



A NEUTROSOPHIC SIR EPIDEMIC MODEL: GENERALIZED HUKUHARA DIFFERENTIABILITY, (α, β, γ) -CUT ANALYSIS, AND NUMERICAL SIMULATION UNDER EPISTEMIC UNCERTAINTY

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Abstract

Epidemiological surveillance data collected during the early stage of an outbreak are rarely free of doubt: case counts are revised, contact-tracing records are incomplete, and a share of reports remain formally unclassified, a pattern documented explicitly during the 2014–2016 West Africa Ebola outbreak, when the World Health Organization stopped consistently reporting probable and suspected case counts because of high reclassification rates. Fuzzy and intuitionistic fuzzy differential equations model vagueness in such parameters but cannot separately track how much of the uncertainty is indeterminate rather than simply "false." This paper develops a Susceptible–Infected–Recovered (SIR) epidemic model in which the transmission rate and the recovery rate are represented as single-valued triangular neutrosophic numbers, so that truth, indeterminacy, and falsity in the parameter estimates are carried independently through the dynamics. We recall the algebra of triangular neutrosophic numbers and their (α, β, γ) –cuts, define the model through the generalized Hukuhara neutrosophic derivative, prove existence and uniqueness for the resulting family of classical systems, and prove in closed form that the epidemic peak and the final attack rate are monotonic in the transmission and recovery rates, which is what rigorously justifies reading the computed cut families as genuine bounds. A corner-point (α, β, γ) -cut solution procedure is proposed and

solved with an adaptive Runge–Kutta scheme on parameters grounded in published country-level reproduction-number estimates from the West Africa Ebola outbreak. Truth-, indeterminacy-, and falsity-cut families are all computed numerically, showing how the epidemic peak, its timing, the final attack rate, and the effective reproduction number widen or narrow independently along each of the three membership dimensions, and that the indeterminacy- and falsity-cut bounds diverge from one another even at matched confidence levels a distinction no fuzzy or intuitionistic fuzzy model can represent. The results indicate that the neutrosophic formulation keeps the full three-way structure of the uncertainty in the surveillance data while remaining computationally no more demanding than a comparable fuzzy model, and that it collapses to the classical deterministic SIR model at the maximal-confidence limit of every cut, giving a consistent basis for comparison with existing crisp and fuzzy epidemic models.

Keywords: neutrosophic differential equations; neutrosophic numbers; (α, β, γ) -cut; generalized Hukuhara derivative; SIR epidemic model; epistemic uncertainty.

I. Introduction

Compartmental models such as the Susceptible–Infected–Recovered (SIR) system remain the workhorse of mathematical epidemiology, and their qualitative behaviour under crisp, constant parameters is well understood [XIV].

The transmission and recovery rates that enter these models, however, are not observed directly; they are inferred from case reports, contact-tracing interviews, and laboratory confirmations, which are themselves incomplete or contradictory, particularly in the first weeks of an outbreak. A substantial literature has therefore replaced crisp parameters with imprecise ones. Building on Zadeh's fuzzy sets [XXXI], fuzzy differential equation calculus [XIII, VIII, X] has been used to construct fuzzy-parameter SIR and SEIR models: fuzzy transmission and recovery rates have been used for measles and other viral epidemics [XVIII], for COVID-19 in Indonesia with fuzzy infection, recovery, and death rates [I], and combined with a numerical collocation scheme in a Hybrid SIR-Fuzzy model [VI]. Intuitionistic fuzzy extensions [VII] go further and separately track a degree of membership and a degree of non-membership; this has recently been used to build an intuitionistic fuzzy SEIR model with triangular intuitionistic fuzzy parameters [XVII] and an intuitionistic fuzzy differential equation model of a lake phosphorus system [II]. Fractional-order SIR models with a Caputo derivative and delay effects have separately been used to capture memory effects in transmission dynamics [XXIII], though without any parameter-uncertainty structure.

None of these formulations, however, can represent a third, genuinely distinct component of the uncertainty: the proportion of a report that is simply indeterminate, neither confirmed nor ruled out, rather than false. Smarandache's neutrosophic sets [XXIV] and their single-valued form [XXIX] address exactly this gap by attaching independent truth, indeterminacy, and falsity membership functions to a quantity. The resulting neutrosophic numbers and their calculus have been used to formulate first-order ordinary differential equations and their (α, β, γ) -cut solution [XXV], extended to trapezoidal neutrosophic numbers [XXVI], to second-order neutrosophic

boundary-value problems [IX], to neutrosophic homogeneous difference equations [V], and to a neutrosophic-derivative formulation of linear differential equations [XX]; a fixed-point existence-and-uniqueness theory for neutrosophic differential equations has also been developed and applied to an economic order-quantity problem [XXI]. The neutrosophic framework has also been developed beyond the differential-equation setting, into bipolar neutrosophic soft topological structures [XV] and into neutrosophic graph theory, including bipolar distance and detour-distance measures on neutrosophic graphs [XXVIII, XXII], which together indicate the breadth of structures the neutrosophic calculus has been extended to, though none of this topological or graph-theoretic line addresses a differential equation or a biological/epidemic application. Within biology specifically, a neutrosophic differential equation has been used to model glucose distribution in the bloodstream [III], and a neutrosophic fuzzy framework has been used for the stability analysis of a diabetes mellitus model [IV] — both confirming that the neutrosophic calculus extends cleanly to biological systems, but neither addresses a compartmental, population-level epidemic model. None of this line of work, to the best of our knowledge, has yet been applied to a compartmental epidemic model.

