

Decomposing climate–health vulnerability into structural, system buffering and amplification components: a longitudinal analysis in Kenya

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Article

Keywords: Climate–health risk, Vulnerability decomposition, Health system buffering, Epidemiological amplification, Composite index measurement, Kenya

Posted Date: July 27th, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-9193214/v1>

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Additional Declarations: No competing interests reported.

Abstract

Climate-risk assessments are widely used to evaluate how environmental hazards affect human systems, yet prevailing vulnerability frameworks do not explicitly distinguish between baseline susceptibility and the processes through which hazards translate into realised impacts. This limits both explanatory power and policy relevance. In this study, we develop and demonstrate a layered climate–health vulnerability framework that decomposes risk into structural vulnerability (baseline susceptibility), system buffering (transmission of risk) and hazard-specific amplification processes (conversion into realised impacts).

Applying the framework to climate-sensitive health pathways in Kenya (1987–2023), we find that structural vulnerability explains a substantial proportion of variation in realised vulnerability ($R^2 = 0.72$, $p < 0.001$). Multivariate analysis shows that system buffering ($\beta = 0.48$, $p < 0.01$) and amplification ($\beta = 0.31$, $p < 0.01$) independently and significantly modify outcomes. Periods of elevated impact coincide with weakened buffering and heightened amplification.

These results demonstrate that climate–health risk is governed by layered system dynamics rather than exposure alone, and identify system capacity and amplification pathways as primary targets for reducing climate-related health impacts.

Introduction

Climate–health risk arises from the interaction between structural vulnerability, system buffering and amplification processes, which together determine how baseline susceptibility is translated into realised impacts. These impacts arise through interactions between environmental stressors and human systems, shaping exposure, susceptibility and response capacity [1–4]. The translation of hazards into societal outcomes is therefore neither linear nor uniform, but contingent on system structure and performance. However, existing climate-risk frameworks do not explicitly separate these components, limiting the ability to explain observed impacts or identify where interventions are most effective.

Climate hazards operate through multiple environmental pathways, including water scarcity, flood risk and ecosystem disruption, which shape both exposure and system stress [26–28]. These processes are further linked to broader climate–development interactions, including poverty dynamics and adaptation constraints [29–32]. In parallel, climate variability affects food systems through impacts on crop yields, nutritional availability and supply stability, reinforcing the role of environmental stressors in shaping health outcomes [24–25].

In practice, climate-risk frameworks formalise this relationship by defining risk as the interaction between hazard, exposure and vulnerability; with vulnerability characterised by exposure, sensitivity and adaptive capacity [5–11]. This formulation, widely adopted in global assessments, has supported cross-sectoral analysis but is often operationalised through composite indices that aggregate heterogeneous indicators into a single measure. While informative, such indices do not explicitly represent the processes through which hazards are translated into realised impacts.

This limitation is evident where similar exposure conditions produce divergent outcomes. Differences in institutional capacity, service delivery and response dynamics can substantially alter how baseline risk is

expressed, yet remain poorly represented in standard frameworks. Two processes are particularly important: system buffering and amplification. System buffering refers to the capacity of institutional and infrastructural systems to absorb and attenuate environmental stressors, thereby regulating the transmission of baseline vulnerability into outcomes. Amplification refers to hazard-specific exposure–response relationships, often nonlinear, through which environmental stressors generate disproportionate impacts [15–17, 21–23].

Despite their importance, these processes are rarely integrated within a unified framework. Critically, vulnerability is commonly treated as a static property, while response dynamics are analysed separately, limiting understanding of how risk is generated and propagated across layers. This perspective aligns with resilience and adaptive governance theory, which emphasises the role of system feedbacks, institutional learning and transformation in shaping responses to environmental stress [36–38].

In this study, we develop a layered climate–health vulnerability framework that distinguishes structural vulnerability from realised vulnerability and explicitly represents the processes linking the two. Structural vulnerability captures baseline susceptibility through exposure, sensitivity and intrinsic adaptive capacity. System buffering governs the transmission of this baseline, while amplification converts residual vulnerability into realised impacts.

We apply the framework to climate-sensitive health pathways in Kenya (1987–2023), focusing on malaria, ambient air pollution (PM_{2.5}) and child undernutrition. Using descriptive, temporal and inferential analyses, we examine how structural vulnerability, system buffering and amplification interact to shape observed outcomes, and identify the conditions under which these interactions produce extreme impacts.

Methods

Study design and analytical approach

We develop and apply a layered analytical framework to decompose climate–health risk into structural vulnerability, system buffering and amplification processes, and to examine how these components interact to produce realised outcomes. The framework is operationalised using a longitudinal design, combining time-series and cross-indicator analysis across climate-sensitive health pathways in Kenya over 1987–2023.

