

Article

A One Health Approach to Water as an Ecological Enabler for Leptospirosis: A System Dynamics Model

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Abstract

Leptospirosis is a neglected zoonotic disease of global importance and remains a persistent public health challenge in Fiji, where outbreaks frequently occur following extreme weather events. This study developed a One Health System Dynamics model to simulate interacting human, rodent, and environmental subsystems over a 24-month outbreak-relevant period, parameterised using published epidemiological and ecological data. Model validity and robustness were assessed through the reproduction of known epidemiological patterns, including seasonal trends. Results indicate that leptospirosis incidence is primarily driven by the interaction between rodent population dynamics and water systems, with standing water acting as a critical ecological enabler for both *Leptospira* persistence and rodent habitat suitability. Extreme scenario testing indicated that eliminating water contamination drove cases to near zero within 14 months, whereas soil contamination had minimal effect. Sensitivity analyses identified rodent birth and death rates as the most influential parameters governing system behaviour. While an integrated One Health intervention produced the greatest cumulative reduction in cases (21.07%), intervention effects were additive rather than synergistic, reflecting the dominance of underlying ecological feedback structures. This study offers a novel system-based framework for understanding leptospirosis burden in Fiji. It advances beyond static risk models by capturing endogenous dynamics, feedback loops, and non-linear interactions. The findings highlight the need for genuine cross-sectoral coordination and ecologically based rodent management near water bodies to sustainably reduce Fiji's disease burden.

Keywords: system dynamics; One Health; leptospirosis; Fiji; rodent ecology; water management



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1. Introduction

Leptospirosis is a neglected zoonotic disease caused by infection with pathogenic bacteria of the genus *Leptospira*. The disease presents significant public health challenges globally, particularly in tropical and subtropical regions [1]. Estimates suggest a worldwide annual burden of approximately 1.03 million cases and 58,900 deaths [2]. The incidence and severity of outbreaks are projected to increase due to rising temperatures and extreme weather events [3]. These shifts are driven by a complex interplay of climatic, socio-economic, and environmental conditions, further intensified by the compounding effects of climate change, flooding, and agricultural intensification [4,5].

Fiji experiences a high leptospirosis burden, with regular outbreaks often triggered by extreme weather events such as floods and cyclones [6,7]. In 2012, severe flooding resulted

in 576 reported cases with a case fatality rate of 7% [7]. More recently, from January to May 2022, 1528 confirmed cases were reported [8]. Despite the growing threat, accurately estimating morbidity and mortality remains challenging due to limitations in surveillance and diagnostic capacity [9].

Leptospira transmission is driven by complex interactions between animals, humans, and the environment. Most mammalian species, including rodents, horses, and livestock, act as reservoirs, shedding the bacteria through urine [10]. Humans are incidental hosts, with infection typically occurring through contact with infected animals or contaminated water and soil [1,11]. While progress has been made in understanding individual transmission components, existing research often focuses on single factors while neglecting broader systemic interactions [7,11,12]. For example, Bierque et al. [12] posited that environmental reservoirs are central drivers of *Leptospira* transmission. This perspective suggests a critical shift in policy focus, moving away from individual personal behaviours towards managing the ecological and environmental systems that sustain the pathogen [13].

A significant gap exists in understanding the synergy between water and rodents as drivers of transmission. While some research focuses on rainfall-induced runoff as the primary source of contamination [14,15], this study proposes that water acts as an “ecological enabler”. Rodents, particularly *Rattus norvegicus*, are strongly associated with riverine habitats where moisture supports both nesting and bacterial survival [16,17]. Thus, transmission is not merely a result of transient weather events but is sustained by the ecological relationship between standing water and rodent behaviour.

To address these research gaps, this study applies system dynamics (SD) modelling to simulate the dynamics of leptospirosis in Fiji. By adopting a One Health framework, which recognises the interdependence of human, animal, and environmental health, this research explores the feedback loops and interrelationships that drive incidence over time [18–20]. The study aims to identify and test critical leverage points to provide evidence-based recommendations for reducing the burden of leptospirosis in Fiji. Through this approach, the research identifies the interaction between rainfall and rodent ecology, with standing water as a key enabler, as the primary driver of infection.

2. Materials and Methods

2.1. Study Design and Modelling Approach

This study utilised an SD modelling approach to explore the complex, non-linear transmission of *Leptospira* in Fiji. The methodology followed Sterman’s [20] “systems method,” which consists of three primary stages: (1) problem articulation; (2) dynamic hypothesis formulation via a causal loop diagram (CLD) (Appendix A); and (3) formulation, testing, and application of a simulation model. A One Health framework was adopted to integrate the human, animal (rodent), and environmental domains into a single, cohesive quantitative structure [18,19].

2.2. Model Formulation and Subsystems

A pre-developed CLD (Appendix A) served as the foundation for the model formulation phase [13]. This conceptual model was developed through an iterative process to capture the key feedback mechanisms and circular causalities of *Leptospira* transmission in Fiji. Major inputs for the conceptual model included Bierque et al. [12], McPherson et al. [21], and Reid et al. [13]. The causal relationships identified in the CLD provided the structural basis for the development of the quantitative Stock and Flow Model, which was constructed using Vensim® DSS Version 10.3.1 (Appendix B) [22]. To maintain focus on the primary ecological drivers, qualitative feedback loops from the initial CLD regarding reactive public health responses, such as health communication and dynamic behaviour

change, were excluded from the quantitative simulation. Model structure and parametrisation were informed by Fiji-specific epidemiological data, systematic reviews, and historical records. In instances where empirical data were unavailable, specifically regarding certain environmental transmission pathways, parameters were based on logical assumptions derived from system behaviour and expert opinion. These assumptions were not arbitrary; they were constrained by logical limits and subsequently tested through rigorous sensitivity analysis to mitigate uncertainty. The model comprised three interconnected subsystems:

- **Human Sub-model:** This subsystem followed a susceptible–exposed–infected–recovered (SEIR) framework [23]. The susceptible population, based on Fiji’s average density of 50 people/km² [24], entered the transmission cycle where a proportion of the population became “exposed” via exposure to contaminated environmental stocks and infected rodents. This “infecting” flow was determined by the total infectious contacts, which combine the calculated risk from both the environmental pathway (contaminated soil and water) and the animal pathway (direct contact with infected rodents). It was assumed that 90% of the exposed population progressed to asymptomatic infection, while 10% developed symptomatic disease with a 5% case fatality rate [25,26].
- **Animal (Rodent) Sub-model:** Rodents (*Rattus* spp.) were identified as the primary biological reservoir. The model assumed a constant infected rodent fraction (26.7%) to determine the density of carriers within the population [27]. Rodent population dynamics were modelled based on a population dynamics framework [20] and driven by rainfall deviations, where increased rainfall supported births through improved resource availability [28,29]. The rodent subsystem “bridged” to the environment via a defined contamination fraction, where the rate of transition from clean to contaminated water stocks was proportional to the number of infected rodents and their respective contamination constants.
- **Environmental Sub-model:** This tracked the accumulation and decay of *Leptospira* in contaminated soil and water stocks. Water accumulation was determined by monthly rainfall and a runoff coefficient of 0.2 [30,31]. The soil system was modelled over a 1000 km² area, with an initial contamination level of 20%. Bacterial survival was set at 0.9 months for soil and 2 months for water [12]. In instances where empirical data were unavailable for specific environmental parameters, values were based on logical assumptions derived from system behaviour and expert opinion.