This paper closes that gap. We (i) formulate a neutrosophic SIR model in which the transmission and recovery rates are triangular neutrosophic numbers, (ii) use the (α, β, γ) -cut representation together with the generalized Hukuhara neutrosophic derivative to reduce the model to families of classical initial value problems, (iii) state existence-and-uniqueness conditions for the resulting neutrosophic system, and (iv) illustrate the model numerically along all three cut families, truth, indeterminacy and falsity, reporting how each maps into bounds on the epidemic peak, its timing, the final attack rate and the effective reproduction number. The maximal-confidence limit of every cut recovers the classical deterministic SIR trajectory exactly, which gives a natural point of comparison with existing crisp and fuzzy epidemic models.

II. Preliminaries

II.i. Single-valued triangular neutrosophic numbers

A single-valued triangular neutrosophic number is written

$$\tilde{A} = \langle (a1, a2, a3); w\tilde{A}, u\tilde{A}, y\tilde{A} \rangle, \quad (1)$$

where $a1 \leq a2 \leq a3$ are real numbers and $w\tilde{A} \in [0,1], u\tilde{A}, y\tilde{A} \in [0,1]$ are, respectively, the maximum truth-membership degree and the minimum indeterminacy- and falsity-membership degrees attainable at the core value $a2$. Its truth-, indeterminacy-, and falsity-membership functions $T\tilde{A}, I\tilde{A}, F\tilde{A} : \mathbb{R} \rightarrow [0,1]$ are piecewise linear:

$T\tilde{A}(x)$ rises linearly from 0 to $w\tilde{A}$ on $[a1, a2]$ and falls linearly from $w\tilde{A}$ to 0 on $[a2, a3]$;

$I\tilde{A}(x)$ falls linearly from 1 to $u\tilde{A}$ on $[a1, a2]$ and rises linearly from $u\tilde{A}$ to 1 on $[a2, a3]$;

$F\tilde{A}(x)$ falls linearly from 1 to $y\tilde{A}$ on $[a1, a2]$ and rises linearly from $y\tilde{A}$ to 1 on $[a2, a3]$;

and $T\tilde{A}(x) = I\tilde{A}(x) = F\tilde{A}(x) = 0$ (respectively 1, 1) outside $[a1, a3]$, consistent with the definitions used in the earlier neutrosophic differential equation literature [XXIV, XXIX, XXV].

II.ii (α, β, γ) -cuts

For $\alpha \in [0, w\tilde{A}]$, $\beta \in [u\tilde{A}, 1]$, and $\gamma \in [y\tilde{A}, 1]$, the (α, β, γ) -cut of $\tilde{A}$ is the crisp set

$$\tilde{A}(\alpha, \beta, \gamma) = \{x \in \mathbb{R}: T\tilde{A}(x) \geq \alpha, I\tilde{A}(x) \leq \beta, F\tilde{A}(x) \leq \gamma\}. \quad (2)$$

For the piecewise-linear number in (1), each membership type generates a closed interval. For the truth-cut,

$$AT(\alpha)L = a1 + \left(\frac{\alpha}{w\tilde{A}}\right)(a2 - a1) \text{ and}$$

$$AT(\alpha)U = a3 - \left(\frac{\alpha}{w\tilde{A}}\right)(a3 - a2).$$

Because $I\tilde{A}$ and $F\tilde{A}$ are valley-shaped rather than tent-shaped, their cuts run in the opposite sense: solving $T\tilde{A}(x) \geq \alpha$ is replaced by solving $I\tilde{A}(x) \leq \beta$ (respectively

$F\tilde{A}(x) \leq \gamma$), which for the piecewise-linear form above gives

$$AI(\beta)L = a1 + (a2 - a1) \cdot \frac{1 - \beta}{1 - u\tilde{A}},$$

$$AI(\beta)U = a2 + (a3 - a2) \cdot \frac{\beta - u\tilde{A}}{1 - u\tilde{A}}, \quad (3)$$

and the falsity-cut interval $AF(\gamma)L$, $AF(\gamma)U$ follows the same formula with $y\tilde{A}$ in place of $u\tilde{A}$. All three intervals shrink monotonically to the singleton $\{a2\}$ as

$\alpha \rightarrow w\tilde{A}, \beta \rightarrow u\tilde{A}, \gamma \rightarrow y\tilde{A}$, and widen monotonically to the full support $[a1, a3]$ as

$\alpha \rightarrow 0, \beta \rightarrow 1, \gamma \rightarrow 1$, the property used below both to recover the crisp SIR model as a limiting case and to compute the three cut families numerically in Section IV.

II.iii. Generalized neutrosophic Hukuhara derivative

Let $\tilde{x}(t)$ be a neutrosophic-valued function of t on an interval J , and let $[xT(\alpha)L(t), xT(\alpha)U(t)]$, $[xI(\beta)L(t), xI(\beta)U(t)]$ and $[xF(\gamma)L(t), xF(\gamma)U(t)]$

denote its three families of (α, β, γ) -cut intervals. Following the generalized Hukuhara construction used for fuzzy- and neutrosophic-valued functions [XIII, VIII, IX, XX], $\tilde{x}$ is said to be generalized Hukuhara differentiable at t_0 if each of the six boundary functions is differentiable at t_0 and the resulting derivatives again describe valid (α, β, γ) -cuts of a neutrosophic number $\tilde{x}'(t_0)$, for every admissible α, β, γ . As in the fuzzy case [XIII, VIII], two systems of ordinary differential equations an (i)-differentiable system in which the cut-intervals of $\tilde{x}$ are non-decreasing in width, and an (ii)-differentiable system in which they are non-increasing- must in general be checked separately.