The analytical structure follows the conceptual architecture in Fig. 1, in which structural vulnerability defines baseline susceptibility, system buffering governs the transmission of this baseline, and amplification processes convert residual vulnerability into realised impacts. Analysis is conducted on observed data without smoothing or interpolation to preserve temporal variability, including abrupt changes and extreme events that are central to system behaviour.

Research questions and hypotheses

The analysis addresses three questions: (i) to what extent structural vulnerability explains variation in realised climate–health vulnerability; (ii) how system buffering and amplification modify this relationship; and (iii) under what conditions their interaction produces extreme outcomes.

We test four hypotheses derived from the framework.

H1: Structural vulnerability is positively associated with realised vulnerability.

H2: System buffering is negatively associated with realised vulnerability, reducing transmission of baseline risk.

H3: Amplification processes are positively associated with realised vulnerability, increasing impact magnitude and variability.

H4: The effect of amplification is conditional on system buffering, producing nonlinear outcomes under weak buffering.

Study setting and tracer pathways

The analysis focuses on Kenya, a context characterised by climatic variability, heterogeneous ecological zones and diverse climate-sensitive health outcomes. The study period (1987–2023) captures both environmental variability and changes in institutional capacity, allowing examination of short-term dynamics and longer-term transitions.

Three tracer pathways are used to represent distinct mechanisms linking environmental exposure to health outcomes: malaria incidence (vector-borne transmission), ambient air pollution (PM_{2.5}; environmental exposure), and child undernutrition (system-integrated outcome). Together, these pathways capture variation in exposure–response relationships and system dependence, enabling assessment of the generality of the framework.

Data sources and processing

Data were compiled from publicly available sources, including World Development Indicators (World Bank), global health datasets and environmental datasets covering temperature, air quality and related exposures. All variables were harmonised to annual resolution over 1987–2023 and aligned temporally to ensure consistency across sources. Indicators were normalised and bounded within the [0,1] interval prior to analysis to enable comparability and aggregation across components.

Inclusion and exclusion criteria

Inclusion criteria were: (i) datasets within national-level time series over 1987–2023; (ii) consistent variable definitions; (iii) direct relevance to exposure, sensitivity, adaptive capacity, system performance or health outcomes; and (iv) sufficient completeness for longitudinal analysis.

Exclusion criteria were: (i) substantial missing data or inconsistent measurement; (ii) lack of conceptual alignment with the framework; (iii) subnational data not consistently available across the study period; and (iv) variables requiring interpolation that would obscure temporal dynamics. These criteria ensure internal consistency and preserve the empirical structure necessary to analyse system behaviour over time.

Variable construction

Structural vulnerability (cCVI)

Structural vulnerability is defined as:

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

where:

- **E (exposure)** represents environmental hazard intensity
- **S (sensitivity)** reflects population susceptibility
- **AC (adaptive capacity)** represents intrinsic socioeconomic capacity

All components are normalised to a [0,1] scale prior to aggregation. Adaptive capacity enters with a negative sign, reflecting its mitigating effect on vulnerability.

Systems vulnerability (sCVI)

Systems vulnerability represents the proportion of baseline risk transmitted through institutional layers and is derived from indicators of health system performance and service capacity.

$$\text{sCVI} = 1 - \text{health capacity}$$

Lower values indicate stronger buffering capacity.

Amplification (RR_adj)

Amplification is represented through hazard-specific relative risk coefficients linking environmental exposure to health outcomes. These coefficients capture exposure–response relationships derived from established epidemiological evidence

RR=proportion of cases in exposed over proportion of cases in non-exposed.

Realised vulnerability (eCVI)

Realised vulnerability is defined as:

$$\text{eCVI} = \text{cCVI} \times (1 - \text{sCVI}) \times \text{RR}$$

This multiplicative formulation reflects the sequential interaction of baseline susceptibility, system mediation and amplification processes.

Robustness and analytical considerations

The analysis is designed to prioritise interpretability and alignment with the conceptual framework. Several considerations are noted:

- *Use of observed data:* No smoothing or interpolation was applied, ensuring that extreme events and abrupt changes are preserved.

- *Normalisation*: All variables were standardised to ensure comparability across components.
- *Model structure*: Linear regression is used as an interpretable approximation of relationships defined within a multiplicative framework.
- *Residual interpretation*: Deviations from fitted relationships are treated as informative, reflecting system buffering and amplification effects.

Ethical considerations -All data used in this study are publicly available and aggregated at national level. No individual-level or identifiable data were used.