Full details on model inputs, equations, units, and references are provided in the Appendix C.

The model was simulated over a 24-month period with a 0.25-month (one week) time step to capture seasonal fluctuations in Fiji’s transmission cycle across the time period covered by the 2012 to 2013 leptospirosis outbreak in Fiji.

2.3. Model Validation and Calibration

Model validity was assessed through direct structure tests and behaviour pattern tests [20,32]. The model was calibrated by establishing a ‘base case’ that reproduced Fiji’s typical seasonal endemic curve, which peaks between December and February. Accuracy was further verified by successfully replicating the 2012 Western Division outbreak, using historical rainfall data and an adjusted population density of 53 people/km² [33].

2.4. Sensitivity Analysis and Extreme Scenario Testing

Model uncertainty was addressed using inbuilt functions in Vensim® DSS Version 10.3.1 [22]. Monte Carlo simulations (1000 iterations) simultaneously varied rodent mortality, environmental contact, and contaminated rainfall within a uniform distribution to

assess outcome variance. A tornado analysis individually varied $\pm 10\%$ from base case values to rank the relative influence on cumulative symptomatic cases. Finally, extreme scenario testing isolated high-leverage drivers (e.g., water contamination, rodent birth rate) by either removing (setting to zero) or amplifying variables to evaluate their specific impact on transmission dynamics compared to the established base case.

2.5. Intervention Scenario Design

Four multisectoral interventions were evaluated against the model's established base case. Intervention parameters were designed using conservative estimates informed by stakeholder analysis and existing policy frameworks in Fiji [21].

1. Rodent Control: A 15% increase in coordinated control effectiveness to reduce reservoir populations.
2. Personal Protective Equipment (PPE) Use: A 15% increase in compliance among agricultural and outdoor workers.
3. Water Management: A 10% increase in infrastructure effectiveness, such as improved drainage to reduce environmental contamination.
4. One Health Intervention: An integrated strategy combining all three individual measures to assess potential synergistic or additive effects.

2.6. Declaration of Generative AI

During the preparation of this study, generative artificial intelligence (AI) tools were employed to assist with modifying the tone and style of the written content. All intellectual decisions, critical analysis, synthesis of sources, and final technical wording remain the authors' own, and the final manuscript reflects the authors' personal academic work and understanding of the SD model.

3. Results

3.1. Model Subsystems

A high-level schematic overview of the model is presented in Figure 1, which illustrates the interconnected human (red), rodent (blue), and environment (green) subsystems. The SD model was developed based on the pre-existing CLD (Appendix A) to map the interconnections between these subsystems [13]. While Figure 1 provides the structural framework for the transmission pathways, the full, detailed model structure is further described in Appendix B. The components of the SD model, including stocks, flows, and auxiliary variables, are detailed in Appendix C.

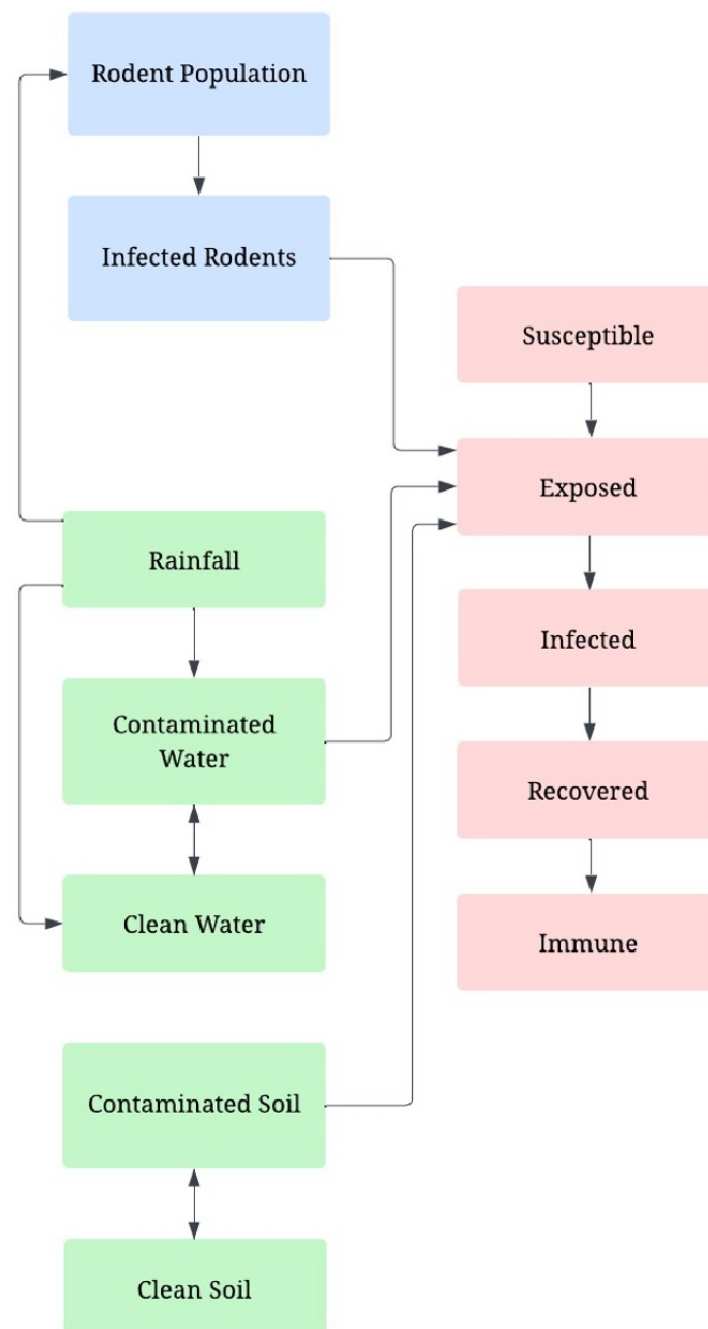


Figure 1. Schematic overview of the *Leptospira* transmission SD model, showing the human (red), rodent (blue), and environment (green) subsystems.