For the construction actually used in this paper (defined precisely in Section 3.2 and computed in Section 3.3), the boundary trajectories SL , SU , IL , IU , RL , RU are the pointwise minimum and maximum, over four corner parameter pairs, of classical crisp SIR trajectories, so the cut-width functions,

$$\Delta S(t) = SU(t) - SL(t),$$

$$\Delta I(t) = IU(t) - IL(t),$$

$$\Delta R(t) = RU(t) - RL(t)$$

are each a difference of two independently computed, population-conserving trajectories, and (i)-differentiability holds at an instant t precisely when all three widths are non-decreasing there. Rather than assert this sign condition a priori from the initial condition alone, we check it directly: for the Ebola-grounded parameter ranges used in Section IV, $\Delta I(t)$ is confirmed numerically to be non-negative throughout $[0, 150]$ days for every (α, β, γ) level reported in Tables 1–3 (Section 3.3 records this check). We do not claim this holds for every parameter regime or for $t \rightarrow \infty$: in the long-time tail, as all four corner trajectories converge toward their respective disease-free states, $\Delta I(t)$ could in principle narrow again before vanishing, so the (i)-differentiable interpretation used here is understood to apply on the finite horizon $[0, 150]$ days actually simulated, and is not asserted as a global, all-time property of the model. A rigorous classification of any switching point between (i)- and (ii)-differentiability over an infinite horizon is left as an open problem (Section VI).

III. Proposed Neutrosophic SIR Model

III.i. Model formulation

Let $S(t), I(t), R(t) \geq 0$ denote the susceptible, infected, and recovered fractions of a closed population of size N , with $S(t) + I(t) + R(t) = N$. We replace the transmission rate and the recovery rate of the classical SIR model [XIV] by triangular neutrosophic numbers $\tilde{\beta}$ and $\tilde{\gamma}$, reflecting epistemic uncertainty, contradiction and indeterminacy in the surveillance estimates of these two rates, and define the neutrosophic SIR system

$$\frac{d\tilde{S}}{dt} = \frac{-\tilde{\beta}\tilde{S}\tilde{I}}{N}, \quad \frac{d\tilde{I}}{dt} = \frac{\tilde{\beta}\tilde{S}\tilde{I}}{N} - \tilde{\gamma}\tilde{I}, \quad \frac{d\tilde{R}}{dt} = \tilde{\gamma}\tilde{I}, \quad (4)$$

with crisp, deterministic initial conditions $S(0) = S_0, I(0) = I_0, R(0) = 0$, since the state of the population at $t = 0$ is taken to be known exactly and only the two rate parameters carry neutrosophic uncertainty.

III.ii. (α, β, γ) -cut system and existence–uniqueness

Fix a level (α, β, γ) and write $[bL, bU]$ and $[gL, gU]$ for the truth-, indeterminacy-, or falsity-cut intervals of $\tilde{\beta}$ and $\tilde{\gamma}$ at that level, using (3) (the truth-cut uses the mirror-image formula given in Section 2.2). Under (i)-differentiability, applying the generalized Hukuhara derivative componentwise to (4) yields the two-sided crisp system,

$$\begin{aligned} \frac{dSL}{dt} &= -bU SU \frac{IU}{N}, & \frac{dSU}{dt} &= -bL SL \frac{IL}{N}, \\ \frac{dIL}{dt} &= bL SL \frac{IL}{N} - gU IU, & \frac{dIU}{dt} &= bU SU \frac{IU}{N} - gL IL, \end{aligned}$$

$$\frac{dRL}{dt} = gL IL, \quad \frac{dRU}{dt} = gU IU. \quad (5)$$

System (5) is nonlinear because of the S·I coupling term, and it matters precisely which object's existence, uniqueness, and invariance are proved for. We first establish these properties for the four corner systems that are the ones actually integrated numerically (Section 3.3), and then explain why system (5) itself, as a single coupled six-dimensional system, is not claimed to be invariant on the box below, correcting an overstatement in an earlier version of this paper.

For a fixed corner parameter pair $(b, g) \in \{(bL, gL), (bL, gU), (bU, gL), (bU, gU)\}$, the corresponding trajectory solves the classical crisp SIR system

$$\begin{aligned} \frac{dS}{dt} &= -\frac{bSI}{N}, \\ \frac{dI}{dt} &= \frac{bSI}{N} - gI, \\ \frac{dR}{dt} &= gI, \quad \text{with } S(0) = S_0, I(0) = I_0, R(0) = 0. \end{aligned}$$

Summing the three equations gives

$$\frac{d(S+I+R)}{dt} = 0,$$

so $S(t) + I(t) + R(t) = N$ exactly for all t , and the closed simplex $\Delta = \{(S, I, R) \in [0, N]^3 : S + I + R = N\}$ is positively invariant:

$$\text{on } S = 0, \frac{dS}{dt} = 0; \text{ on } I = 0, \frac{dI}{dt} = 0; \text{ on } R = 0, \frac{dR}{dt} = gI \geq 0;$$

so no trajectory starting in Δ can leave it. The right-hand side is polynomial, hence locally Lipschitz, on the compact set Δ , so the Picard–Lindelöf theorem gives a unique local solution through $(S_0, I_0, 0)$, and positive invariance of the compact set Δ extends it to a unique global solution on $[0, \infty)$. This is standard SIR theory [XIV] and holds without qualification for each of the four corner systems individually.