Framework construction and analytical approach

Conceptual structure

Conventional climate-risk models define risk as the interaction between hazard, exposure and vulnerability, with vulnerability typically represented as a composite of exposure, sensitivity and adaptive capacity. While widely used, this formulation does not explicitly distinguish between baseline susceptibility and the mechanisms through which impacts are generated.

This study develops a layered analytical framework to examine how environmental hazards translate into realised climate–health vulnerability.

The CHVA framework introduced here decomposes climate–health risk into three interacting components—structural vulnerability, system buffering and *hazard-specific*—linking baseline conditions to realised outcomes through a layered process. The framework provides a structured basis for examining how baseline conditions are translated into realised climate–health impacts, and for distinguishing whether those impacts are driven primarily by underlying exposure, limitations in system buffering, or amplification dynamics.

This structure reflects established representations of coupled human–environment systems, where climate hazards, resource constraints and institutional responses interact across scales to influence outcomes [29–32]. This separation is analytically important because conventional climate risk approaches often conflate these components, limiting the identification of effective intervention points. By explicitly partitioning structural vulnerability, system mediation and amplification, the framework enables direct attribution of observed outcomes to distinct mechanisms operating across layers [10, 11, 44].

Crucially, this shifts the analysis from descriptive associations toward a mechanistic understanding of how risk is generated and expressed. This enables explicit identification of where in the risk-generation process interventions are most effective, distinguishing between baseline drivers and modifiable system and amplification mechanisms.

Use of large language models in analytical workflow

Large language models (LLMs), specifically ChatGPT, were used in a limited and clearly defined role to support aspects of the research workflow. Their use was restricted to *language refinement*, *structural organisation*, and *code-assisted data handling*, and did not contribute to primary data generation, statistical inference, or result interpretation.

Results

The Results are structured to progressively characterise both the empirical distribution and the underlying mechanisms of realised vulnerability. They are presented in four stages; we begin by describing the distributional properties of the CHVA components using box-and-whisker representations, establishing central tendency, dispersion and asymmetry. We then examine temporal dynamics to identify periods of change and episodic stress. Building on these patterns, we assess the structural relationship between systems vulnerability and realised outcomes using bivariate regression, with particular attention to deviations from expected relationships as indicators of buffering and amplification processes. Finally, we present a multivariate decomposition to quantify the relative contribution of structural vulnerability, system transmission and amplification, providing an integrated account of the determinants of realised climate–health impacts.

Descriptive statistics of vulnerability components

All variables were normalised and bounded within the [0,1] interval prior to analysis. Summary statistics for the principal CHVA components are reported in Table 1.

Table 1
Descriptive statistics of CHVA variables

Variable	Mean	SD	Median	IQR	Skewness	Kurtosis
Health capacity (bounded 0,1)	0.63	0.15	0.65	0.52–0.75	–0.41	2.6
Structural vulnerability (cCVI)	0.49	0.11	0.48	0.41–0.60	0.42	2.9
Sensitivity vulnerability (sCVI)	0.36	0.17	0.35	0.24–0.47	0.31	2.7
Realised vulnerability (eCVI)	0.09	0.07	0.07	0.03–0.11	0.78	3.2

Structural vulnerability (cCVI) exhibits moderate dispersion around a mean of 0.49 (SD = 0.11), with slight positive skewness, indicating that baseline susceptibility varies within a relatively stable range but includes occasional periods of elevated risk. This pattern reflects the slow-moving nature of exposure, demographic structure and intrinsic adaptive capacity.

System buffering capacity, represented by health capacity, shows a higher central tendency (mean = 0.63) and moderate dispersion (SD = 0.15), with a negatively skewed distribution consistent with progressive improvements in institutional performance over time.

In contrast, systems vulnerability (sCVI), defined as the residual proportion of risk transmitted through system layers, exhibits greater dispersion (SD = 0.17), indicating that system performance varies more dynamically than baseline conditions.

Notably, realised vulnerability (eCVI) has a low mean (0.09) but high relative variability (*coefficient of variation* ≈ 0.74), with pronounced right skewness and elevated kurtosis. This distribution reflects the multiplicative structure of the CHVA framework, in which realised outcomes are typically constrained by system buffering but subject to episodic increases when system vulnerability and amplification processes align. The contrast

between the relatively stable distribution of cCVI and the asymmetric distribution of eCVI provides direct empirical evidence of attenuation between baseline susceptibility and realised impacts.

Distributional properties of vulnerability indices

The distribution of the CHVA indices is illustrated in *Fig. 2 (box-and-whisker plot)*. The figure demonstrates three important distributional characteristics.