3.2. Model Validation and Behaviour Pattern Testing

The model successfully reproduced the oscillating annual trends of leptospirosis incidence in Fiji, with peak incidence typically occurring between December and February. Validation was further confirmed by replicating the 2012 Western Division outbreak. By adjusting rainfall data and increasing the population density to 53 people per km², the model successfully simulated the sharp case peak in March followed by a rapid decline by June, aligning with the real-world 2012 outbreak trajectory (Figure 2) [34]. Mass balance tests and dimensional consistency checks verified that no flows were unintentionally constructed or destroyed, ensuring the structural integrity of the model [20].

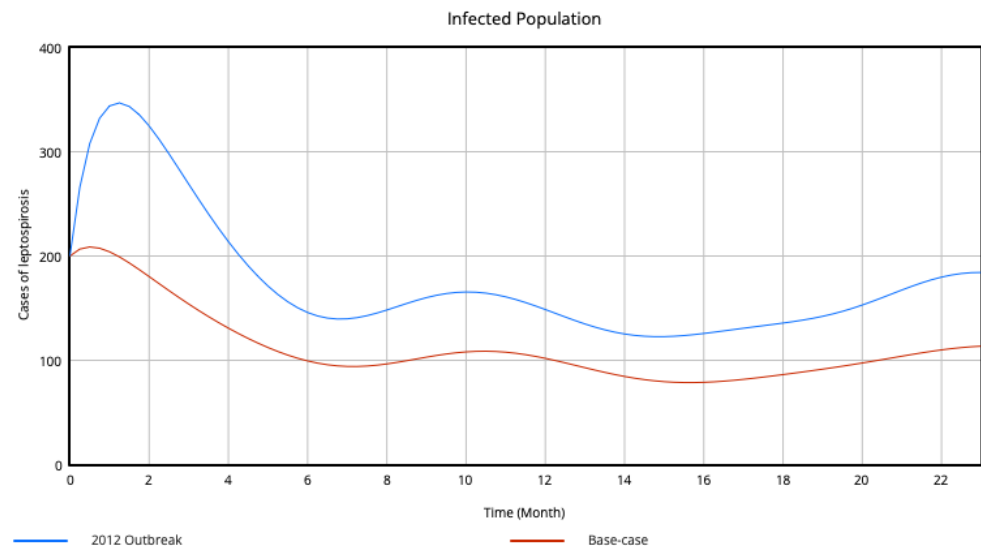


Figure 2. Incidence of symptomatic leptospirosis cases in the human population over two years, comparing the base case model (red) with a scenario approximating the 2012 leptospirosis outbreak in Fiji's Western Division (blue). Month 0 corresponds to January 2012.

3.3. Sensitivity Analysis

Two methods of sensitivity analysis were utilised to identify the primary drivers of uncertainty and transmission within the system.

3.3.1. Monte Carlo Sensitivity Simulation

A 1000-run simulation varied the rodent death rate, environmental contact rate, and contaminated rainfall fraction. The analysis revealed that uncertainty increased substantially over the simulation period. By month 22, symptomatic cases ranged from approximately 200 (50th percentile) to nearly 800 (100th percentile), indicating that small variations in rodent and environmental parameters produce increasingly divergent outcomes over time (Figure 3).

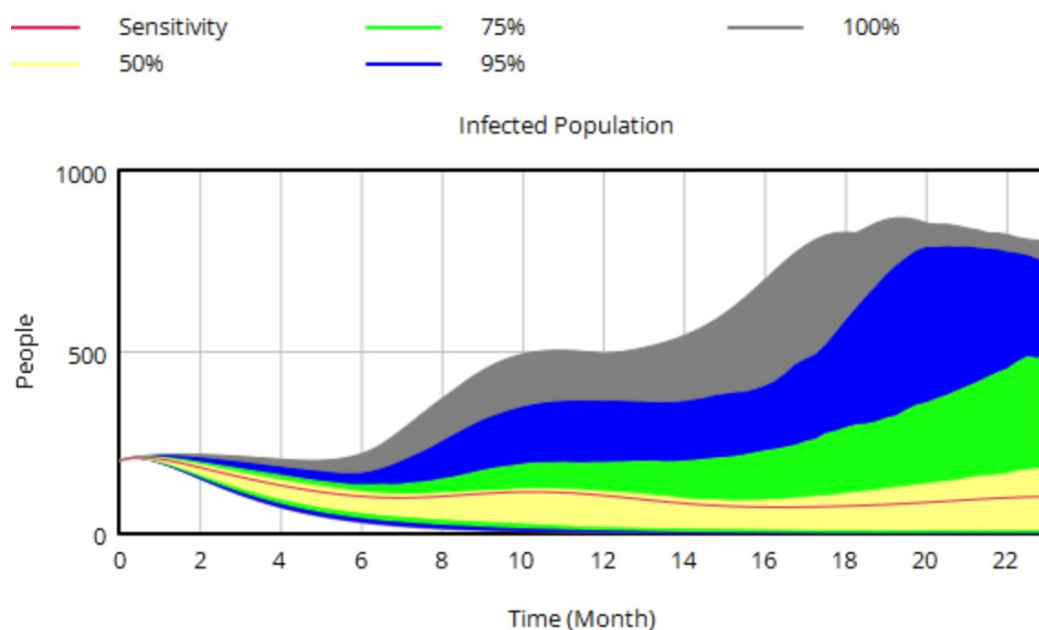


Figure 3. Monte Carlo sensitivity simulation modelled uncertainty in symptomatic leptospirosis cases due to variability in rodent death rate, environmental contact rate, and contaminated rainfall fraction.

3.3.2. Tornado Sensitivity Analysis

This analysis varied modifiable parameters by $\pm 10\%$ to rank their relative influence on cumulative symptomatic cases (Table 1). The results (Figure 4) identified rodent-related variables (e.g., rodents per litter, rodent birth rate, and rodent death rate) that produced the largest deviations in transmission outcomes. Environmental factors such as average rainfall and contact rates showed a moderate impact, while intervention-related variables like PPE use and water management demonstrated significantly smaller changes in total case numbers.

Table 1. Input values for key parameters varied in the tornado sensitivity plot.

Variable	−10% Value	Base Case	+10% Value	Reference
PPE Use	0.05	0.1	0.15	[21]
Environment contact rate	1800	2000	2200	Estimation
Water Management	0	0.1	0.2	[15]
Average rainfall	149.4	166	182.6	[35]
Rodent control measures	0.05	0.1	0.15	[21,36]
Rodent death rate	0.29	0.39	0.49	[37]
Rodent birth rate	0.15	0.25	0.3	[38]
Rodents per litter	3	4	5	[38]

Abbreviations: PPE = personal protective equipment.