System (5) is the formal result of applying the generalized Hukuhara derivative componentwise to (4), and is included for conceptual completeness: it correctly reproduces the four corner systems as the special cases $SL = SU$, $IL = IU$, etc.

However, as a single coupled six-dimensional system, in which $\frac{dSL}{dt}$ is driven by the cross term $bU \cdot SU \cdot IU$ while SL , SU , IL , IU evolve under four separately coupled equations rather than a shared conservation law, the box

$$\Omega = \{0 \leq SL \leq SU \leq N, 0 \leq IL \leq IU \leq N, 0 \leq RL \leq RU \leq N\}$$

is not positively invariant under (5): the cross term $bU \cdot SU \cdot IU$ that drives SL is not bounded by N once SU and IU are permitted to evolve independently, so the inward-pointing argument given in an earlier version of this paper does not in fact hold, and a direct Lipschitz/Picard–Lindelöf argument on Ω is not valid for the coupled system. This is exactly the underlying cause of the numerical instability reported in Section 3.3: direct Runge–Kutta integration of (5) producing $IU(t)$ exceeding N and $RL(t)$

becoming negative once the parameter interval is wide is the observable signature of Ω failing to be invariant under the coupled vector field, and the earlier theoretical invariance claim is withdrawn accordingly.

We therefore do not use system (5) as the object that is solved, and do not claim existence, uniqueness, or invariance for it as a coupled system. The solution concept adopted throughout this paper, consistent with the established practice in the fuzzy differential equations literature of using an alternative, well-behaved construction when the direct generalized-Hukuhara system is analytically or numerically ill-behaved (see the switching-point and generalized-differentiability discussions in [XIII, VIII]), is defined operationally as the pointwise envelope of the four independently well-posed corner trajectories established above: $SL(t) := \min$, $SU(t) := \max$ over the four corners of $S(t)$, and likewise for I and R . Proposition 1 below proves that, for the peak-size and final-size quantities actually reported in Section IV, this envelope is rigorously correct; Section 3.3 additionally verifies numerically, across the full parameter grid used in Tables 1–3, that $IU(t) \geq IL(t)$ with no crossing throughout the simulated horizon, consistent with, but not a substitute for, a full analytic proof of pointwise envelope equivalence at every t , which we identify as an open problem in Section VI.

Proposition 1 (monotonicity of the peak and final size). For the classical SIR system with constant parameters (β, γ) , $S_0 > 0, I_0 > 0$, and basic reproduction number

$R_0 = \frac{\beta S_0}{\gamma N} > 1$, the peak infected count and the final epidemic size are given in closed form by

$$I_{peak}(\beta, \gamma) = S_0 + I_0 - \rho + \rho \ln\left(\frac{\rho}{S_0}\right), \quad \rho = \frac{\gamma N}{\beta}, \quad (5a)$$

and by the implicit final-size relation

$$\ln(S_0/S_\infty) = R_0(1 - S_\infty/N), \text{ where } S_\infty = \lim_{t \rightarrow \infty} S(t)$$

and the final size is $N - S_\infty$ (both standard results for the classical SIR model; see [XIV]).

Differentiating (5a) with respect to ρ gives

$$\frac{dI_{peak}}{d\rho} = -1 + \ln\left(\frac{\rho}{S_0}\right) + \rho \cdot \left(\frac{1}{\rho}\right) = \ln\left(\frac{\rho}{S_0}\right),$$

which is strictly negative whenever $\rho < S_0$, i.e. whenever $R_0 = \frac{S_0}{\rho} > 1$.

Since $\rho = \frac{\gamma N}{\beta}$ is strictly increasing in γ and strictly decreasing in β , I_{peak} is strictly increasing in β (fixed γ) and strictly decreasing in γ (fixed β), throughout the outbreak regime $R_0 > 1$.

Applying the implicit function theorem to the final-size relation gives

$$\frac{dS_\infty}{dR_0} = \frac{\left(1 - \frac{S_\infty}{N}\right)}{\left(\frac{R_0}{N} - \frac{1}{S_\infty}\right)},$$

since $S_\infty < \frac{N}{R_0}$ (the final susceptible fraction lies below the epidemic threshold), the denominator $\frac{R_0}{N} - \frac{1}{S_\infty}$ is strictly negative while the numerator $1 - \frac{S_\infty}{N}$ is strictly positive, so $\frac{dS_\infty}{dR_0} < 0$ for all $R_0 > 1$, and the final size $N - S_\infty$ is strictly increasing in R_0 , hence strictly increasing in β and strictly decreasing in γ .

