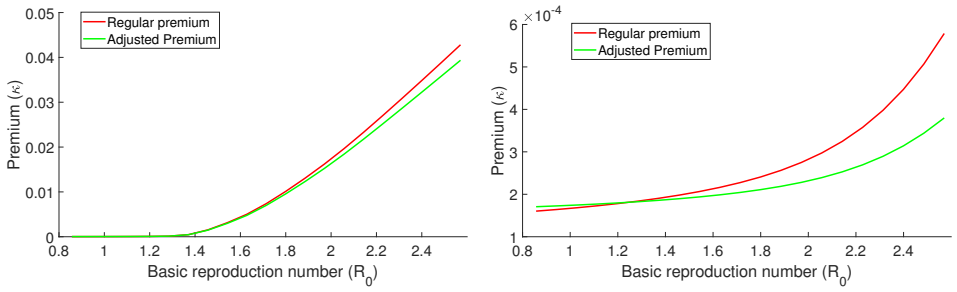


Figure 6. The dynamic of the benefit reverse function using: (a) $\kappa = 0.003$, and (b) $\kappa = 0.0025$.

In Figure 6, we display the dynamic of the reverse function that denotes the difference between total premium payments and total claim payments. We solved the differential equation given in Eq. 2.14 by pairing with Eq. 2.3 and performed numerical simulation to determine the reserve function. Using basic reproduction number $R_0 = 1.28$ and premium $\kappa = 0.003$, the value of the reserve function is positive throughout the observation time as presented in Figure 6(a). Now, by reducing the value of the premium to $\kappa = 0.0025$, the benefit reserve function shows an unexpected negative result (see Figure 6(b)). The premium value calculated by Eq. 2.11 is sufficient to cover medical expenses in the future. However, if the premium value is lowered to a certain level, it is possible for the insurer to suffer losses because the claim payments are greater than the total premium payments. Unwanted negative reserves during the policy term can also be interpreted that not enough premiums can be collected by the insurer from investors and the insurer needs to increase the nominal of the premium to guarantee positive reserves.

To ensure that the investment reserves owned by the insurer are sufficient to pay claims submitted by the insured for future medical expenses, the premium value needs to be defined so that the reserve function is always positive. We propose a new premium calculation as given in Eq. 2.17 which is the minimum premium limit that guarantees sufficient premium income to cover claim costs. In Figure 7(a), the value of adjusted premium indicated by the green line is smaller than the regular premium indicated by the red line. Using a larger value of force of interest, $\sigma = 1$, Figure 7(b) presents that the value of adjusted premium is higher than the regular premium for a relatively small basic reproduction number, and the adjusted premium value will be smaller than the regular premium for a large R_0. The more affordable premium value will certainly benefit the insurer by potentially attracting more investors to take advantage of insurance policies. Eventhough the premium

value is affordable, the insurer ensures that the premium income is guaranteed to be sufficient for the insurance coverage that will be claimed by the insured. Hence, a lower premium value as long as it is enough to cover claims will benefit both parties, the insurer paying the claim and the insured receiving compensation.



(a) (b)

Figure 7. Comparison between the value of regular premium and adjusted premium with variations in the basic reproduction number and different force of interest: (a) $\sigma = 0.05$, and (b) $\sigma = 1$.

From a practical point of view, applying the insurance mathematical model seems intricate. The number of infected individuals can be obtained from data on dengue case reports recorded by government health institutions. However, the number of susceptible individuals is difficult to determine due to the lack of specific records or valid scientific methods to estimate their numbers during the observation period. The main reasons behind the difficulty of determining the true number of susceptible individuals in reality are the large number in the population and the difficulty of distinguishing a susceptible individual from an immune individual. Therefore, it is important to calculate insurance premiums by prioritizing the number of infected people and highlighting the I_h function, instead of S_h.

4. Conclusion

In this study, we propose a mathematical insurance model to estimate the present financial risk and the economic burden related to dengue epidemic. The economic burden due to disease epidemics is a crucial budgeting challenge, especially in dengue-affected areas in the tropics and subtropics. Considering the classic compartment model, the SIR model is modified by setting the infection rate parameter to be a time-dependent parameter and the model output fitted to the actual dengue data recorded in Semarang. The periodic sinusoidal function is adopted as a parameter of the infection rate to capture the seasonal pattern of dengue cases which tends to recur every wet period along with the abundance of the mosquito population as the primary dengue vector. Local stability analysis and basic reproduction number derivation were also performed to explain the behavior of the model. For the SIR model with a time-dependent infection rate parameter, the average of β_h, denoted by β_0, is considered the best representative to determine the value of R_0.

Next, we utilize the concept of insurance to construct an insurance mathematical model by redefining the dengue transmission model. Susceptible individuals who are at risk to experience dengue infection in the future have the potential to suffer

financial losses due to medical expenses once they become infected. Susceptible individuals contribute to the insurance system as parties who pay premiums at an amount according to the insurer's policy after joining health insurance. During an outbreak, infected individuals may request compensation from the insurer to cover the medical expenses while being hospitalized as a form of claim payment. The amount of the insurance premium is determined based on the present value of the total premium income and total claim payments. Also, we introduced a reserve function which is measured by the difference between total premium and total claim as the basis for determining the lower limit on the premium value. The minimum value of the premium guarantees that the value of the reserve function is always positive, indicating that the insurance reserves from collecting premium are adequate to pay future claims. The insurer can set an affordable premium value to attract more investors to join the insurance and on the other hand the insurer has a low risk of experiencing a loss with a premium income greater than expenditures.

This study has numerous limitations that can be extended further. We neglect the secondary infection in dengue transmission which can potentially result in additional financial loss. The second limitation is that expenditure covered by insurance are only medical expenses during hospitalization. The insurance model can be extended by allocating funds to control and prevention strategies. Indirect costs induced by the dengue epidemic such as lost productivity, lost human hours, and untimely death can also be integrated into the premium pricing model. Measuring the economic burden caused by an outbreak is important to assist the government and related parties in determining the best strategy for dealing with the outbreak. Further, the insurance model can be implemented for other infectious diseases.

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