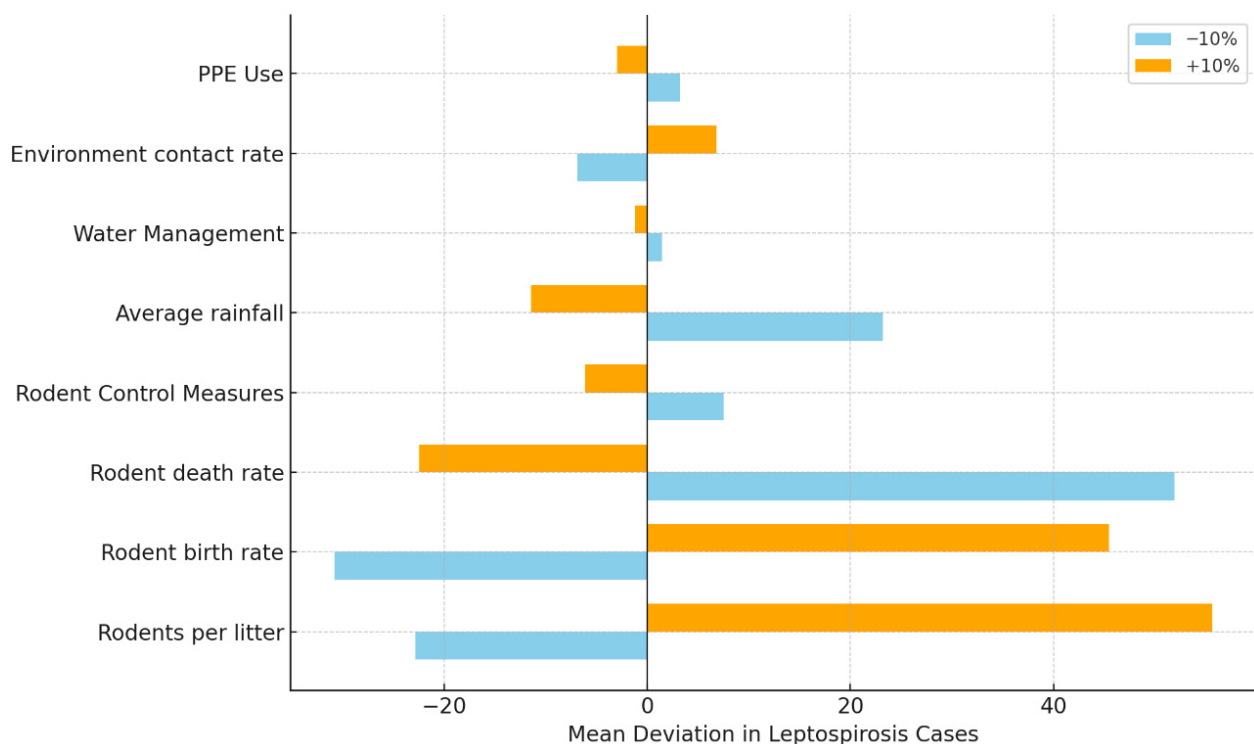


Figure 4. Tornado sensitivity plot illustrating the mean deviation in symptomatic leptospirosis cases resulting from $\pm 10\%$ changes in key model parameters. Abbreviations: PPE = personal protective equipment.

3.4. Extreme Scenario Testing: Identifying Key Drivers

Extreme parameter testing was conducted to isolate the role of specific animal, human, and environmental variables by either removing them or amplifying them by $+10\%$. The results of these simulations, compared against the predefined base case scenario, are presented in Figure 5.

