

System dynamics modelling of layered climate–health vulnerability: structure, calibration and empirical validation

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Abstract

Background: Climate–health vulnerability is widely assessed using conventional frameworks based on exposure, sensitivity and adaptive capacity. However, these approaches provide limited representation of how health-system capacity and epidemiological processes modify the translation of conventional vulnerability into realised impacts, constraining their utility for programme action.

Methods: We developed a layered system dynamics–based measurement model extending conventional vulnerability through explicit health-system buffering and epidemiological modulation. The model was parameterised using longitudinal data from Kenya (1987–2023) and evaluated through empirical alignment, elasticity, dominance, residual diagnostics, uncertainty propagation and global sensitivity analysis.

Results: The conventional vulnerability formulation (cCVI) showed moderate alignment with realised vulnerability ($R^2 = 0.54$, $P < 0.001$). Incorporating system buffering (sCVI) strengthened alignment to $R^2 = 0.80$ ($P < 0.001$), while epidemiological modulation (RR_adj) strengthened it further to $R^2 = 0.86$ ($P < 0.001$). Elasticity indicated a transition regime at cCVI ≈ 0.5 – 0.6 . Dominance and residual analyses showed distributed, state-dependent contributions, while Monte Carlo and Sobol analyses demonstrated robustness under parameter and input variation.

Conclusions: The layered model strengthens conventional vulnerability assessment by explicitly representing health-system buffering and epidemiological modulation, providing a transparent basis for programme planning, intervention prioritisation, monitoring, review and evaluation.

1. Introduction

Problem

Climate–health vulnerability is widely assessed, but its measurement remains inconsistently defined and operationalised. Conventional approaches commonly conceptualise vulnerability through exposure, sensitivity and adaptive capacity, providing an established basis for characterising underlying susceptibility to climate-related health risks [1, 2]. These approaches are valuable for identifying populations and places with elevated baseline vulnerability, but conventional vulnerability does not by itself specify how that vulnerability is transmitted through health systems or expressed as realised health impacts.

Climate and environmental risks increasingly place simultaneous pressures on populations, health services and the systems on which health protection depends [3–5]. Health-system capacity can influence the extent to which underlying vulnerability is translated into morbidity and mortality, while hazard-specific exposure–response relationships determine how environmental exposures are expressed as measurable health effects [5–7]. These processes represent distinct stages in the generation of realised vulnerability. Treating them as interchangeable dimensions within a single

composite measure can therefore obscure the mechanisms through which conventional conditions are buffered, transmitted or amplified.

This distinction has practical implications. A population may exhibit high conventional vulnerability while experiencing comparatively lower realised impacts where health-system capacity effectively buffers risk. Conversely, similar conventional conditions may produce greater realised impacts where system capacity is weaker or where exposure–response relationships amplify health effects. A measurement system intended to support programme planning and evaluation therefore needs to distinguish *baseline conventional vulnerability from system mediation and outcome realisation*.

Limitations of existing approaches

From a measurement perspective, vulnerability is a multidimensional construct inferred from multiple indicators. Its operationalisation requires explicit decisions concerning construct definition, indicator selection, normalisation, weighting and aggregation [8–11]. Composite-indicator methodology further emphasises the need to examine uncertainty, sensitivity and robustness because alternative specifications can affect the resulting index and its interpretation [10–12]. Where conventionally different components are combined without explicit ordering, however, a composite score can provide limited information about which processes are responsible for observed vulnerability or which components are potentially amenable to intervention.

Existing approaches can be broadly grouped into three categories. *Composite vulnerability indices* aggregate indicators of exposure, sensitivity and adaptive capacity into summary measures [1, 2, 10, 11]. Their principal strength is multidimensional representation, but aggregation can combine conventional conditions, system capacity and other attributes without explicitly specifying their functional relationships. This can limit interpretability and complicate identification of the pathways through which vulnerability is generated.

Epidemiological burden approaches instead estimate realised health effects using exposure–response relationships, including measures such as attributable mortality or disease burden [6, 7]. These approaches provide strong representation of outcome relationships, but their primary analytical focus is realised health burden rather than the upstream conventional and institutional conditions that modify the translation of exposure into health impacts.

They therefore provide an important component of vulnerability assessment without necessarily constituting a complete measurement system for vulnerability generation.

The *IPCC risk framework* provides an established conceptual basis for understanding climate risk through interactions among hazard, exposure and vulnerability [13]. It provides an important common language for climate-risk assessment and adaptation planning, but its conceptual architecture does not itself specify a formal measurement model for operationalising the sequential transformation of conventional vulnerability through health-system capacity to realised health outcomes. The resulting

methodological gap is therefore not a deficiency in the conceptual framework itself, but the absence of an explicitly specified measurement structure for representing these sequential processes within a single model.

A further limitation concerns *system behaviour*. Conventional composite approaches generally provide a summary measure of vulnerability but do not explicitly represent state-dependent responses, regime transitions or the residual structure generated when multiple interacting components are combined. Similarly, epidemiological models typically characterise exposure–response relationships rather than the broader system through which conventional vulnerability is mediated. Formal treatment of uncertainty and global sensitivity is also variable across composite vulnerability assessments, despite their importance for evaluating the robustness of multidimensional indicators [10, 14–16].

These limitations are particularly relevant where vulnerability assessment is expected to support decisions beyond descriptive profiling. Programme planning, intervention prioritisation, monitoring, review and evaluation require information on whether observed vulnerability reflects underlying conventional conditions, limitations in system capacity, or pathway-specific amplification. A model that preserves these distinctions can therefore provide a more actionable representation than a single undifferentiated vulnerability score.

CHVA extension

The Climate–Health Vulnerability Assessment (CHVA) framework extends the established conventional vulnerability architecture by explicitly representing the processes through which conventional vulnerability is mediated and realised. It does not replace the conventional formulation based on exposure, sensitivity and adaptive capacity. Rather, it establishes this formulation as the *baseline layer* and adds two analytically distinct layers representing health-system buffering and epidemiological modulation.

The first layer is *conventional Climate Vulnerability and Adaptation Index (cCVI)*. This represents baseline susceptibility arising from exposure, sensitivity and native adaptive capacity. Exposure captures the magnitude and frequency of relevant hazards; sensitivity represents susceptibility to harm; and native adaptive capacity represents conditions that enable populations and systems to withstand and recover from climate-related shocks [1, 2, 13]. The cCVI therefore provides the starting state of the model against which subsequent extensions are evaluated.

The second layer is *system buffering capacity (sCVI)*. This represents the ability of health systems to moderate the translation of conventional vulnerability into realised health impacts. Health systems can absorb shocks, maintain essential services and reduce the extent to which underlying vulnerability is expressed as adverse outcomes [5]. In the CHVA formulation, system capacity is therefore treated as a mediating process, rather than being absorbed into the conventional vulnerability construct. This permits the model to distinguish underlying susceptibility from the capacity of health services and institutions to buffer that susceptibility.

The third layer is *epidemiological modulation*, represented through relative risk (RR), Population at risk (PAR) and Attributable Risk fraction (ARF), incorporated into *enhanced vulnerability* (eCVI).

Epidemiological relationships provide an evidence-based mechanism for representing the extent to which environmental exposure and underlying vulnerability are expressed as realised health risk [6, 7]. The multiplicative formulation used in CHVA permits hazard-specific exposure–response information to modulate the vulnerability remaining after conventional conditions and system buffering have been represented.

The fourth layer translates realised vulnerability into *economic burden of disease* (EBD). This downstream component extends the framework from vulnerability measurement toward assessment of potential economic consequences, providing a basis for linking realised health impacts to resource allocation and programme decision-making while retaining the analytical distinction between upstream conventional and system processes.