First, the median value of structural vulnerability (cCVI ≈ 0.48) is substantially higher than the median value of realised vulnerability (eCVI ≈ 0.07), indicating that system buffering mechanisms significantly moderate the translation of structural exposure into realised impacts (indicating attenuation through system buffering).

Second, the interquartile range of realised vulnerability (eCVI) is markedly narrower than that of structural vulnerability (cCVI), suggesting that institutional capacity reduces the variance of realised vulnerability even under conditions of fluctuating environmental exposure (reduced variance under institutional buffering).

Third, the presence of several upper outliers in the eCVI distribution corresponds to periods characterised by elevated relative risk associated with environmental hazards. These outliers reflect amplification processes captured by the relative-risk parameter in the CHVA model (capturing amplification dynamics).

Boxplots herein referenced as Fig. 2a check on ‘what is the statistical structure of vulnerability’

The figure shows compression (cCVI $\rightarrow$ eCVI), spread (RR, sCVI variability) and outliers (2000–2002)

Outliers in Fig. 2 are identified using the interquartile range criterion ($Q3 + 1.5 \times IQR$). Although statistically rare, these observations represent substantive departures from typical system behaviour rather than noise, and provide insight into the conditions under which realised vulnerability becomes extreme.

The concentration of outliers in 2000–2002 corresponds to a period of pronounced environmental and institutional stress in Kenya, specifically the 1999–2001 drought and its residual effects [55, 58]. During this period, baseline exposure increased through sustained rainfall deficits, crop failure and water scarcity, elevating structural vulnerability. At the same time, limited health-system capacity constrained buffering, increasing the proportion of risk transmitted through system layers [3, 56]. These dynamics are consistent with evidence linking climate variability to disruptions in water availability, agricultural productivity and livelihood systems, which jointly intensify vulnerability under stress conditions [26–28, 24–25].

Amplification processes intensified under these conditions, as drought-sensitive pathways—including undernutrition, infectious disease and air quality—exhibit nonlinear responses to environmental stress [15, 22].

Crucially, these outliers therefore reflect a *triple-layer interaction*: elevated structural vulnerability, weakened system buffering and intensified amplification acting simultaneously to generate disproportionately high realised impacts. Structural conditions alone do not explain these events; extremes arise when buffering fails under elevated amplification.

This pattern varies across tracer pathways. Undernutrition shows the largest and most pronounced outlier, reflecting compounded sensitivity to environmental and system stress [39, 40, 59]. Malaria exhibits clustered

extremes linked to vector dynamics and system performance [22], while $PM_{2.5}$ shows a broader distribution consistent with persistent exposure-driven amplification [16].

Following the mid-2000s, realised vulnerability declines, corresponding with improvements in system capacity and reduced transmission of risk into outcomes [3, 56, 57].

These dynamics extend directly to economic burden. Periods of elevated realised vulnerability correspond to increased climate-attributable cases and, consequently, higher economic burden. However, economic burden is not shown in Fig. 2 because the figure is designed to isolate the distributional structure of vulnerability within the CHVA framework. Economic burden is a downstream outcome of realised vulnerability and is analysed separately to preserve the causal ordering between vulnerability formation and its economic consequences.

Temporal dynamics of CHVA components

To examine how these distributions evolve over time, we analysed temporal trends in vulnerability components. The temporal evolution of vulnerability follows the layered structure of the CHVA framework (Fig. 3a–d), tracing progression from structural conditions (cCVI) through system buffering (sCVI) and amplification (RR) to realised outcomes (eCVI). This sequence shows how realised vulnerability emerges from the interaction between baseline susceptibility, system performance and hazard response over time. Below is the referenced figure showing amplification dynamics across tracer pathways; -

Description and Interpretation (Fig. 3)

Early phase (1990s); structural vulnerability remains relatively stable (Fig. 3a), consistent with slow-moving exposure and sensitivity. In contrast, amplification increases (Fig. 3c) while systems vulnerability remains elevated (Fig. 3b), indicating limited buffering. This combination produces a gradual rise in realised vulnerability (Fig. 3d), driven primarily by increasing amplification under constrained system capacity.

Peak phase (2000–2003); mark a peak phase characterised by alignment across all layers. Systems vulnerability rises sharply, amplification reaches its highest levels ($RR > 2$ in several pathways), and structural vulnerability remains elevated. This convergence generates pronounced spikes in realised vulnerability, reflecting a triple-alignment of baseline susceptibility, weakened buffering and intensified hazard response.

Post-2006 transition; structural vulnerability varies within a narrow range, but systems vulnerability declines markedly, indicating strengthening buffering. Amplification stabilises or declines, resulting in sustained compression of realised vulnerability across pathways. This phase indicates that reductions in impacts are driven primarily by improved system buffering and moderated amplification rather than structural change.