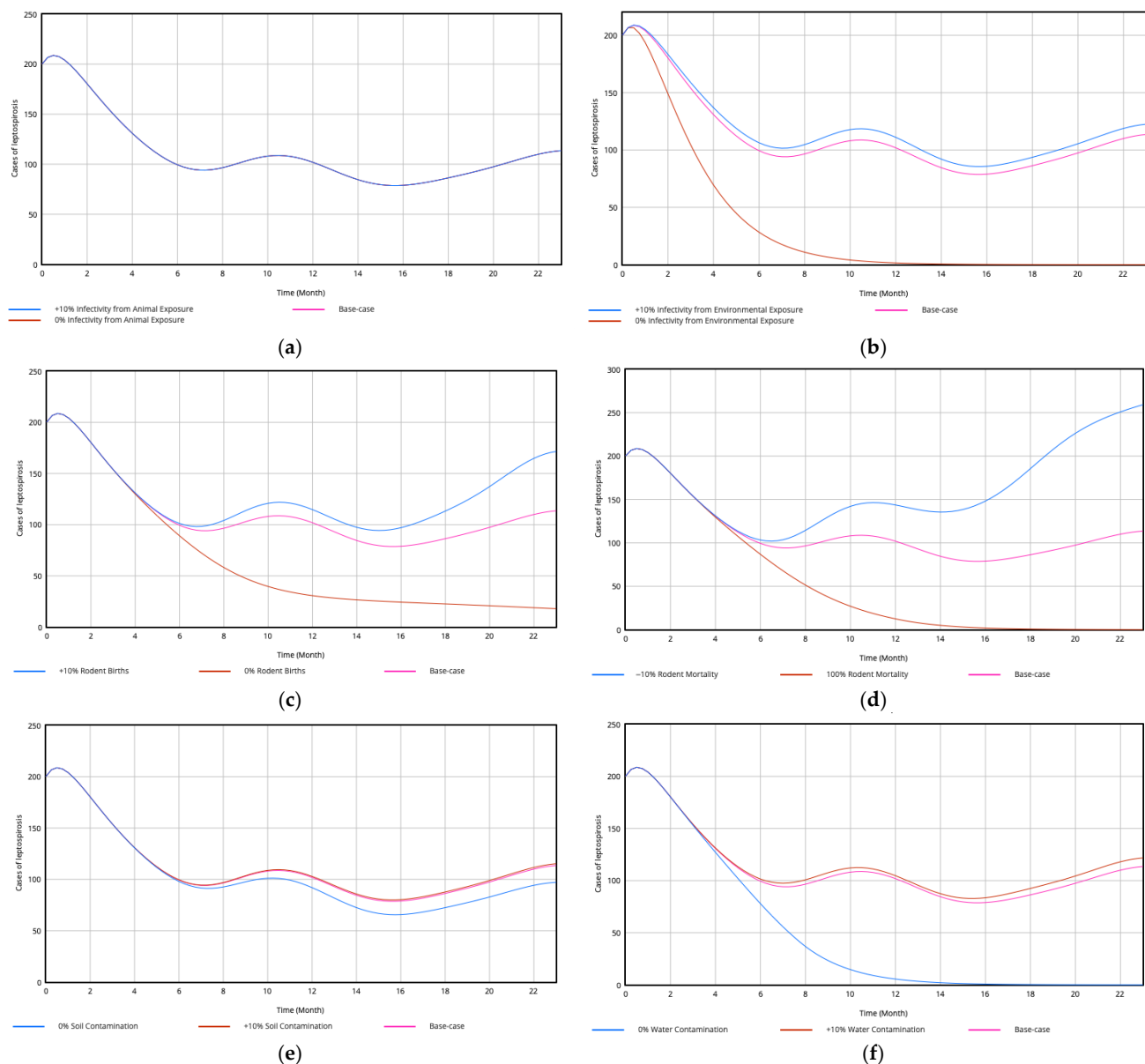


Figure 5. Impact of extreme scenario testing of human leptospirosis cases over time. (a) infectivity from animal exposure; (b) infectivity from environmental exposure; (c) rodent births; (d) rodent mortality; (e) soil contamination; (f) water contamination. Note: (a) base case and infectivity from animal exposure are superimposed.

- **Animal vs. Environmental Exposure:** Removing infectivity from environmental exposure drove cases toward zero by month 12 (Figure 5b). However, eliminating or increasing infectivity from direct animal exposure produced no notable differences compared to the base case (Figure 5a), suggesting direct contact is not a primary driver of overall transmission dynamics in this system.
- **Rodent Ecology:** Eliminating rodent births resulted in a sharp exponential decline until month 12 (Figure 5c), while increasing rodent mortality led to case numbers nearing zero by month 16 (Figure 5d). Increasing rodent births caused cases to rise substantially above the base case after month 17 (Figure 5c).
- **Water vs. Soil Contamination:** Eliminating water contamination caused a strong exponential decline in cases, approaching zero by month 14 (Figure 5f). Conversely, removing soil contamination led to only a minor reduction in cases, which continued to

follow the seasonal base case pattern (Figure 5e). This suggests soil is a less important driver of symptomatic disease compared to water.

3.5. Intervention Scenario Testing

Four intervention strategies were simulated and compared against the base case over the 24-month period (Table 2).

Table 2. Cumulative and stepwise reduction (%) in leptospirosis cases over 24 months under different intervention scenarios in Fiji.

Intervention Scenarios	Stepwise Reduction $\bar{R}_{step,t}$ (SD)	Cumulative Reduction
Rodent control	−17.49% (15.66)	−13.92%
PPE use	−8.82% (3.2)	−7.69%
Water management systems	−1.42% (1.13)	−1.13%
One Health	−25.51% (16.99)	−21.07%

Abbreviations: PPE = personal protective equipment, $\bar{R}_{step,t}$ = mean stepwise reduction (%) at time step t , standard deviation (SD).

- **One Health Intervention:** This integrated approach (combining rodent control, PPE, and water management) was the most effective, resulting in a 21.07% cumulative reduction in symptomatic cases.
- **Rodent Control:** A 15% increase in coordinated control effectiveness was the second most effective strategy, achieving a 13.92% reduction. Notably, both rodent control and One Health interventions helped prevent the resurgence of cases in the later months of the simulation, whereas other scenarios saw renewed increases.
- **PPE Use and Water Management:** Increased PPE use resulted in a modest 7.69% reduction, while improved water management systems yielded the smallest impact at 1.13%.

While the interventions reduced the magnitude of the incidence, all curves followed the same general temporal trend, with peaks and troughs corresponding to the underlying seasonal rainfall patterns (Figure 6). This indicates that the simulated interventions reduced the total burden but did not fundamentally alter the underlying transmission dynamics of the system. Furthermore, the effects of the One Health intervention were additive rather than synergistic, suggesting that the current leverage points operate independently.

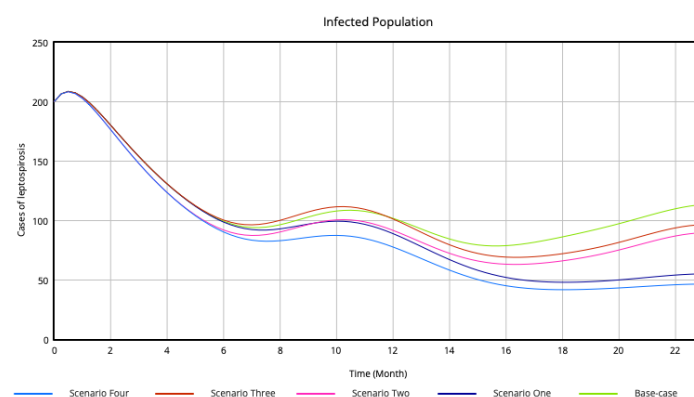


Figure 6. Simulated incidence of symptomatic human leptospirosis under proposed intervention scenarios in Fiji, including the base case. Scenario 1 = 15% increase in rodent control; Scenario 2 = 15% increase in PPE use; Scenario 3 = 10% increase in water management systems; Scenario 4 = combined 15% rodent control, PPE use +15%, and 10% water management systems.

4. Discussion

4.1. Model Validity and Theoretical Contributions

The SD model demonstrated strong alignment with real-world epidemiological trends, successfully reproducing the oscillating annual trends of reported leptospirosis cases in Fiji and the specific magnitude of the 2012 Western Division outbreak [34]. However, while the model captures the appropriate magnitude representing an outbreak, real-world peaks are often greater because the current model structure does not accommodate reporting bias [39]. In Fiji, reporting bias is frequently driven by surges in public awareness and intensive public health campaigns that typically accompany an outbreak. These campaigns lead to increased testing and case identification, producing a peak in surveillance data that is exacerbated by human behaviour rather than ecological drivers alone [40]. Because the model cannot currently calculate this behavioural feedback, it may not produce the exact intensified peaks seen in real-life records [39,41]. Despite this limitation, the model's base case provides a strong approximation of the outbreak data over the 24-month period, demonstrating particularly close alignment from month 4 onwards. This validation confirms the model's credibility in capturing complex disease transmission dynamics and reinforces its value for testing theoretical assumptions and identifying fundamental systemic patterns that drive the disease in Fiji.

While the initial conceptual model (CLD; Figure A1, Appendix A [13]) suggested soil and bacterial survival were critical nodes, the quantitative simulation revealed that these factors contribute to background behaviour but do not function as primary leverage points. This highlights the model's value in testing theoretical assumptions and shifting the focus from static conceptualisations of risk toward dynamic ecological feedback. This quantitative transition was essential because the initial CLD was informed by prior quantitative and qualitative research, including work that emphasised soil as a critical node in the transmission system to develop Fiji's national strategy [12,13,21]. By simulating these dynamics, the model proves that while soil is a necessary environmental reservoir, it is insufficient to sustain the transmission peaks observed in Fiji, thereby providing the evidence-based rigor required to shift policy toward the true system leverage points identified in this study.