The resulting architecture therefore specifies an ordered system:

Conventional vulnerability → system buffering → epidemiological realisation → economic translation

This ordering provides explicit separation between *drivers, mediators and outcomes*. It also permits the behaviour of individual layers and their combined system behaviour to be assessed rather than treating vulnerability as a static composite quantity.

Table 1

Comparative methodological positioning A comparison of the principal approaches and the proposed CHVA framework is provided in Table 1.

Dimension	Composite indices	Epidemiological burden approaches	IPCC risk framework	CHVA (this study)
Primary objective	Aggregate multidimensional vulnerability indicators into a composite score [1, 11]	Estimate attributable health impacts using exposure–response functions [6, 7]	Conceptualise climate risk as a function of hazard, exposure and vulnerability [13]	Measure vulnerability as a structured, layered system linking drivers, mediators and outcomes
Treatment of drivers, mediators and outcomes	Frequently combined within a single index, limiting interpretability [2, 15]	Focus on realised outcomes; limited representation of upstream structure [6]	Conceptually separated but not operationalised	Explicitly separated and sequentially linked across layers
Measurement structure	Implicit aggregation; often lacks a formal measurement model [10, 11]	Statistical modelling of exposure–outcome relationships	Conceptual framework without formal measurement implementation	Explicit measurement model with defined constructs and transformations
Handling of system capacity	Included as indicators within composite, often conflated with adaptive capacity [1]	Indirect or absent	Included conceptually as adaptive capacity [13]	Modelled explicitly as system buffering capacity (sCVI)
Link to realised outcomes	Weak or indirect; outcomes often excluded or inconsistently integrated [2]	Direct estimation of burden, including mortality and disease burden	Conceptual linkage only	Explicit through epidemiological modulation (eCVI = sCVI × RR)
Causal ordering / system logic	Not defined; aggregation can obscure pathways [15]	Partial exposure → outcome representation, with limited system mediation	Conceptual ordering without empirical structure	Fully specified sequential system: conventional → buffering → realised → economic
Interpretability	Limited by aggregation of heterogeneous indicators [2]	High for outcomes, limited for system structure	High conceptually, limited empirically	High; components remain analytically distinct

Dimension	Composite indices	Epidemiological burden approaches	IPCC risk framework	CHVA (this study)
Identifiability	Can be compromised by indicator mixing and collinearity [15]	Not addressed as a composite-system property	Not explicitly enforced	Enforced through separation of drivers, mediators and outcomes
Dynamic/system behaviour	Not represented	Limited to dose–response relationships	Not represented	Explicit through elasticity, regime transitions and residual diagnostics
Uncertainty treatment	Rarely formalised or limited to sensitivity checks [10, 14]	Statistical uncertainty in models	Not specified	Explicit propagation using Monte Carlo simulation, Dirichlet weights and Sobol indices
Policy relevance	Broad but often non-specific because of limited pathway interpretability	High for burden estimation and attribution	High at conceptual level	High; supports pathway interpretation and decision-relevant diagnostics
Key limitation	Conflation of components can reduce interpretability and identifiability	Limited conventional representation of vulnerability pathways	Absence of an operational measurement system	Addresses these limitations through an explicit layered measurement system

Table 1 makes the intended methodological position explicit: CHVA is an extension of the established conventional vulnerability approach, adding system buffering and epidemiological modulation as analytically distinct model layers rather than treating these processes as interchangeable components of a single composite index.

Modelling proposition and objective

The central proposition of this study is that *progressive incorporation of system buffering and epidemiological modulation should strengthen the empirical representation of realised climate–health vulnerability relative to the conventional formulation alone*. The conventional formulation therefore provides the baseline model. Incorporation of system buffering tests whether explicit representation of health-system mediation accounts for additional structure in realised vulnerability, while subsequent incorporation of epidemiological modulation tests whether hazard-specific exposure–response information provides further empirical alignment.

The model is parameterised using longitudinal data from Kenya covering 1987–2023. Model performance is evaluated progressively through empirical response alignment, *elasticity, dominance, residual diagnostics, uncertainty propagation and global sensitivity analysis*. This design permits assessment not only of the degree of empirical alignment achieved by successive model layers, but also of whether the resulting system retains boundedness, distributed component contributions and interpretable behaviour under parameter and input uncertainty.

The principal modelling question is therefore:

Does progressive extension of the conventional vulnerability formulation with explicit health-system buffering and epidemiological modulation strengthen the representation of realised climate–health vulnerability while preserving a coherent and robust measurement system?

The study addresses this question through a structured sequence of model construction, calibration and validation. The resulting framework is intended to support *comparative vulnerability assessment and programme planning, intervention prioritisation, monitoring, review and evaluation*, while maintaining analytical separation between conventional conditions, health-system capacity and realised epidemiological impacts.

2. Methods

2.1 Conceptual framework

The CHVA framework conceptualises climate–health vulnerability as a **layered measurement system** in which vulnerability is generated through sequential transformations from baseline conventional conditions to realised health impacts and, ultimately, economic consequences. Rather than representing vulnerability as a single undifferentiated composite, the framework specifies analytically distinct components and their ordering. This structure is intended to preserve interpretability of the relationships among drivers, mediating system capacity and realised outcomes [1, 2, 18].

Four sequential components are specified: **conventional vulnerability (cCVI)**, **system buffering capacity (sCVI)**, **epidemiological vulnerability (eCVI)**, and **economic burden of disease (EBD)**. The first represents baseline conventional susceptibility; the second represents health-system mediation; the third incorporates hazard-specific epidemiological relationships; and the fourth translates realised vulnerability into an economic outcome. The ordering permits each layer to be examined separately while retaining its dependence on preceding layers.

The framework is illustrated in Fig. 1.

Layer 1: Conventional vulnerability (cCVI)

Conventional vulnerability represents baseline susceptibility to climate-related health risks through *exposure, sensitivity and adaptive capacity*. Exposure captures the magnitude and frequency of relevant

hazards; sensitivity represents population susceptibility to adverse effects; and adaptive capacity represents conditions that enable populations and systems to withstand, absorb and recover from climate-related stress [1, 2, 13]. The conventional vulnerability index is defined as:

$$\text{cCVI} = \text{E} + \text{S} - \text{AC}$$

Adaptive capacity is therefore represented as a moderating term that reduces structural vulnerability. The resulting index is bounded to [0,1] following normalisation, with higher values indicating greater structural vulnerability. This formulation provides the baseline representation against which subsequent system-mediated and epidemiologically modulated transformations are evaluated.

Layer 2: System buffering capacity (sCVI)

System buffering capacity represents the capacity of the health system to moderate the translation of structural vulnerability into realised health impacts. Health systems can absorb shocks, maintain essential services and reduce excess morbidity and mortality during climate-related stress [5]. In CHVA, system capacity is represented through composite indicators including; *coverage (Universal Health Coverage Service Index)*, *service delivery (Composite Service Index)*, *preparedness (Infrastructure, Preparedness and Response)* and *governance (Health Systems Expenditure)*. These domains are incorporated as an explicit second layer rather than being absorbed into the structural vulnerability construct:

$$\text{sCVI} = \text{cCVI} - (\text{UHC} + \text{SI} + \text{CSI} + \text{IPR} + \text{HSE}) / 4$$

This formulation allows settings with comparable structural vulnerability to exhibit different levels of realised vulnerability according to differences in system performance. The adjustment therefore operationalises *health-system buffering as a mediating process*, preserving its analytical distinction from the structural determinants represented by cCVI.

Layer 3: Epidemiological modulation (eCVI)

The third layer represents the extent to which vulnerability remaining after system adjustment is expressed through empirically observed exposure–outcome relationships. Epidemiological measures such as relative risk provide pathway-specific information on the strength of associations between environmental exposure and health outcomes [6, 7, 18].