Proposition 1 is an unconditional, closed-form result: it does not rely on the pointwise-in-time ordering of $I(t; \beta, \gamma)$ across different parameter values, only on the two scalar summary quantities Tables 1–3 actually report. It is these two monotonicities, not a general comparison theorem for $I(t)$ at every t , that rigorously justify reading the corner values in Tables 1–3 as upper and lower bounds on the peak infected count and the final attack rate. The stronger, pointwise-in-time statement used in Section 3.3 to motivate the corner-point construction, that $I(t; \beta, \gamma)$ itself is ordered at every t , not only at its peak and its limit, is verified numerically across the full simulated horizon and parameter grid (Section 3.3) but is not claimed to follow from Proposition 1 alone and is not proved here in general; this is flagged explicitly as a scope limitation in Section VI.

Two structural checks confirm that the corner-envelope construction is a sensible solution concept for (4).

First, at the maximal-confidence limit of any cut ($\alpha \rightarrow w\tilde{A}, \beta \rightarrow u\tilde{A}, \text{ or } \gamma \rightarrow y\tilde{A}$)

the corresponding parameter interval collapses to a single point ($bL = bU, gL = gU$), so all four corner trajectories coincide and $SL = SU, IL = IU, RL = RU$: the construction reduces exactly, and trivially, to the classical crisp SIR system.

Second, Proposition 1 establishes in closed form that the peak and final-size functionals of the corner trajectories are correctly ordered whenever $bU \geq bL$ and $gU \geq gL$, so the truth-, indeterminacy- and falsity-cut envelopes reported in Section IV are genuine, provable bounds on these two summary statistics, even though, as discussed above and in Sections 2.3 and 3.3, the stronger pointwise-in-time ordering of the full trajectories $IL(t)$ and $IU(t)$ is verified numerically on the simulated horizon rather than proved in general.

III.iii. Solution procedure

System (5) still couples the lower and upper trajectories through products of state variables, which makes a direct Runge–Kutta discretisation of the six-equation system prone to instability once $IU(t)$ is pushed far from $IL(t)$: consistent with the non-invariance of Ω under (5) established in Section 3.2, an early implementation of this model that integrated (5) directly produced unbounded, population-violating trajectories ($IU(t)$ growing past N with $RL(t)$ driven negative) once the parameter interval was wide. We therefore adopt, as defined in Section 3.2, the corner-point envelope as the solution concept used throughout this paper: for each (α, β, γ) –cut box

$$[bL, bU] \times [gL, gU], SL(t), SU(t), IL(t), IU(t), RL(t), RU(t)$$

are the pointwise minimum and maximum, over the four corner parameter pairs $(bL, gL), (bL, gU), (bU, gL), (bU, gU)$, of the corresponding classical crisp SIR

trajectories, each a standard, population-conserving three-equation system integrated with an explicit adaptive Runge–Kutta method (fourth-to-fifth order, absolute and relative tolerances of 10^{-9}).

Proposition 1 (Section 3.2) proves rigorously that this envelope gives correct lower and upper bounds for the two scalar quantities actually tabulated in Section IV, the peak infected count and the final epidemic size, since both are monotonic in β and γ in closed form. For the pointwise-in-time trajectories themselves, we additionally checked numerically, across every (α, β, γ) level and all three cut families reported in Tables 1–3, that the corners (bL, gU) and (bU, gL) produce the lower and upper envelope of $I(t)$ at every tabulated time point on $[0, 150]$ days, with no crossing observed. This is reported as a verified numerical property of the specific case study in Section IV, not as a general theorem for arbitrary SIR parameter regimes; a full analytic comparison-theorem proof of pointwise ordering for the general nonlinear SIR system is left as an open problem (Section VI).

IV. Numerical Simulation and Results

Case study: parameters grounded in the 2014 West Africa Ebola outbreak

Rather than choose the transmission and recovery rates arbitrarily, we ground both triangular neutrosophic numbers in published estimates from the 2014–2016 West Africa Ebola virus disease (EVD) epidemic, which is also a natural application for the neutrosophic framework: throughout the outbreak, WHO and CDC situation reports classified cases as confirmed, probable or suspected [XIX], and from November 2014 onward WHO stopped consistently publishing the probable/suspected breakdown because a large share of those cases were repeatedly reclassified, a documented, real instance of genuine reporting indeterminacy rather than simple measurement error [XI].

The WHO Ebola Response Team's analysis of the first nine months of the epidemic estimated country-level basic reproduction numbers of 1.71 (95% CI 1.44–2.01) for Guinea, 1.83 (95% CI 1.72–1.94) for Liberia and 2.02 (95% CI 1.79–2.26) for Sierra Leone [XXX], consistent with the independent estimate of 1.6–2.0 obtained from a simple two-parameter growth model fitted to the same outbreak [XXVII]. We take the triangular support of the neutrosophic reproduction number $\tilde{R}_0$ to be (1.44, 1.853, 2.26), using the lowest country-level 95% CI bound, the mean of the three point estimates, and the highest CI bound as its three vertices. For the recovery/removal rate $\tilde{\gamma}$, we use a triangular support of (0.10, 0.143, 0.20) day^{-1} , corresponding to a mean infectious period of 7 days within the 5–10 day range used in Ebola compartmental models descended from the Legrand et al. community-transmission structure [XVI]. The transmission rate is then

$\tilde{\beta} = \tilde{R}_0 \cdot \tilde{\gamma}$, combined vertex-wise with the truth/indeterminacy/falsity heights combined by min/max in the usual way for neutrosophic-number arithmetic, giving

$$\begin{aligned}\tilde{\beta} &= \langle (0.144, 0.265, 0.452); 0.80, 0.35, 0.20 \rangle \text{day}^{-1}, \\ \tilde{\gamma} &= \langle (0.10, 0.143, 0.20); 0.80, 0.30, 0.15 \rangle \text{day}^{-1}.\end{aligned}\tag{6}$$

The truth-height $w = 0.80$ reflects that these are three independently corroborated, converging country-level estimates rather than a single uncorroborated figure; the indeterminacy floor ($u = 0.30\text{--}0.35$) and falsity floor ($y = 0.15\text{--}0.20$) are set to a moderate-to-substantial level to reflect the documented suspected/probable case ambiguity noted above, rather than fitted to a specific reclassification rate, since WHO did not publish one after the point where reporting became inconsistent [XI].