4.2. The Synergy Between Water and Rodent Ecology

A central finding of this study is that standing water acts as an "ecological enabler" of transmission rather than a mere spatial correlate of risk. Previous research often describes river proximity as a risk factor without explaining the underlying mechanism [7]. This model clarifies that rodents, particularly *R. norvegicus* (the brown rat), are biologically tethered to riverine habitats where moisture supports both nesting and long-term bacterial survival [16,27,42]. This synergy explains why environmental exposure via water was identified as the most influential driver of transmission in the model [43,44]. The findings suggest that rainfall facilitates the spread of *Leptospira* through water and simultaneously drives rodent population surges, reinforcing the threat posed by climate change as the frequency of extreme weather events increases [4,13]. Crucially, the removal of water from the model drove cases toward zero within a year, whereas soil contamination had a negligible impact, appearing to contribute only when mediated by water saturation or flooding [9].

4.3. Challenging Prevailing Paradigms: Rodents vs. Livestock

The results challenge existing literature that identifies livestock as the primary driver of *Leptospira* in Fiji [45]. While spatial correlations often link livestock presence to human infection [14], the SD model indicates that rodent ecological behaviour is the more central biological driver. Rodents frequently co-occur with livestock in riverine environments

where water, food, and shelter are abundant [16,46]. Consequently, high livestock areas may simply be overlapping habitats for rodents, which act as the primary amplifiers within the shared environment [47,48]. This distinction is critical for public health, as it shifts the priority toward ecologically based rodent management (EBRM) rather than focusing solely on livestock or soil-based interventions [49–52].

4.4. Intervention Scenarios and the One Health Approach

Among the tested scenarios, the One Health intervention produced the greatest reduction in symptomatic cases (21.07%), confirming the value of multisectoral coordination [53]. However, the finding that results were additive rather than synergistic suggests that current leverage points operate independently within the system. Without genuine cross-sectoral integration—including shared planning and coordinated implementation—the potential for these interventions to reinforce one another remains constrained [53].

- **Rodent Control:** This was the second most effective strategy, yielding a 13.92% reduction. The model suggests that EBRM to disrupt breeding sites is more effective than reactive culling, as rodent birth rates and litter size have a greater cumulative impact than death rates alone [49,50].
- **PPE and Water Management:** These interventions were less effective (7.69% and 1.13% reductions, respectively). PPE use is often confined to occupational settings and does not address everyday environmental exposure [7]. Similarly, water management strategies often target runoff rather than the existing standing water that already sustains the rodent–pathogen cycle [47].

4.5. Cultural Relevance and Future Directions

Future interventions must bridge ecological risk with the cultural significance of water in Fiji. Water governance is deeply embedded in local social systems and spiritual practices [43,44,54]. Any strategy aiming to limit rodent access to water or reduce human contact with contaminated areas must be developed in partnership with local communities and water committees to ensure sustainable compliance.

4.6. Limitations

The model's use of a monthly time step limits its ability to capture dynamics occurring on shorter scales, such as the 12-day human incubation period or short-term bacterial survival fluctuations in soil [12,25,26]. Additionally, several environmental parameters were based on logical assumptions due to a lack of empirical data. While sensitivity analyses mitigated these uncertainties by identifying the most influential components, the model should be viewed as a conceptual tool for hypothesis testing rather than a precise predictive instrument.

5. Conclusions

This study provides a novel system-based framework for understanding and addressing the burden of leptospirosis in Fiji, advancing beyond static risk models by identifying dynamic ecological drivers and feedback loops. The central conclusion of this research is that *Leptospira* transmission in Fiji is fundamentally governed by the synergy between standing water and rodent ecology. This interaction reframes water not merely as a medium for rainfall-induced runoff but as a critical “ecological enabler” that supports both reservoir nesting habitats and the long-term environmental persistence of the pathogen.

The findings challenge prevailing assumptions in existing literature that prioritise soil contamination or livestock as primary drivers. Simulation results demonstrated that while soil may contribute to background environmental presence, it is insufficient to sustain

the transmission peaks observed in Fiji without the mediating role of water. Instead, transmission is a system-governed process shaped by climatic feedback where rainfall drives rodent population surges and maintains the standing water sources necessary for reservoir–host interaction.

From a policy perspective, the model provides a valuable decision-support tool for evaluating multisectoral interventions. While the One Health approach achieved the greatest reduction in symptomatic cases (21.07%), its effects were found to be additive rather than synergistic, suggesting that current interventions in Fiji operate as independent “parallel silos”. To achieve true synergy, future strategies must move toward genuine cross-sectoral integration, prioritising EBRM that targets habitat modification near critical water bodies. Ultimately, by bridging ecological risk with the sociocultural and cultural significance of water in Fiji, public health authorities can develop more sustainable, context-specific interventions to reduce the nation’s leptospirosis burden.

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Acknowledgments: During the preparation of this manuscript/study, the authors used Notebook LM for the purposes of superficial text editing, including grammar and formatting. The authors have reviewed and edited the output and take full responsibility for the content of this publication.

Conflicts of Interest: The authors declare no conflicts of interest.

Abbreviations

The following abbreviations are used in this manuscript:

SD	System Dynamics
CLD	Causal Loop Diagram
SEIR	Susceptible-exposed-infected-recovered
PPE	Personal Protective Equipment
EBRM	Ecologically Based Rodent Management
OR	Odds Ratio