Realised epidemiological vulnerability is defined as:

$$\text{eCVI} = \text{sCVI} \times \text{RR}$$

This multiplicative formulation amplifies vulnerability where empirical relationships are strong and attenuates it where they are weaker, producing an evidence-informed measure integrating conventional conditions, system capacity and epidemiological response.

Layer 4: Economic translation

The final layer translates realised vulnerability into economic terms by linking health risk to financial impact:

$$\text{EBD} = f(\text{eCVI})$$

The economic layer extends the framework from vulnerability measurement to impact quantification, allowing realised vulnerability to be translated into financial consequences relevant to resource allocation, adaptation planning and investment prioritisation. The economic transformation remains downstream of the vulnerability system and does not alter the definition of the preceding structural, system or epidemiological layers.

Overall system logic

Across the four layers, the framework specifies a sequential transformation:

structural conditions → *system buffering* → *epidemiological realisation* → *economic translation*

Each layer is analytically distinct but dependent on the preceding layer. This ordering permits stage-specific assessment of model behaviour and prevents structural vulnerability, system capacity and realised outcomes from being treated as interchangeable components of a single composite measure. The framework consequently represents vulnerability as an *emergent property of an ordered measurement system*, rather than solely as a static aggregate.

This distinction is central to the model evaluation. The structural formulation provides the baseline representation; incorporation of system buffering tests whether explicit health-system mediation strengthens that representation; and incorporation of epidemiological modulation tests whether hazard-specific exposure–response information provides further empirical alignment.

2.2 Measurement model

Vulnerability is operationalised as a structured measurement system in which the principal constructs occupy defined positions within the causal-ordering logic of the framework. Conventional vulnerability (cCVI) and system buffering capacity (sCVI) are specified as *formative constructs*, because their values arise from the combination of multiple component indicators representing distinct dimensions of the respective constructs [8–11, 19]. Epidemiological vulnerability (eCVI) is a derived construct obtained through the transformation of sCVI by the relevant epidemiological risk relationship, while economic burden of disease (EBD) functions as a downstream outcome anchor.

The distinction between formative and derived constructs is important because the indicators within cCVI and sCVI are not treated as interchangeable manifestations of a single latent trait. Rather, they collectively define the respective constructs through their specified domains and aggregation rules [19].

This approach is consistent with the need to avoid measurement-model misspecification when multidimensional indices combine conceptually distinct indicators.

Identifiability is supported by the explicit separation of *drivers*, *mediators* and *outcomes* within the model architecture. Structural indicators enter cCVI; health-system indicators enter the buffering layer; epidemiological relationships enter the realisation layer; and economic measures are retained as downstream outcomes. This separation reduces conflation between conventional conditions and realised impacts and provides a defined structure within which component contributions and model behaviour can be assessed.

Construct validity is assessed through the conceptual specification of domains, their placement within the layered architecture, and their empirical relationships with realised vulnerability and downstream outcomes [2, 10, 11]. Reliability is considered in relation to temporal consistency and robustness to variation in parameters and inputs, recognising that uncertainty introduced at earlier stages may propagate through subsequent transformations.

The measurement model therefore does not assume that the component indices are statistically independent. Rather, it explicitly acknowledges their *functional dependence and potential collinearity*, while preserving their conceptual and computational separation. This distinction is central to interpretation of the regression, residual, dominance and sensitivity analyses presented subsequently.

2.3 Index construction

All component indicators are normalised using **min–max scaling** to place measures on a common bounded scale and permit aggregation across heterogeneous units. Within domains, indicators are combined using **equal weighting**, reflecting the absence of a defensible prior basis for assigning differential weights and maintaining transparency in the construction of the composite measures [10, 11].

Data-driven weighting approaches such as principal component analysis are not adopted because resulting weights can be sensitive to the covariance structure and composition of the specific dataset and may reduce the interpretability of the resulting index [20, 21]. Equal weighting therefore provides an explicit and reproducible specification in which each pre-defined indicator contributes according to the declared measurement architecture rather than an empirically generated weighting scheme.

The structural vulnerability index is constructed from the normalised exposure, sensitivity and adaptive-capacity domains. The resulting structure is nonlinear in its underlying construction because the epidemiological layer acts multiplicatively on the system-adjusted vulnerability. System capacity therefore functions as an attenuating component, while epidemiological relationships determine the extent to which the remaining vulnerability is expressed through the relevant exposure–outcome pathway.

For empirical estimation of relationships between model components, *linear regression is used as an interpretable approximation to the relationships generated by the underlying layered formulation*. This permits estimation of empirical alignment without replacing the underlying nonlinear measurement structure with a linear model. Regression residuals are retained for diagnostic analysis because departures from the fitted relationship may contain information about state-dependent buffering and amplification within the layered system.

The resulting approach distinguishes between the *construction of the vulnerability measure* and the *statistical approximation used to evaluate relationships among its components*. This distinction is maintained throughout the subsequent analyses.

2.4 Validation framework

The model is evaluated using *pre-specified analytical criteria addressing empirical alignment, system behaviour and robustness*, rather than treating statistical significance alone as evidence of model validity. The validation framework is designed to determine whether the layered measurement system exhibits the expected boundedness, state dependence, component balance and stability under uncertainty.

Elasticity

Elasticity assesses the responsiveness of realised vulnerability to changes in conventional structural vulnerability. The analysis examines both raw estimates and LOESS-smoothed relationships to identify whether the response remains bounded and whether its magnitude varies across the vulnerability range. State dependence is evaluated particularly around the transition region identified in the empirical relationship.

Dominance

Component dominance is assessed using variance-based decomposition of standardised model contributions. The analysis evaluates whether one component accounts for a disproportionate share of model behaviour and applies a **60% dominance threshold** as a diagnostic criterion [14, 15]. The purpose is not to establish causal attribution, but to determine whether the layered structure remains distributed across its specified components rather than collapsing into effective single-component control.

Residual structure

Residual analysis is used as a diagnostic of model behaviour. Deviations from fitted relationships are examined for systematic structure rather than treated solely as unexplained statistical error. Negative deviations are interpreted within the framework as evidence consistent with stronger buffering, while positive deviations indicate patterns consistent with amplification. This interpretation is conditional on the model architecture and is not treated as evidence of independent causal effects.

Uncertainty propagation and sensitivity

Robustness is evaluated by propagating uncertainty through the layered measurement system. Monte Carlo simulation incorporates uncertainty in weights and model inputs, including *Dirichlet-distributed weights* and Gaussian perturbations. Global sensitivity is assessed using *Sobol variance-based sensitivity indices*, which quantify the contribution of model inputs to output variance [15, 17, 22].

The stability criterion is defined by preservation of the principal model properties under perturbation: *boundedness, distributed component contribution and residual structure*. A model configuration is considered robust when these properties remain materially preserved across the uncertainty space rather than depending on a narrow combination of parameter values.

Together, these analyses provide complementary tests of the measurement system. Empirical response alignment assesses correspondence with observed relationships; elasticity assesses state-dependent response; dominance assesses component balance; residual diagnostics examine systematic departures; and uncertainty and sensitivity analyses assess robustness to parameter and input variation.

The validation framework therefore evaluates *internal coherence and robustness of the layered measurement system*, rather than claiming causal validation of the underlying climate–health relationships. This distinction is maintained in interpretation of all subsequent model results.

3. Results

3.1 System response and progressive decomposition of climate–health vulnerability

The relationship between conventional vulnerability (cCVI) and realised vulnerability (eCVI) across the three climate-sensitive risks and the 1987–2023 observation period is shown in Fig. 2. Each observation represents a risk–year combination.

Realised vulnerability increased with conventional vulnerability, with the baseline structural formulation showing a moderate empirical alignment ($R^2 = 0.54$, $P < 0.001$; Fig. 2a). This establishes the conventional vulnerability formulation as a measurable baseline but also indicates that structural conditions alone do not account for the full variation in realised vulnerability. Dispersion around the fitted relationship is consistent with the additional influence of system buffering and epidemiological modulation within the layered measurement system.