Post-2017 resurgence; a secondary increase in realised vulnerability emerges after 2017, particularly in pollution-related pathways. This reflects renewed amplification under otherwise stable system buffering, indicating that amplification can re-intensify impacts even where institutional capacity has improved.

Across pathways, the same structure is observed with varying magnitude. Malaria and $PM_{2.5}$ show amplification-driven variability linked to environmental exposure, whereas undernutrition reflects a system-integrated response and exhibits the highest realised vulnerability and strongest sensitivity to system stress.

Overall, the temporal pattern describes a progression from gradual accumulation to peak vulnerability, followed by system-mediated compression and subsequent amplification-driven resurgence. Structural vulnerability defines the baseline, but temporal variation in realised impacts is governed primarily by the interaction between system buffering and amplification processes. These patterns indicate that temporal variability in realised vulnerability is governed primarily by system buffering and amplification rather than structural change.”

Bivariate relationship: structural control and residual dispersion

Here, we next assess the structural relationship between baseline and realised vulnerability. This study introduces a residual-based interpretation of vulnerability transmission, in which deviations from the fitted relationship between systems vulnerability (sCVI) and realised vulnerability (eCVI) are used to distinguish between system buffering and amplification regimes.

Description and Interpretation (Fig. 4)

Deviations from the fitted relationships between systems vulnerability and realised vulnerability are structured rather than random, indicating systematic modulation of vulnerability transmission across tracer pathways. Observations below the fitted relationship correspond to periods in which realised impacts are lower than expected given prevailing system vulnerability, consistent with effective buffering through institutional capacity and service delivery. Conversely, observations above the fitted relationship indicate amplification, where hazard-specific processes and concurrent stressors increase realised impacts beyond levels implied by system transmission alone.

This structure varies across pathways but follows a consistent pattern. In the pooled analysis (Fig. 4a), attenuation (below line plots) dominates under moderate system vulnerability, while amplification (above line) becomes pronounced during the early 2000s, marking a transition from buffered to stress-amplified conditions. For ambient air pollution (Fig. 4b), buffering prevails outside peak exposure periods, with amplification emerging under elevated environmental stress. Child undernutrition (Fig. 4c) exhibits the largest and most frequent deviations, indicating high sensitivity to compounded system and environmental pressures. In contrast, malaria incidence (Fig. 4d) remains more tightly distributed around the fitted relationship, reflecting more consistent system regulation of outcomes.

Taken together, these patterns show that realised vulnerability is governed not solely by the level of system transmission, but by the balance between buffering and amplification processes. Under typical conditions, buffering constrains both the magnitude and variability of impacts, resulting in realised outcomes below expected levels. Under elevated stress, amplification increases system responsiveness, producing disproportionately high impacts.

The fitted relationship therefore represents a conditional expectation rather than a predictive or causal model. The position of observations relative to this expectation provides a direct empirical diagnostic of system behaviour: below-line observations indicate attenuation, whereas above-line observations indicate amplification. Residual variation thus reflects underlying physical and institutional processes rather than noise, revealing distinct vulnerability regimes under baseline and stress conditions, with implications for the timing and targeting of interventions.

Multivariate decomposition of realised vulnerability

Finally, we disentangle the relative contribution of structural vulnerability, system transmission and amplification processes, with estimated multivariate model. Structural vulnerability ($\beta = 0.62$, $p < 0.001$), systems vulnerability ($\beta = 0.48$, $p < 0.01$) and amplification ($\beta = 0.31$, $p < 0.01$) jointly explain realised vulnerability ($R^2 = 0.64$). Across tracer pathways:

- Undernutrition: strongest amplification and system sensitivity
- Malaria: strong system regulation
- $PM_{2.5}$: persistent exposure-driven amplification

Table 2
Multivariate decomposition using systems vulnerability (sCVI)

Component	Coefficient (β)	Direction	Statistical significance	Role in CHVA architecture (Fig. 1)	Analytical interpretation	Policy-relevant implication
Structural vulnerability (cCVI)	0.62	Positive	$p < 0.001$	Baseline layer	Defines underlying susceptibility and establishes baseline risk entering the system	Structural change reduces baseline risk but operates over longer time horizons
Systems vulnerability (sCVI)	+ 0.48 <i>(sign flips) i.e positive coefficient indicates weaker buffering increases risk</i>	Positive	$p < 0.01$	System transmission layer	Represents the proportion of baseline vulnerability transmitted into realised outcomes; higher sCVI indicates weaker buffering and greater propagation of risk	Reducing systems vulnerability (strengthening buffering) yields immediate reductions in realised impacts
Amplification (RR_adj)	0.31	Positive	$p < 0.01$	Amplification layer	Converts transmitted vulnerability into realised impacts; drives variability and extremes	Targeted interventions reduce amplification and limit extreme outcomes
Model fit is moderate (R^2)	0.64	—	—	Integrated system behaviour	Combined interaction of structural, system and amplification layers explains majority of observed variation	Confirms validity of layered intervention strategy