Appendix A

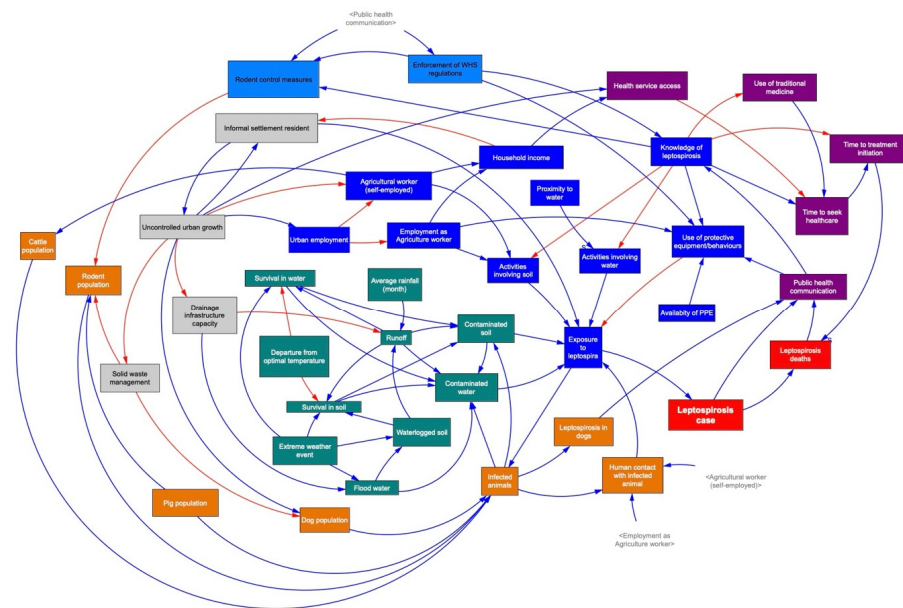


Figure A1. Causal loop diagram of the *Leptospira* transmission system in Fiji [13]. Colours indicate the subsystems: animal reservoir (orange; environmental transmission (green); socio-behavioural exposure (dark blue); health care access and response (purple); governance and response (light blue); urbanisation and infrastructure (grey).

Appendix B

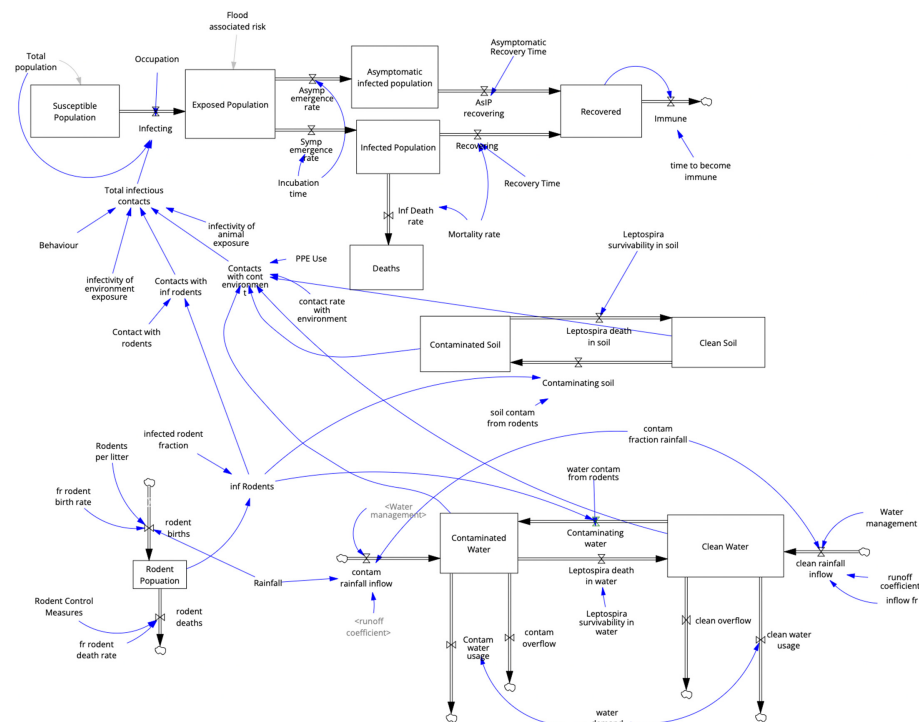


Figure A2. System dynamics model of Fiji's *Leptospira* transmission system, including human, animal, and environment subsystems. Grey arrows indicate inflows and outflow associated with system stocks, while blue arrows represent auxiliary variables within the model.

Appendix C

Table A1. Stocks, flows, and variables used in the model, and their type, units, equations, and values.

Variable Name	Type	Units	Equation/Value	Reference
Human Subsystem				
Susceptible Population	Stock	People	INTEG (Min (0, –Infecting), Total population – Initial asymptomatic population – Initial exposed population – Initial recovered population – Initial infected population)	
Exposed Population	Stock	People	INTEG (Infecting – Asymptomatic emergence rate – Symptomatic emergence rate, (IF THEN ELSE (Flood associated risk = 1, 1, 3.37)) × Initial exposed population)	
Asymptomatic infected population	Stock	People	INTEG (Max (0, Asymptomatic emergence rate – Asymptomatic infected recovering), Initial asymptomatic population)	
Infected Population	Stock	People	INTEG (Symptomatic emergence rate – Infected Death rate – Recovering, Initial infected population)	
Deaths	Stock	People	INTEG (Infected Death rate, 0)	
Recovered	Stock	People	INTEG (Asymptomatic infected recovering + Recovering – Immune, Initial recovered population)	
Infecting	Flow	People/month	IF THEN ELSE (Susceptible Population > 0, (IF THEN ELSE (Occupation = 1, 1, 0.35)) × Total infectious contacts × Susceptible Population ÷ Total population × UC3, 0)	
Asymptomatic infected recovering	Flow	People/month	Asymptomatic infected population ÷ Asymptomatic Recovery Time	
Symptomatic emergence rate	Flow	People/month	Max (0, (1 – Asymptomatic ratio) × Exposed Population ÷ Incubation time)	
Asymptomatic emergence rate	Flow	People/month	max (0, Asymptomatic ratio × (Exposed Population) ÷ Incubation time)	
Recovering	Flow	People/month	IF THEN ELSE (Infected Population > 0, Infected Population × (1 – Mortality rate) ÷ Recovery Time ÷ UC2, 0)	
Asymptomatic infected recovering	Flow	People/month	(Asymptomatic infected population ÷ Asymptomatic Recovery Time)	
Infected Death rate	Flow	People/month	IF THEN ELSE (Infected Population > 0, SMOOTH3 ((Infected Population × Mortality rate), stime2), 0)	
Immune	Flow	People/month	Recovered × time to become immune	[55]
Initial exposed population	Constant	People	1000	Estimation
Initial asymptomatic population	Constant	People	1000	Estimation
Initial infected population	Constant	People	200	Estimation
Initial recovered population	Constant	People	10,000	Estimation
Total population	Auxiliary	People	Population density × Land size ÷ km ² to m ² conversion	
Total infectious contacts	Auxiliary	Per month	SMOOTH3 ((IF THEN ELSE (Behaviour = 1, 1, IF THEN ELSE (Behaviour = 2, 2.5, IF THEN ELSE (Behaviour = 3, 4.5, 3)))) × (Contacts with contaminated environment × infectivity of environment exposure + Contacts with infected rodents × infectivity of animal exposure), 3)	
Contacts with contaminated environment	Auxiliary	Contacts/month	(Contaminated Water ÷ Clean Water Contaminated Soil ÷ Clean Soil) × (Contact rate with environment ÷ (1 + PPE Use))	
Contacts with infected rodents	Auxiliary	Contacts/month	Contact rate with rodents × Infected Rodents	
Population density	Constant	People/km ²	50	[24]
Infectivity of environment exposure	Constant	Per contact	0.50	Estimation
Infectivity of animal exposure	Constant	Per contact	0.00223	[27]
Contact rate with environment	Constant	Contacts/Month	2000	Estimation
Contact rate with rodents	Constant	Contacts/Month	10	Estimation
Behaviour	Constant	Dimensionless	1 = general population, 2 = swimming, 3 = fishing, 4 = walking barefoot	Swimming (OR 2.5), fishing (OR 4.5), and walking barefoot (OR 3.0) [11].