Incorporation of system buffering capacity (sCVI) produced systematic divergence around the baseline relationship. Observations at or below the median sCVI clustered at lower eCVI values, whereas observations above the median aligned with higher eCVI values, indicating state-dependent expression of vulnerability across levels of system capacity. The resulting relationship showed substantially stronger empirical alignment ($R^2 = 0.80$, $P < 0.001$; Fig. 2b) than the structural formulation alone.

The further incorporation of epidemiological modulation (RR_{adj}) produced the strongest correspondence with realised vulnerability ($R^2 = 0.86$, $P < 0.001$; Fig. 2c). The progression from $R^2 = 0.54$ to **0.80 and 0.86** therefore represents progressive strengthening of the empirical representation as system buffering and epidemiological exposure–response information are incorporated into the structural model.

The regression lines represent least-squares fits within a collinear composite measurement system. The successive increases in R^2 are consequently interpreted as *improved internal alignment of the layered formulation*, rather than as incremental causal explanatory effects of sCVI or RR. This distinction is important because the component indices are functionally related within the model.

The distribution of observations also indicates a transition in system behaviour. Lower realised vulnerability predominates at approximately $cCVI \leq 0.5$, while responses become increasingly amplified beyond approximately $cCVI \approx 0.6$.

The consistency of this transition across the three risk categories indicates that the pattern is associated with the behaviour of the layered system rather than being confined to a single epidemiological pathway.

Key result

incorporation of health-system buffering and epidemiological modulation progressively strengthens empirical alignment with realised vulnerability beyond the conventional structural formulation.

3.2 Elasticity (1987–2023 data derived)

Elasticity of realised vulnerability with respect to conventional vulnerability across the 1987–2023 observation period is shown in Fig. 3.

Raw elasticity estimates showed substantial dispersion across the cCVI domain, including near-zero values and higher estimates concentrated around $cCVI \approx 0.5–0.6$ (Fig. 3a). The coexistence of low and high elasticity within this interval indicates heterogeneous system response under similar levels of conventional vulnerability.

LOESS-smoothed estimates showed a **bounded and continuous increase** in elasticity across the cCVI domain (Fig. 3b). Elasticity remained relatively low at lower levels of structural vulnerability, increased through the intermediate range of approximately **0.5–0.6**, and subsequently stabilised. The corresponding pattern was observed across the three risk categories.

The LOESS estimates with 95% confidence intervals showed the greatest uncertainty within the **0.5–0.6 transition zone** (Fig. 3c), with narrower uncertainty outside this range. This concentration of uncertainty coincides with the region in which the system response changes from predominantly buffered to increasingly amplified behaviour.

The raw estimates therefore provide evidence of substantial local heterogeneity, whereas the smoothed response identifies the underlying system-level pattern. The difference between the two representations is important because local elasticity estimates can be sensitive to the spacing and variability of observations, whereas the smoothed relationship provides a more stable representation of the response across the vulnerability domain.

As elasticity is derived from relationships among components of the composite measurement system, it is interpreted as a measure of *internal system responsiveness*, rather than as a causal marginal effect of conventional vulnerability on an independently measured outcome. Overall, the elasticity analysis identifies a *bounded, state-dependent response*, with the greatest heterogeneity and uncertainty concentrated in the intermediate vulnerability range where buffering and amplification coexist.

Summative interpretation

The coexistence of low and high elasticity within this interval indicates heterogeneous system response, with buffering and amplification occurring under similar conventional conditions. Part of this variation reflects numerical sensitivity in local estimation rather than instability. Overall, elasticity reveals a bounded, regime-dependent transmission mechanism, with maximum heterogeneity and uncertainty concentrated in the transition zone.

3.3 Dominance (standardised coefficients)

Dominance analysis based on standardised coefficients showed that contributions to realised vulnerability were distributed across the three principal model components (Fig. 4). Relative influence was highest for epidemiological modulation (RR), followed by conventional vulnerability (cCVI) and system buffering capacity (sCVI).

The standardised coefficients were approximately *0.54 for RR, 0.29 for cCVI and 0.26 for sCVI*. No individual component therefore accounted for the majority of the variation represented by the layered measurement system.

The relatively similar coefficients for cCVI and sCVI indicate comparable contributions of conventional conditions and system buffering to the model response. This is consistent with the layered formulation in which system capacity modifies the transmission of structural vulnerability rather than constituting a component of the structural baseline. The higher coefficient for RR indicates the greater relative contribution of epidemiological modulation to variation in realised vulnerability within the calibrated system.

Because the components are interdependent and partly collinear by construction, these coefficients represent *relative contributions within the measurement system*, rather than independent causal effects. The analysis therefore provides a test of component balance rather than a causal ranking of determinants.

The absence of a dominant single component supports the intended distributed structure of the model. Realised vulnerability (eCVI) is consequently represented as the combined result of structural conditions, system buffering and epidemiological modulation, with the relative contribution of epidemiological modulation increasing within the observed range of realised vulnerability.

3.4 Residual structure

Residuals showed systematic, non-random structure across the range of predicted eCVI values (Fig. 5), indicating that departures from the fitted relationships were not adequately characterised as purely stochastic variation.

Negative residuals predominated across the central range of predicted values, while positive residuals occurred towards both lower and higher extremes. This pattern indicates relative overestimation of realised vulnerability in the intermediate range and relative underestimation towards the extremes.

The curvature of the residual distribution is consistent with the bounded nonlinear response generated by the transformation from conventional vulnerability through system buffering and epidemiological modulation. Concentration of negative residuals at intermediate values corresponds to the **cCVI $\approx$ 0.5–0.6 transition region**, where the elasticity analysis also identified changing system responsiveness.

Positive residuals at higher predicted values are consistent with stronger-than-expected amplification within the measurement system, while positive residuals at lower values are consistent with residual buffering. The persistence of these patterns across the prediction range supports interpretation of residuals as diagnostic information about system behaviour rather than as undifferentiated error.

Because residuals are generated within a collinear composite system, they represent deviations within the constructed measurement space and should not be interpreted as independent causal effects. Their structured distribution nevertheless provides complementary evidence that the relationship between conventional and realised vulnerability is *state dependent rather than uniformly linear*.

Taken together with the elasticity results, the residual structure supports the interpretation of a bounded system in which the relative expression of vulnerability changes across the cCVI domain.

3.5 Robustness and uncertainty analysis under parameter and input variation

Robustness was assessed through parameter perturbation, input variability and uncertainty propagation to determine whether the principal properties of the measurement system remained stable under plausible variation. The analysis focused on preservation of *bounded response, distributed component contributions and interpretable residual structure*. Figure 6 represents robustness of the layered measurement system under indicator and parameter variation

Uncertainty propagation

Panel A shows propagation of uncertainty in indicator weights and input values through the composite measurement system. Indicator weights were represented as random variables drawn from a Dirichlet distribution:

$$\mathbf{w} \sim \text{Dir}(\alpha_1, \dots, \alpha_k)$$

which preserves the unit-sum constraint while allowing variability across indicators. Symmetric concentration parameters (α_i) reflect baseline equal weighting, with stochastic variation across simulations.