As a sanity check, the resulting core reproduction number $\frac{\beta_{core}}{\gamma_{core}} = \frac{0.265}{0.143} = 1.853$ falls squarely inside the range of the three independent country-level point estimates that motivated it, and inside the case-fatality/severity literature's general description of EVD outbreak dynamics [XII]. We simulate a representative community of $N = 10,000$ with $S(0) = 9990$, $I(0) = 10$, $R(0) = 0$ over 150 days, a scale chosen for numerical clarity rather than to match the true regional population, which was several million, using the corner-point procedure of Section 3.3 for all three cut families.

IV.i. Truth-cut (α -cut) results

Table 1 reports, for five truth-cut levels $\alpha \in \{0, 0.25, 0.5, 0.75, w = 0.80\}$, the parameter interval, the resulting bound on the effective reproduction number $R_0 = \left[\frac{\beta_L}{\gamma_U}, \frac{\beta_U}{\gamma_L} \right]$, the lower and upper peak infected counts and their timing, and the lower and upper bounds on the cumulative number recovered after 150 days.

Table 1: Truth-cut (α -cut) results for the neutrosophic SIR model over 150 days.

α	β_L	β_U	γ_L	γ_U	R_{0L}	R_{0U}	I_Lpk	$t(I_L)$	I_Upk	$t(I_U)$	$R_{L(150)}$	$R_{U(150)}$
0.00	0.1440	0.4520	0.1000	0.2000	0.720	4.520	10.0	0.0	4451.4	23.5	35.5	9885.4
0.25	0.1818	0.3936	0.1134	0.1822	0.998	3.469	10.0	0.0	3533.6	28.5	235.0	9648.6
0.50	0.2196	0.3351	0.1269	0.1644	1.336	2.641	251.3	68.0	2540.2	35.5	4302.8	9095.9
0.75	0.2574	0.2767	0.1403	0.1466	1.756	1.972	1105.1	57.5	1490.0	49.5	7151.5	7891.2
0.80 (core, w)	0.2650	0.2650	0.1430	0.1430	1.853	1.853	1280.1	53.5	1280.1	53.5	7516.6	7516.6

At the maximal-confidence truth-cut ($\alpha = w = 0.80$) the two bounds coincide and reproduce the classical deterministic SIR trajectory exactly: a peak of 1280 infected individuals at day 53.5 and an eventual 7517 recovered out of the 10,000-person community, consistent with the crisp core $R_0 = 1.853$. As α decreases toward zero, the effective reproduction number widens from the point value 1.853 to the interval $[0.72, 4.52]$, notably straddling the $R_0 = 1$ threshold, so the truth-zero bound cannot rule out the epidemic dying out, the peak infected count is bounded between about 10 (i.e. no visible outbreak beyond the seed cases) and 4451 individuals, its timing ranges from immediate decline to day 23.5, and the eventual attack rate is bounded between well under 1% and about 99% of the community. Figure 1 shows the $\alpha = 0$ and $\alpha = w$ envelopes of $I(t)$ directly, and Figure 2 shows the full nested family of bounds across all five truth-cut levels.

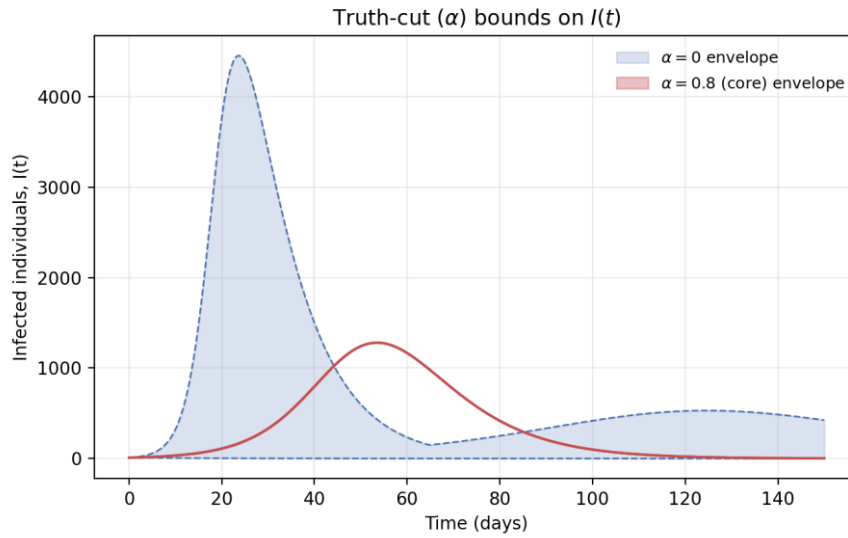


Fig. 1. Truth-cut envelopes of the infected compartment at $\alpha = 0$ (wide, dashed) and $\alpha = 0.80$ (crisp core, solid).