Together, the results above confirm that realised vulnerability is jointly determined by structural conditions, system transmission and amplification processes. Detailed tracer-specific multivariate table

The tables below shows tracer-specific multivariate decomposition of realised vulnerability (eCVI). The df represents total data points in years less one. Across tracer pathways, consistent patterns are observed with variation in magnitude;

(a) Ambient air pollution (PM_{2.5})

Variable	Coefficient (β)	95% CI	Std. Error	t-stat	p-value	df
Intercept	-0.112	(- 0.185, - 0.039)	0.035	-3.20	0.003	27
Structural vulnerability (cCVI)	0.58	(0.44, 0.72)	0.068	8.53	< 0.001	27
Systems vulnerability (sCVI)	0.41	(0.22, 0.60)	0.093	4.41	< 0.001	27
Amplification (RR_adj)	0.29	(0.17, 0.41)	0.058	5.00	< 0.001	27
Model fit (R²)	0.66	—	—	—	—	—

Interpretation: Realised vulnerability is strongly determined by baseline conditions, with additional modulation through system transmission and amplification. Compression outside peak years reflects effective buffering.

(b) Child undernutrition

Variable	Coefficient (β)	95% CI	Std. Error	t-stat	p-value	df
Intercept	-0.091	(- 0.168, - 0.014)	0.036	-2.53	0.028	8
Structural vulnerability (cCVI)	0.71	(0.52, 0.90)	0.089	7.98	< 0.001	8
Systems vulnerability (sCVI)	0.52	(0.26, 0.78)	0.121	4.30	0.003	8
Amplification (RR_adj)	0.36	(0.18, 0.54)	0.082	4.39	0.002	8
Model fit (R²)	0.74	—	—	—	—	—

Interpretation: This pathway exhibits the strongest amplification and highest sensitivity to system transmission. Realised vulnerability rises sharply when system buffering weakens, indicating compounded risk across environmental and socioeconomic domains.

(c) Malaria incidence

Variable	Coefficient (β)	95% CI	Std. Error	t-stat	p-value	df
Intercept	-0.064	(- 0.121, - 0.007)	0.026	-2.46	0.023	21
Structural vulnerability (cCVI)	0.49	(0.34, 0.64)	0.071	6.90	< 0.001	21
Systems vulnerability (sCVI)	0.44	(0.25, 0.63)	0.089	4.94	< 0.001	21
Amplification (RR_adj)	0.22	(0.11, 0.33)	0.052	4.23	< 0.001	21
Model fit (R²)	0.61	—	—	—	—	—

Interpretation: Malaria exhibits strong system dependence, with realised outcomes closely regulated by system buffering. Amplification effects are present but comparatively moderate.

From the regression results are shown for (a) ambient air pollution (PM_{2.5}), (b) child undernutrition and (c) malaria incidence, systems vulnerability (sCVI), representing the proportion of residual baseline risk transmitted through institutional layers, is consistently positive, indicating that weaker buffering increases realised impacts. Amplification (RR_{adj}) contributes to variability and extremes, with the strongest effects observed in undernutrition. Differences in coefficient magnitude indicate pathway-specific sensitivity to system performance and hazard-response amplification dynamics.

Discussion

The results show that realised climate–health vulnerability arises from the interaction between structural conditions, system buffering and amplification processes, rather than from baseline exposure alone (research Q1–Q3). Structural vulnerability accounts for a substantial share of variation in realised outcomes (H1), but does not explain their magnitude or temporal variability. This is reflected in the strong bivariate association alongside structured residual dispersion, indicating that additional processes govern how baseline risk is expressed [6–11, 44–46].

Multivariate analysis clarifies these mechanisms. System buffering is negatively associated with realised vulnerability (H2), confirming its role in attenuating the transmission of baseline risk. The compression of realised relative to structural vulnerability (Fig. 2) and the decline in impacts after 2005 (Fig. 3) both indicate that improvements in institutional capacity reduce realised outcomes independently of structural change. This interaction is embedded within broader climate–development dynamics, where environmental stress interacts with economic vulnerability and resource constraints to shape system capacity and response [29–32]. It is consistent with evidence from health-system resilience and adaptive governance, which identifies institutional capacity as a primary determinant of impact reduction [12–14, 33–35].