The Dirichlet formulation preserves the unit-sum constraint while allowing the relative contribution of individual indicators to vary across simulations. Symmetric concentration parameters represent the baseline equal-weight specification, with stochastic variation introduced around that specification. The resulting weight distributions are centred on the equal-weight specification (1/3 for each of three indicators), providing controlled dispersion around the baseline weighting structure without imposing directional preference. This approach is consistent with probabilistic treatment of uncertainty in composite-indicator weighting and robustness assessment [12, 14]. Input uncertainty is introduced via additive Gaussian perturbation;

$$X_i^* = X_i + \epsilon_i, \epsilon_i \sim N(0, \sigma_i^2)$$

where σ_i reflects the observed variability associated with input X_i . These perturbations were propagated through the full index-construction process using *10,000 Monte Carlo iterations*, providing a systematic assessment of how input and weighting uncertainty propagates through the layered measurement system [15, 17]. The bounded formulation constrains the resulting output range, limiting divergence under perturbation and preserving the relative ordering of observations across simulations.

Variance decomposition and sensitivity

Panel (B) presents the **global sensitivity analysis**, based on variance decomposition of the system output using established variance-based sensitivity methods [15, 17]. The analysis partitions output variance into contributions

$$Y = \mathbf{eCVI}.$$

The total variance is partitioned into contributions from individual inputs and their interactions;

$$\text{Var}(Y) = \sum_i V_i + \sum_{i < j} V_{ij} + \dots$$

First-order Sobol indices were calculated to quantify the proportion of output variance attributable to each input independently, following the variance-based sensitivity framework of Sobol [22] and its subsequent methodological development for global sensitivity analysis [15].

$$S_i = V_i / \text{Var}(Y)$$

where V_i represents the variance attributable to input i , and Y is the model output. The indices quantify the first-order contribution of each principal model component—*cCVI*, *sCVI* and *RR*—to output variability [22, 15]. Total-effect indices S_i^T were used to capture both the direct contribution of each input and its higher-order interaction effects with other model components [15, 17]. The resulting variance contributions remained distributed across *cCVI*, *sCVI* and *RR*, with no single component accounting for the majority of system variance.

The similarity between first-order and total-effect indices indicates that interactions contribute to system behaviour, but do not concentrate variance in a single controlling component. This distribution is consistent with the dominance analysis and supports interpretation of realised vulnerability as an *integrated system response* rather than the output of a single controlling factor. The robustness analysis therefore indicates that the principal characteristics of the layered formulation are preserved under parameter and input perturbation.

The sensitivity results further indicate that model behaviour is not dependent on a narrowly specified parameter configuration. The preservation of boundedness and distributed variance contributions under uncertainty supports the stability of the layered formulation, while the presence of interaction effects is consistent with the model's representation of interdependent structural, buffering and epidemiological processes. Interpretation is therefore based on the *joint contribution of model components*, rather than on ranking individual inputs as independent causal determinants [16].

4. Discussion

The results support the treatment of climate–health vulnerability as a *structured measurement system rather than a descriptive composite*. The conventional vulnerability formulation provides the baseline representation, while explicit incorporation of system buffering capacity and epidemiological modulation progressively strengthens its empirical alignment with realised vulnerability. The increase in correspondence from $R^2 = 0.54$ for *cCVI alone*, to $R^2 = 0.80$ following incorporation of *sCVI*, and to $R^2 = 0.86$ following incorporation of *epidemiological modulation* is therefore the principal modelling result. It indicates that structural conditions alone leave substantial variation in realised vulnerability unresolved and that representing system mediation and epidemiological response captures additional structure within the layered formulation.

This finding addresses an important limitation of conventional composite approaches. Composite vulnerability measures provide valuable multidimensional characterisation but can combine drivers, mediating conditions and outcomes in ways that limit interpretability [2, 11]. The CHVA formulation instead maintains these components as sequential but interdependent layers. The improvement in empirical alignment following incorporation of *sCVI* indicates that health-system capacity contributes materially to the representation of how structural vulnerability is expressed. The further improvement

following incorporation of epidemiological modulation indicates that hazard-specific exposure–response information provides an additional layer of correspondence with realised vulnerability. These results support extending, rather than replacing, the established structural vulnerability architecture.

The model's response behaviour provides a second dimension of evidence. Elasticity remained bounded and state dependent, with the principal transition occurring at approximately $cCVI = 0.5–0.6$. This intermediate range represents a change in the responsiveness of realised vulnerability to structural conditions, with buffering and amplification occurring within the same region. The finding is consistent with the bounded formulation of the composite system and demonstrates why a single global linear relationship is insufficient to characterise the full response behaviour. The use of LOESS smoothing provides a stable representation of the underlying response while retaining the observed heterogeneity in the raw elasticity estimates [11, 21].

Dominance analysis further indicates that the model does not collapse into a single controlling component. Epidemiological modulation showed the largest relative contribution, followed by conventional vulnerability and system buffering, but no component exceeded the specified dominance threshold. This distributed contribution is consistent with the intended layered architecture, in which realised vulnerability emerges from the interaction of structural conditions, system capacity and epidemiological response. Because the components are functionally related and partly collinear by construction, these contributions should be interpreted as *relative contributions within the measurement system rather than independent causal effects* [10, 11, 15].

The residual analysis provides complementary evidence of structured system behaviour. Residuals were not randomly distributed across the fitted range but showed systematic departures associated with different levels of predicted vulnerability. Negative deviations were concentrated within the intermediate response range, whereas positive deviations occurred towards the lower and upper portions of the response distribution. In the context of the specified model, these patterns are consistent with state-dependent buffering and amplification. Residual structure therefore provides information about aspects of system behaviour not represented by the linear approximation used for empirical estimation. It should not, however, be interpreted as independent evidence of causal buffering or amplification.

The incorporation of epidemiological information is particularly important for interpretation of realised vulnerability. Structural vulnerability establishes the underlying conditions under which climate-related health risks may occur, but exposure–response relationships provide information on the extent to which those conditions are translated into measurable health effects [6, 7]. The higher empirical alignment obtained after incorporating RR therefore indicates greater correspondence between the layered measurement system and realised vulnerability.

This is an important distinction from treating epidemiological outcomes as additional indicators within a conventional composite index: in CHVA, epidemiological information enters as a specified *realisation layer*.

The uncertainty and sensitivity analyses provide evidence that the principal model properties are not dependent on a narrowly specified set of weights or inputs. Monte Carlo propagation using Dirichlet-distributed weights and perturbed inputs preserved the bounded response and general ordering of observations. Sobol analysis showed distributed variance contributions across cCVI, sCVI and RR, with no single component accounting for the majority of output variance. The similarity between first-order and total-effect contributions indicates that interactions are present but do not concentrate system behaviour in a single component [15–17]. These findings strengthen confidence in the structural robustness of the model while recognising that robustness to specified perturbations is not equivalent to empirical validation across independent populations.

The practical implication is that the model provides information beyond a conventional vulnerability ranking. By separating *structural vulnerability*, *system buffering* and *epidemiological realisation*, the framework permits different types of vulnerability to be distinguished within a common analytical structure. This is relevant to programme planning because structural vulnerability may identify underlying need, while the buffering layer can indicate where health-system constraints may modify risk.

Epidemiological modulation can then identify pathways through which environmental exposure is expressed as health impact. The same structure can support intervention prioritisation, monitoring of system response, programme review and evaluation of whether changes in system capacity are associated with changes in realised vulnerability.

The framework therefore provides a bridge between climate-risk assessment and health-system decision-making. Its principal contribution is not the replacement of existing vulnerability frameworks, but the *formalisation of additional system layers that strengthen the empirical representation of realised vulnerability while preserving the structural baseline*. This distinction is important for policy use because it permits continuity with established vulnerability assessment while providing additional diagnostic information for action.

5. Conclusion

This study developed a *layered measurement model of climate–health vulnerability* that extends conventional structural vulnerability through explicit representation of health-system buffering and epidemiological modulation. The model retains structural vulnerability as the baseline formulation and introduces system capacity and epidemiological response as sequentially defined layers linking underlying conditions to realised impacts.