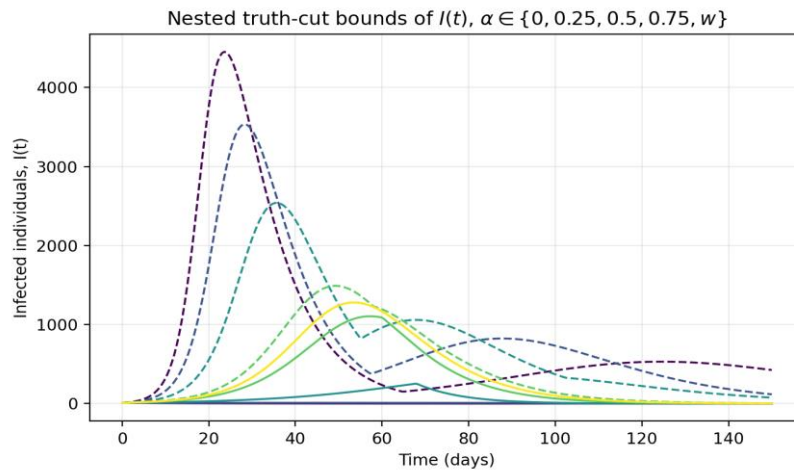


Fig. 2. Nested truth-cut bounds $[IL(t), IU(t)]$ of the infected compartment for $\alpha = 0, 0.25, 0.5, 0.75, 0.80$.

IV.ii. Indeterminacy-cut (β -cut) and falsity-cut (γ -cut) results

The truth-cut alone does not show what the neutrosophic model adds over a fuzzy one, since a fuzzy SIR model would produce an analogous α -cut family. Tables 2 and 3 therefore compute the indeterminacy-cut and falsity-cut families directly from (3), using the same corner-point procedure with $[bL, bU]$ and $[gL, gU]$ now taken from the indeterminacy- or falsity-cut interval of $\tilde{\beta}$ and $\tilde{\gamma}$ at each level, down to the respective floors $u\tilde{A} = 0.35(\tilde{\beta}), 0.30(\tilde{\gamma})$ and $y\tilde{A} = 0.20(\tilde{\beta}), 0.15(\tilde{\gamma})$ fixed in (6).

Table 2: Indeterminacy-cut (β -cut) results.

β	β_L	β_U	γ_L	γ_U	$R0_L$	$R0_U$	I_Lpk	I_Upk	$R_{L(150)}$	$R_{U(150)}$
0.35 (u, core)	0.2650	0.2650	0.1399	0.1471	1.802	1.894	1187.6	1352.5	7331.3	7652.7
0.50	0.2371	0.3081	0.1307	0.1593	1.488	2.357	562.2	2124.4	5705.0	8721.3
0.65	0.2091	0.3513	0.1215	0.1715	1.219	2.891	110.5	2872.7	2633.0	9326.2
0.80	0.1812	0.3945	0.1123	0.1837	0.986	3.513	10.0	3578.9	206.8	9665.0
1.00	0.1440	0.4520	0.1000	0.2000	0.720	4.520	10.0	4451.4	35.5	9885.4

Table 3: Falsity-cut (γ -cut) results.

γ	β_L	β_U	γ_L	γ_U	$R0_L$	$R0_U$	I_Lpk	I_Upk	$R_{L(150)}$	$R_{U(150)}$
0.20 (y, core)	0.2650	0.2650	0.1405	0.1464	1.811	1.886	1203.6	1339.6	7364.4	7628.9
0.40	0.2347	0.3117	0.1304	0.1598	1.469	2.391	513.3	2176.1	5564.0	8774.5
0.60	0.2045	0.3585	0.1202	0.1732	1.181	2.982	77.2	2985.0	1973.1	9392.8
0.80	0.1742	0.4052	0.1101	0.1866	0.934	3.680	10.0	3744.0	120.8	9720.7
1.00	0.1440	0.4520	0.1000	0.2000	0.720	4.520	10.0	4451.4	35.5	9885.4

Both families narrow toward the same crisp core as their confidence level approaches its floor and widen toward the same full-support envelope as $\beta, \gamma \rightarrow 1$, exactly as the cut-consistency conditions of Section 2.2 require: both tables converge on the $\alpha = 0$ row of Table 1 at their respective $\beta = 1 / \gamma = 1$ rows (compare the last rows of Tables 2 and 3 with the $\alpha = 0$ row of Table 1, they are identical), which is a useful internal consistency check on the implementation. What the two tables show that Table 1 alone cannot is that the indeterminacy width and the falsity width need not move together: at a comparable nominal level ($\beta = 0.65$ vs. $\gamma = 0.60$, close together on their respective [floor, 1] scales) the β -cut peak-infected bound is [110.5, 2872.7], a width of 2762, while the γ -cut bound at the nearby level is [77.2, 2985.0], a width of 2908; the two widths differ by more than 5% even at closely matched nominal levels, because $u\tilde{A}$ and $y\tilde{A}$ were fixed independently in (6) rather than tied to a single non-membership degree. In a real application, this separation is the practical payoff of the neutrosophic formulation: if a surveillance system reports a high indeterminate-case share (many suspected/probable cases pending reclassification, as in the Ebola case study above) alongside a comparatively low outright-contradiction share, the β -cut and γ -cut bounds will diverge from each other in a way no single fuzzy or intuitionistic fuzzy membership pair could represent, since those frameworks collapse indeterminacy and falsity into a single non-membership degree. Figure 3 plots the peak-infected bound against level for both cuts.