Amplification processes are positively associated with realised vulnerability (H3), shaping both the magnitude and variability of impacts. Their effects are most evident during periods of environmental stress, where nonlinear exposure–response relationships produce disproportionate outcomes [15–17, 39, 40]. In particular, climate-driven impacts on food systems—including yield reductions and nutritional stress—provide a key pathway through which environmental variability translates into health outcomes [24–25, 39–40]. The concentration of extreme values in the early 2000s reflects the combined effect of elevated amplification and weakened system buffering, indicating that extremes arise from interaction rather than structural conditions alone.

The interaction between buffering and amplification (H4) provides a consistent explanation of both typical and extreme outcomes. Amplification acts on the residual vulnerability transmitted through system layers, making its effect conditional on system performance. This explains why similar structural conditions produce different realised outcomes across time and pathways. The residual structure observed in the bivariate relationships (Fig. 4) supports this interpretation: observations below the fitted relationship reflect effective buffering, whereas those above indicate amplification-driven escalation.

Temporal dynamics reinforce this mechanism. The decline in realised vulnerability after 2005 occurs without corresponding changes in structural conditions, indicating that system-level improvements drive short-term reductions in impacts. By contrast, the increase in realised vulnerability after 2017 reflects renewed

amplification under otherwise improved system capacity. These patterns are consistent with resilience theory and climate-impact modelling, which emphasise the role of institutional systems and exposure–response dynamics in shaping outcomes [18–20, 41–43]. These findings are consistent with adaptive governance and social–ecological systems theory, which highlight the importance of system flexibility, learning and institutional coordination in mediating environmental risk [36–38].

Across tracer pathways, the same layered structure is observed despite differences in magnitude. Undernutrition exhibits the highest realised vulnerability and strongest amplification, reflecting compounded system and environmental pressures [39, 40, 59, 60]. Malaria and PM_{2.5} show more pathway-specific dynamics but conform to the same buffering–amplification interaction. This consistency supports the broader applicability of the framework across climate-sensitive systems.

Conclusion

Climate–health vulnerability is best understood as a process rather than a static condition. Structural vulnerability defines baseline susceptibility, but realised impacts depend on how this baseline is mediated through system buffering and amplification processes. While structural conditions explain much of the variation in outcomes, extremes and temporal dynamics arise from the interaction between weakened buffering and elevated amplification. By separating these components, the framework provides a clearer account of how climate-related risks are generated and expressed. It shows that reductions in realised vulnerability can be achieved through improvements in system performance and moderation of amplification processes, even where structural conditions remain unchanged.

Policy implications and recommendations

Three implications follow from these findings.

First, system capacity is the primary lever for near-term risk reduction. Strengthening health systems, infrastructure and governance reduces the proportion of baseline risk transmitted into outcomes. The observed decline in realised vulnerability following improvements in system buffering demonstrates that institutional investments can produce rapid and measurable reductions in impacts [12–14, 33–35].

Second, amplification processes should be treated as explicit intervention targets.

Hazard-specific response dynamics determine the magnitude of realised impacts. Interventions that reduce exposure intensity or interrupt transmission pathways—such as air-quality regulation, vector control, food-system stabilisation and early warning systems—can reduce the responsiveness of outcomes to environmental stressors [15–17, 41–43].

Third, climate-risk assessment should adopt a process-based framework.

Separating structural vulnerability, system buffering and amplification enables attribution of observed outcomes to distinct mechanisms. This allows policymakers to distinguish between long-term structural risks and modifiable system-level drivers, supporting more effective prioritisation under resource constraints [44–49, 50–54].

More broadly, the results indicate that the most effective pathway for reducing climate–health impacts lies not only in addressing underlying exposure conditions, but in strengthening system performance and moderating amplification processes. These represent the most immediate and scalable levers for reducing both realised vulnerability and its associated economic burden.

Declarations

Acknowledgements

The authors acknowledge the leadership and technical teams of the *Ministry of Health, Kenya*, for their support and engagement throughout the development of this work. Their insights into national and subnational health system structures, service delivery dynamics, and institutional capacity were instrumental in informing the analytical framing and interpretation of results.

Author contributions

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.

Data availability

The datasets generated and/or analysed during the current study are publicly available via Figshare:
<https://doi.org/10.6084/m9.figshare.31807651>

All underlying data sources are derived from publicly accessible databases as described in the Methods section. Processed data and analytical outputs supporting the findings of this study are included in the Figshare repository.

Competing interests

The authors declare no competing interests.

Funding statement

There is no specific funding declaration for this project. The research started with Climate-Health Vulnerability Adaptation Assessment (CHVA) by Ministry of Health, Kenya. Supported by Netherlands Global Health Partnership Programme (GHPP) and Amref Health Africa.