The principal result is the progressive strengthening of empirical alignment across these layers. The structural formulation produced a moderate relationship with realised vulnerability ($R^2 = 0.54$), which increased to $R^2 = 0.80$ following incorporation of system buffering and to $R^2 = 0.86$ following incorporation of epidemiological modulation. This progression indicates that the additional layers capture systematic variation not represented by structural vulnerability alone.

The R^2 sequence is interpreted as *progressive model alignment within the layered measurement system*, rather than as independent causal effects.

The remaining analyses support this interpretation. Elasticity demonstrated a bounded, state-dependent response with a transition region around cCVI 0.5–0.6. Dominance analysis showed distributed contributions across cCVI, sCVI and RR, while residual diagnostics identified structured deviations consistent with state-dependent system behaviour. Monte Carlo uncertainty propagation and Sobol sensitivity analysis demonstrated preservation of the principal model properties under parameter and input variation.

Because the model components are interdependent and partly collinear by construction, these findings describe *system properties rather than independent causal effects*. Within this scope, the framework provides a transparent and reproducible basis for extending structural climate–health vulnerability assessment with explicit representation of system buffering and epidemiological realisation.

The principal policy value is therefore diagnostic as well as descriptive. The framework can support *programme planning, intervention prioritisation, monitoring, review and evaluation* by distinguishing underlying structural vulnerability from the capacity of health systems to buffer that vulnerability and from the epidemiological processes through which risk is realised.

Further application across countries, populations and independent datasets will be required to assess external generalisability and transportability.

6. Limitations

Several limitations should be considered when interpreting the model.

First, the functional specification is model-dependent. The sequential transformations and multiplicative epidemiological formulation provide an explicit and reproducible representation of the proposed system, but alternative functional forms may produce different elasticity profiles, transition characteristics or component relationships. The reported behaviour should therefore be interpreted within the specified model structure.

Second, the model components are interdependent. cCVI, sCVI and RR occupy sequential positions within a constructed composite system and are not statistically independent predictors. Consequently, regression coefficients, dominance measures and sensitivity contributions should not be interpreted as independent causal effects. The analyses instead quantify relative contribution and system behaviour within the specified measurement architecture.

Third, elasticity estimates are sensitive to local variation. Raw elasticity calculated from local changes can be affected by discretisation, measurement noise and the density of observations across the cCVI range. LOESS smoothing provides a more stable representation of the underlying response, but smoothing does not eliminate uncertainty in the underlying observations or establish a unique functional form.

Fourth, uncertainty propagation depends on the specified probability distributions. Dirichlet distributions were used to perturb indicator weights around the equal-weight specification, while Gaussian perturbations were used to represent input variability. These distributions provide a transparent sensitivity framework but may not capture all forms of dependence, measurement error or structural uncertainty within the underlying data. The robustness findings should therefore be understood as conditional on the uncertainty structures specified in the model.

Fifth, the empirical application is based on a single national case study. The Kenya application provides longitudinal evidence for model calibration and assessment, but does not establish external validity across different health systems, climatic regimes or population structures. Application to additional countries and independent datasets is required to assess transportability and determine whether the observed transition behaviour and progressive model strengthening are reproduced under different system conditions.

Sixth, the model is not intended to provide individual-level causal inference. Its purpose is to represent climate–health vulnerability as an integrated measurement and system-behaviour construct. The observed associations and model diagnostics should therefore not be interpreted as estimates of independent causal effects of cCVI, sCVI or RR.

These limitations define the appropriate scope of inference. The evidence supports the *internal coherence, progressive empirical alignment and robustness of the proposed layered formulation within the study setting*, while broader generalisation and causal interpretation require further empirical testing.

Declarations

Author Contribution

S.M.N. conceptualised the study, developed the analytical framework, conducted the analysis, and led the manuscript writing. L.L.B. contributed to methodological review, policy alignment, and critical revision of the manuscript. M.M. contributed to technical review, interpretation of results, and manuscript refinement. All authors reviewed and approved the final manuscript.

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refinement. All authors reviewed and approved the final manuscript. **Disclosures** Conflict of interest The authors declare no competing financial or non-financial interests. **Use of Generative AI** During the preparation of this work the author(s) used chatGPT as a large language model in organisation and formatting of text based on content and ideas developed by the applicant. Specifically, to support language editing and conventional refinement of sections of this application, The tools assisted with improving clarity by editing verbose sentences and developing illustrative images of concepts that sounds too new to explain fully in words. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article. **Ethics statement** This study uses secondary, aggregated data and does not involve human participants, individual-level data or identifiable information. Ethical approval was therefore not required. **Funding statement** No funding to declare. This research started with programme-level Climate-Health Vulnerability Adaptation Assessment (CHVA) by Ministry of Health, Kenya. Supported by Netherlands Global Health Partnership Programme (GHPP) and Amref Health Africa. **Declarations** This manuscript is not under consideration elsewhere. All authors have approved the submission. The authors declare no competing interests. We believe this manuscript will be of interest to readers of Scientific Reports, particularly those working across climate science, public health, and complex systems. The framework provides a reproducible basis for understanding and acting on climate–health risk across diverse settings.

Data Availability

The datasets generated and/or analysed during the current study are publicly available from World Bank (World Development Indicators for Kenya), also Figshare: <https://doi.org/10.6084/m9.figshare.33260784>

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23. Reference architecture.