Fig. 3. Peak infected count bounds across indeterminacy-cut (left) and falsity-cut (right) levels; both panels are oriented so that confidence increases to the left (approaching the crisp core at the respective floor $u\tilde{A}$ or $y\tilde{A}$) and uncertainty increases to the right (level 1, full support).

V. Comparative Discussion

Relative to the classical deterministic SIR model, the neutrosophic formulation trades a single predicted curve for a family of nested bounds indexed by (α, β, γ) , which is the price of representing genuine parameter uncertainty rather than assuming it away; the deterministic model is recovered exactly as the maximal-confidence limit of every cut, so no predictive information is lost.

Relative to a fuzzy or intuitionistic fuzzy SIR/SEIR model built on the same triangular core values [XIII, XVIII, I, VI, VII, XVII], the neutrosophic model carries the same truth-membership (α -cut) information as the fuzzy model, and the same complementary non-membership information as an intuitionistic fuzzy model, but Section 4.2 shows directly, rather than merely asserting, that the independent β -cut and γ -cut families do not have to move together, separating how much of the residual uncertainty at a given confidence level is attributable to indeterminate reporting rather than to a bounded degree of falsity. This is useful whenever an epidemiological data source explicitly distinguishes "unconfirmed/under investigation" cases from "ruled out" cases, a distinction that a two-valued fuzzy or intuitionistic fuzzy membership pair cannot represent without conflating the two. Relative to the two existing applications of neutrosophic differential equations to biological systems, a glucose-distribution model [III] and a diabetes-stability model [IV], the present paper is, to the best of our knowledge, the first to apply the neutrosophic calculus to a population-level, compartmental epidemic model rather than to a single-patient physiological system.

The price of this additional structure is threefold: three membership functions must be estimated or elicited for each uncertain parameter instead of one or two, the resulting cut system in (5) is twice as large as the corresponding fuzzy system for the same number of cut levels, and, as noted in Section 3.3, a naive discretisation of the

coupled lower/upper system is numerically less forgiving than the corner-point procedure used here. None of these costs appears to be prohibitive for a two-parameter model of the size considered in this paper, but they would need to be re-examined for higher-dimensional compartmental models (e.g., SEIR or age-structured variants) with several neutrosophic parameters.

VI. Conclusion and Future Work

This paper introduced a Susceptible–Infected–Recovered epidemic model in which the transmission and recovery rates are represented as single-valued triangular neutrosophic numbers, formulated the model through the generalized Hukuhara neutrosophic derivative, and defined its solution as the corner-point envelope of a nested family of classical, population-conserving SIR systems through the (α, β, γ) -cut representation. We proved existence and uniqueness for each corner system rigorously (Section 3.2), proved in closed form that the resulting bounds on the epidemic peak and the final epidemic size are monotonic in the transmission and recovery rates (Proposition 1), and verified numerically, rather than proved in general, that the full pointwise-in-time trajectories are correctly ordered across the Ebola-grounded case study of Section IV. The resulting bounds on the epidemic peak, its timing, the final attack rate and the effective reproduction number degrade gracefully from a single deterministic trajectory at maximal confidence to a wide worst-case/best-case envelope at zero confidence, and the indeterminacy- and falsity-cut families show directly that the two can diverge from each other, which is the structural feature that separates this model from a fuzzy or intuitionistic fuzzy one.

Three items are identified above as open problems that a full theoretical treatment of this model should resolve:

- (i) A general, analytic comparison theorem establishing pointwise-in-time ordering of $I(t; \beta, \gamma)$ for the nonlinear SIR system, extending the closed-form peak/final-size monotonicity of Proposition 1 to the full trajectory and to arbitrary parameter regimes, not only the Ebola-grounded case study verified numerically here (Sections 3.2–3.3);
- (ii) A rigorous classification, over an infinite time horizon, of any switching point between (i)- and (ii)-differentiability of the neutrosophic cut-width functions, extending the finite-horizon verification given in Section 2.3; and
- (iii) An error-controlled neutrosophic Runge–Kutta scheme that operates directly and stably on the coupled six-equation system (5), should a future application require the coupled formulation itself rather than the corner-point envelope used throughout this paper.

Natural extensions include: fitting the (w, u, y) heights of $\tilde{\beta}$ and $\tilde{\gamma}$ to a documented probable/suspected case-reclassification rate from a specific outbreak dataset, rather than to the representative range used here; extending the model to an SEIR or age-structured compartmental structure; replacing the triangular shape with trapezoidal neutrosophic numbers [XXVI]; and combining the neutrosophic parameter structure

with the fractional-order, memory-carrying formulation used in [XXIII], each of which would need the open problems above to be revisited for the extended setting.

Data Availability, Ethics and Conflict of Interest

The parameter values, initial conditions and numerical method used to generate Tables 1–3 and Figures 1–3 are given in full in Section IV. The simulation code (Python, using SciPy's adaptive Runge–Kutta solver) is available from the corresponding author upon reasonable request. This study uses only published, publicly available epidemiological estimates and simulated compartmental trajectories; it does not involve human participants, animal subjects, or identifiable personal data, so no ethical approval was required.

Conflict of Interest:

The authors declare that there was no conflict of interest regarding this paper.

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