Table A1. Cont.

Variable Name	Type	Units	Equation/Value	Reference
Human Subsystem				
PPE Use	Constant	Dimensionless	0.10	[21]
Occupation	Constant	Dimensionless	1 = General public, 2 = indoor occupation	Indoor Occupation OR 0.35. [11]
Flood-associated risk	Constant	Dimensionless	1 = General population, 2 = flood-associated risk	3.37-fold increase in leptospirosis cases [34]
Incubation time	Constant	Month	1	[25]
Asymptomatic ratio	Constant	Dimensionless	0.90	[25]
Mortality rate	Constant	Per month	0.05	[25]
Asymptomatic Recovery Time	Constant	Month	3	Estimation
Recovery Time	Constant	Month	3	Estimation
Time to become immune	Constant	Per month	0.90	[25]
UC3	Constant	People	1	Unit Convertor
UC2	Constant	Per month	1	Unit Convertor
Rodent Subsystem				
Rodent Population	Stock	Rodents	INTEG (Rodent births – Rodent deaths, Initial Rodent Population)	
Rodent births	Flow	Rodents/month	(Rodent Population ÷ 2) × Rodents per litter × Rodent birth rate fraction × (Rainfall ÷ average rainfall)	
Rodent deaths	Flow	Rodents/month	Rodent death rate fraction × Rodent Population × (1 + Rodent Control Measures)	
Initial Rodent Population	Constant	Rodents	10	Estimation
Infected Rodents	Auxiliary	Dimensionless	(Rodent Population ÷ Initial Rodent Population) × Infected rodent fraction	
Rodents per litter	Constant	Rodents/litter	4	[38]
Rodent birth rate fraction	Constant	Litter/rodents/month	0.25	[38]
Rodent death rate fraction	Constant	Per month	0.30	[37]
Rodent Control Measures	Constant	Dimensionless	0.10	[21,36]
Infected rodent fraction	Constant	Dimensionless	0.267	[27]
Environment Subsystem				
Contaminated Soil	Stock	m ²	INTEG (Contaminating soil – Leptospira death in soil, Land size × 0.2)	[56]
Clean Soil	Stock	m ²	INTEG (Leptospira death in soil – Contaminating soil, Land size × 0.8)	
Contaminated Water	Stock	m ³	INTEG (contaminated rainfall inflow + Contaminating water-contaminated overflow – Contaminated water usage – Leptospira death in water, Water size × 0.2)	[56]
Clean Water	Stock	m ³	INTEG (clean rainfall inflow + Leptospira death in water – Clean overflow – Clean water usage – Contaminating water, Water size × 0.8)	
Leptospira death in soil	Flow	m ² /month	Contaminated Soil ÷ Leptospira survivability in soil × (1 – EXP (–TIME STEP ÷ stime3))	
Contaminating soil	Flow	m ² /month	Min (Clean Soil × UC2, max (0, Clean Soil × Infected Rodents × Soil contaminated from rodents))	
Contaminated rainfall inflow	Flow	m ³ /month	Rainfall × Contaminated fraction rainfall × Runoff coefficient × Land size × mm to m conversion ÷ (1 + Water management)	
Clean rainfall inflow	Flow	m ³ /month	Min (UC2 × (Water size – Clean Water), (inflow fraction × mm to m conversion × Rainfall × (1 – contaminated fraction rainfall) × runoff coefficient × Land size) ÷ (1 + Water management))	
Leptospira death in water	Flow	m ³ /month	SMOOTH3 (max (0, min ((Water size – Clean Water) × UC2, Contaminated Water)), Leptospira survivability in water)	
Contaminating water	Flow	m ³ /month	Min (Clean Water × UC2, Clean Water × Infected Rodents × Water contaminated from rodents)	
Contaminated water usage	Flow	m ³ /month	IF THEN ELSE (Contaminated Water > 0, min (water demand, Contaminated Water × UC2), 0) × Contaminated Water ÷ Clean Water	

Table A1. Cont.

Variable Name	Type	Units	Equation/Value	Reference
Human Subsystem				
Contaminated overflow	Flow	m ³ /month	IF THEN ELSE (Contaminated Water > Water size, Contaminated Water – Water size, 0) × UC2	
Clean water usage	Flow	m ³ /month	IF THEN ELSE (Clean Water > 0, min (Water demand, Clean Water × UC2) × (1 – Contaminated Water ÷ Max (Clean Water + Contaminated Water, 1 × 10 ^{−6})), 0)	
Clean overflow	Flow	m ³ /month	IF THEN ELSE (Clean Water > Water size, Clean Water – Water size, 0) × UC2	
Rainfall	Lookup Table	mm/month	WITH LOOKUP (Rainfall time, ((0, 90) – (24, 400)], (0, 216), ..., (24, 191)))	[31]
Rainfall time	Stock	Month	INTEG (1, 0)	
Land size	Constant	m ²	1.00 × 10 ⁹	Land size. 1000 km ²
Water size	Constant	m ³	1.30 × 10 ⁸	[57]
Leptospira survivability in soil	Constant	Month	0.90	[12]
Soil contaminated by rodents	Constant	Per month	0.025	Estimation
Leptospira survivability in water	Constant	Month	2	[12]
Water contaminated by rodents	Constant	Per month	0.50	[16]
Runoff coefficient	Constant	Dimensionless	0.20	[30]
Average rainfall	Constant	mm/month	207	[35]
Inflow fraction	Constant	Dimensionless	0.07	Estimation
Contaminated fraction rainfall	Constant	Dimensionless	0.005	Estimation
Water demand	Constant	m ³ /month	228,061	[58]
Water management	Constant	Dimensionless	0.10	[15]
mm to m conversion	Constant	m/mm	0.001	Unit Convertor
km ² to m ² conversion	Constant	m ² /km ²	1 × 10 ⁶	Unit Convertor
stime3	Constant	Month	5	Time step
stime2	Constant	Dimensionless	2	Time step

Bold text indicates each subsystem in the model. Abbreviations: INTEG = integer, EXP = exponential.

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