Figures

Figure 1. Layered conceptual framework of climate–health vulnerability

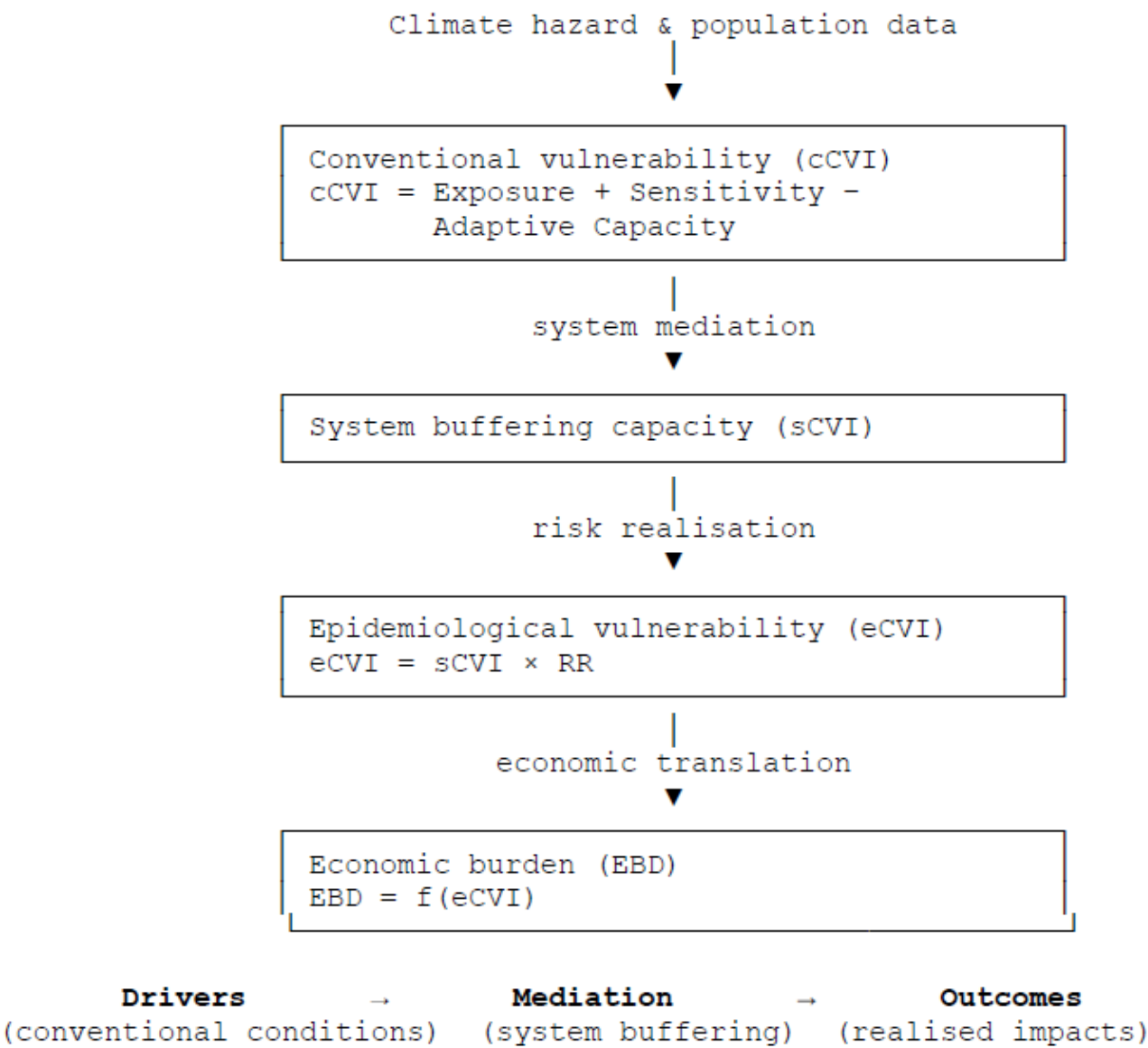


Figure 1

See image above for figure legend.

Figure 2: System response and progressive decomposition of climate–health vulnerability

The relationship between conventional vulnerability (cCVI) and realised vulnerability (eCVI) across the three climate-sensitive risks and the 1987–2023 observation period is shown in Fig. 2. Each observation represents a risk–year combination.

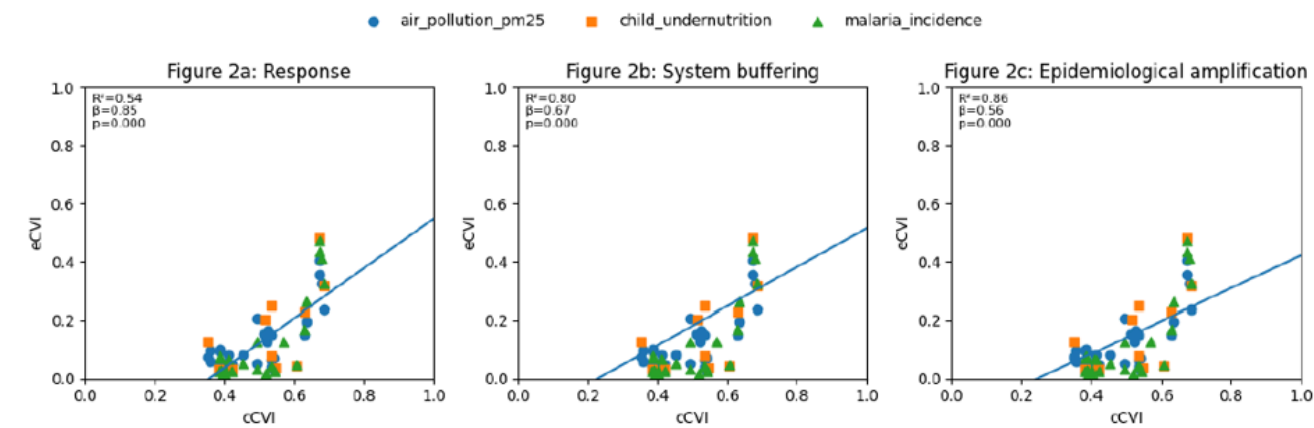
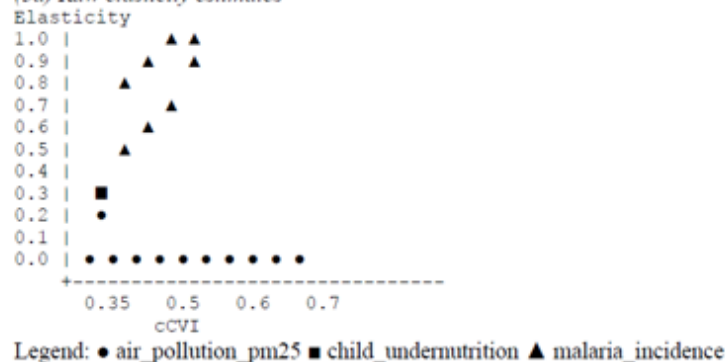


Figure 2

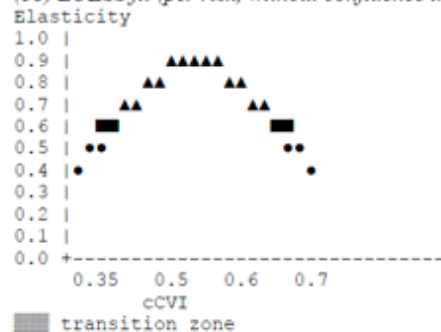
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Figure 3. Elasticity of realised vulnerability across conventional vulnerability and risk categories, 1987–2023

(3a) Raw elasticity estimates



(3b) LOESS fit (per risk, without confidence intervals)



LOESS-smoothed elasticity shows a bounded, continuous increase across the cCVI domain.

(c) LOESS fit with 95% confidence intervals (per risk)

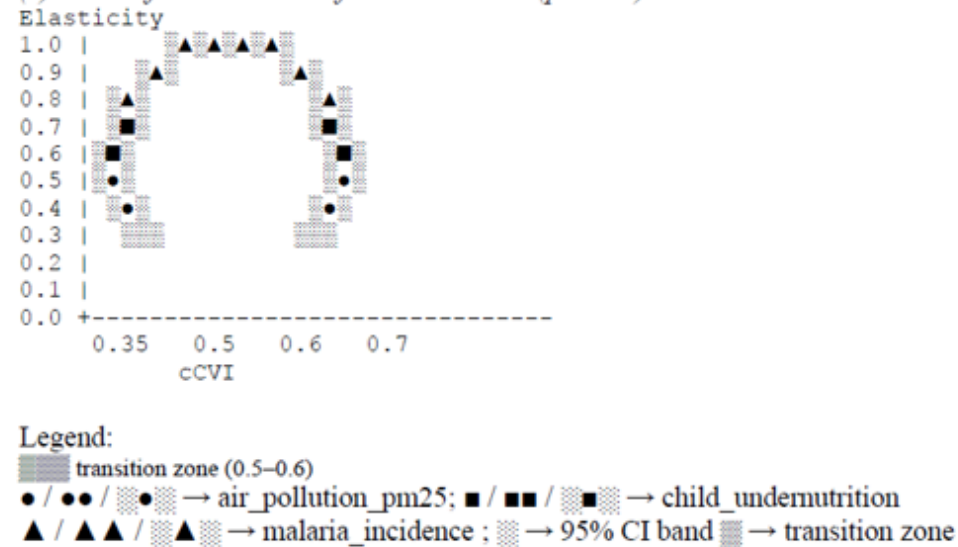


Figure 3

See image above for figure legend.

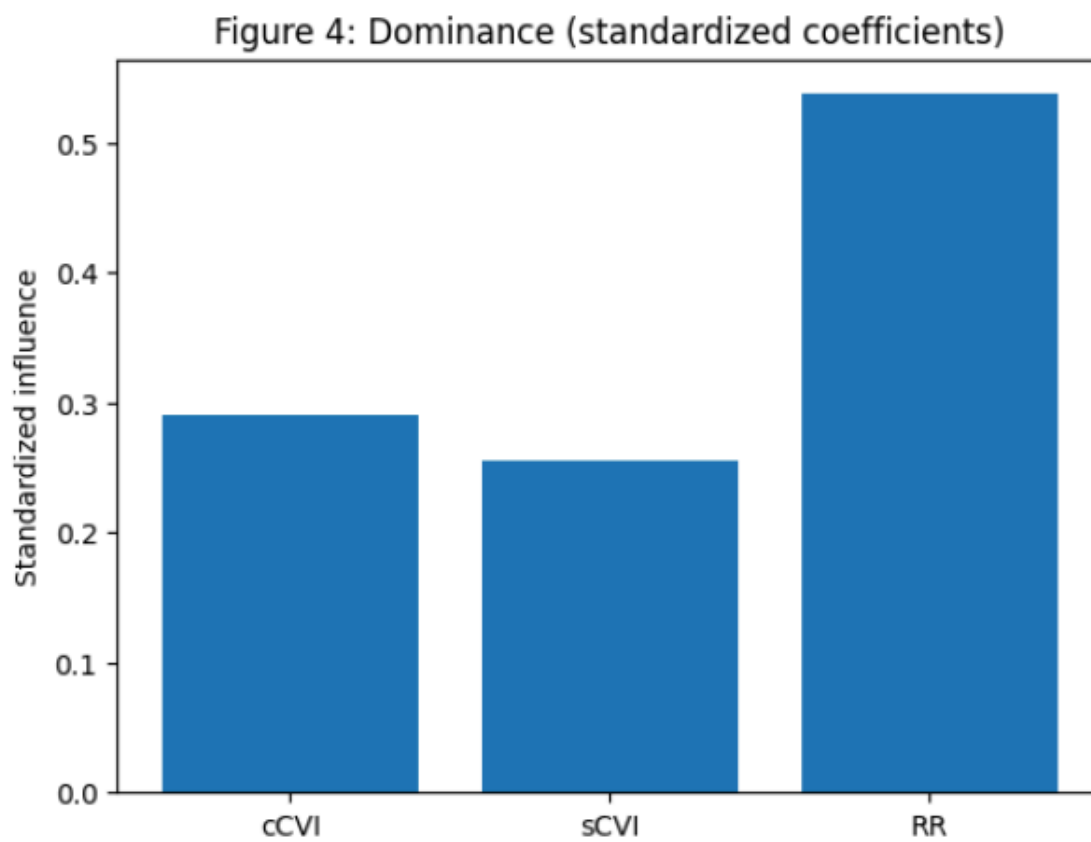


Figure 2: Index dominance (standardised coefficients)

Figure 4

See image above for figure legend.

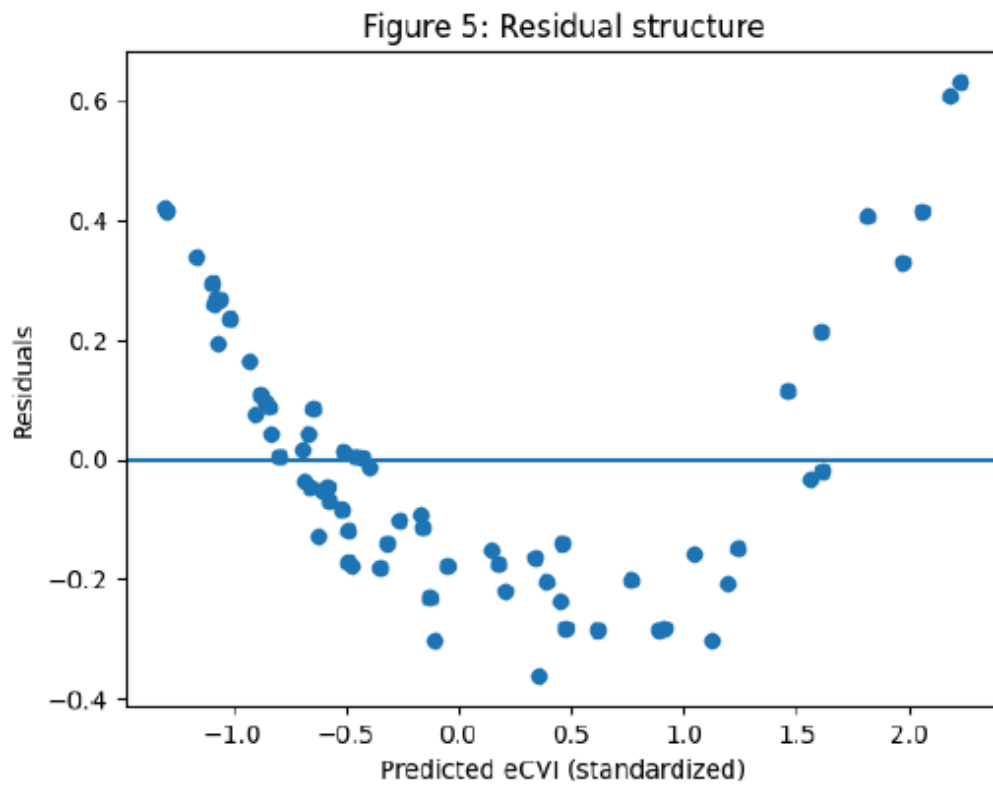


Figure 3: Residual structure (1987–2023 and risk categories)

Figure 5

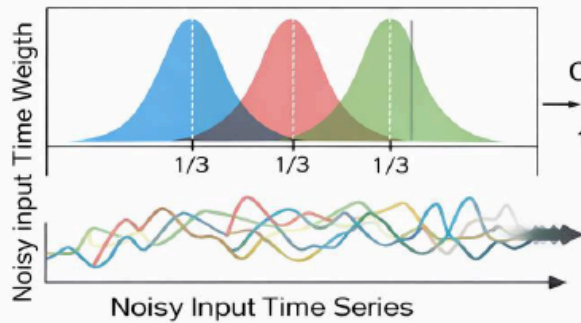
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Robustness

(A) Uncertainty Propagation

$$\mathbf{w} \sim \text{Dir}(\alpha_1, \alpha_2, \dots, \alpha_k)$$

- $\mathbf{w}$ represents the vector of weights assigned to indicators within each composite index, and α_i
- $X_i^* = X_i + \varepsilon_i, \varepsilon_i \sim N(0, \sigma_i^2)$



- Perturbed input indicators via indicator:
 $X_i^* = X_i + \varepsilon_i, \varepsilon_i \sim N(0, \sigma_i^2)$

Composite index simulation: 10,000 iterations

(B) Variance Decomposition

$$\text{Var}(Y) = \sum_{i \in I} V_i + \sum_{i < j} V_{ij} + \dots$$

$$S_i = \frac{V_i}{\text{Var}(Y)} \quad S_i^T = 1 - \frac{V_{\mathbf{e}_{X_i}^{\perp} | \mathcal{X} \setminus \{i\}}}{\text{Var}(Y)}$$

Composite index simulation: 10,000 iterations

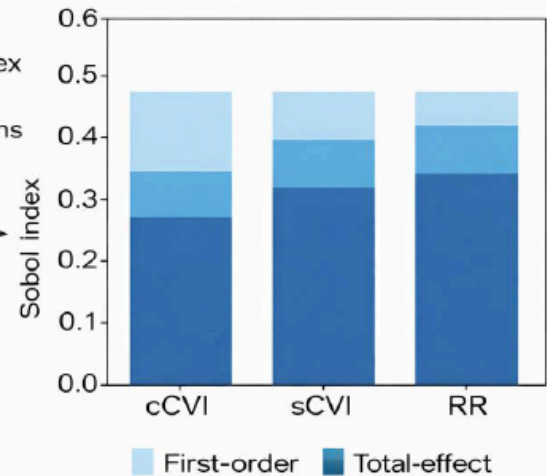


Figure 4: Robustness; indicator resampling under varying assumptions

Figure 6

See image above for figure legend.

Supplementary Files

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- [Referencearchitecture.docx](#)