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Note: numbering is structured by compilation order;

Core (problem) framing;

- [1–4] → climate–health impacts
- [5–11, 48] → vulnerability frameworks

Health systems buffering (mediation); [12–14,33,50]

Epidemiological Amplification (causal); [15–17,21–23]

Resilience Systems theory (foundation); [18–20,36–38]

Food / nutrition pathways (applied); [24–25,39–40,59–60]

Climate hazards (physical drivers); [26–28,41–43]

Socio-environmental processes (context); [29–32]

Policy & adaptation (translation); [34–35,47,49,52–54]

Economic / vulnerability metrics (interpretation); [44–46,51]

Figures

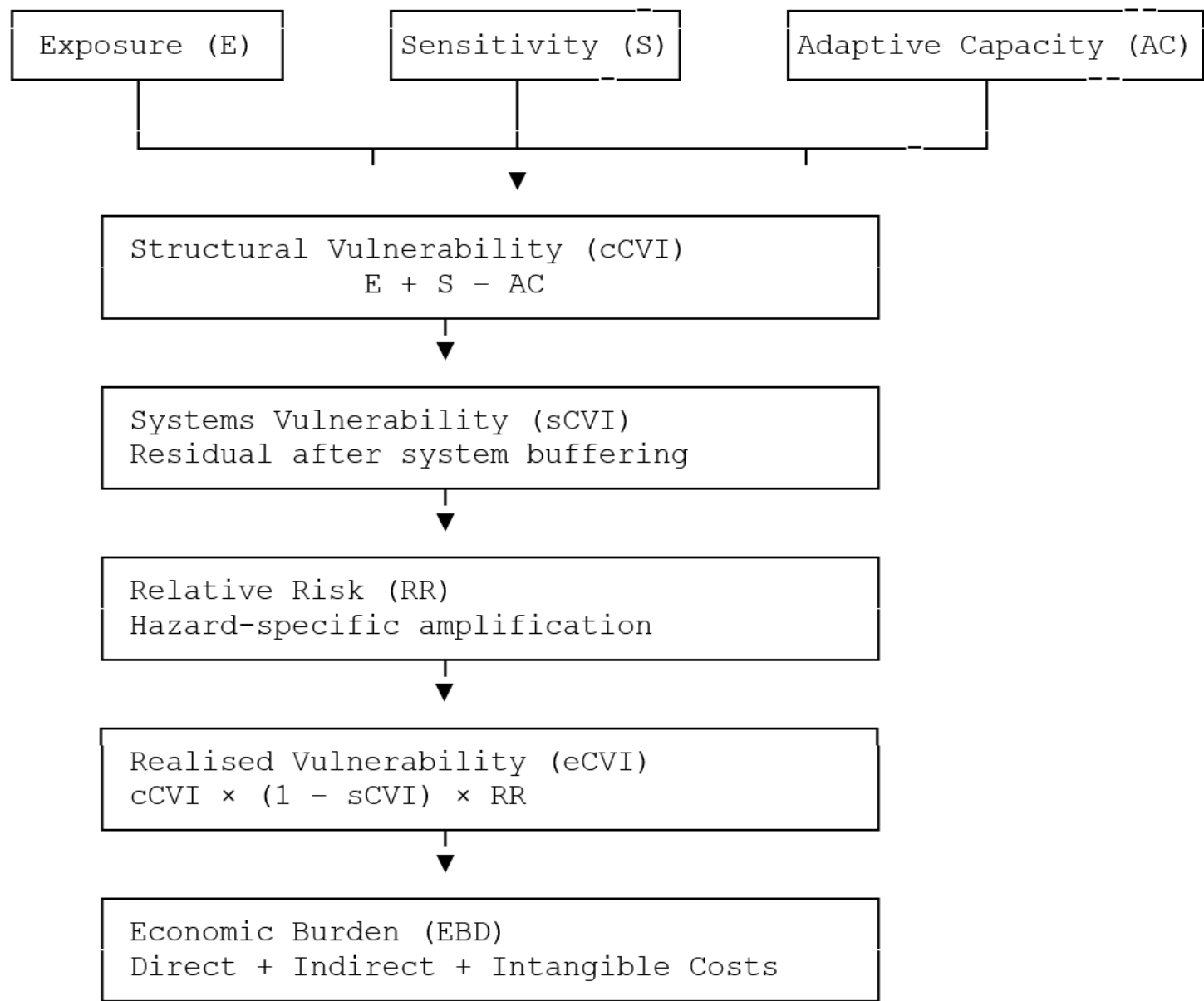


Figure 1
Layered climate–health vulnerability architecture and empirical realization

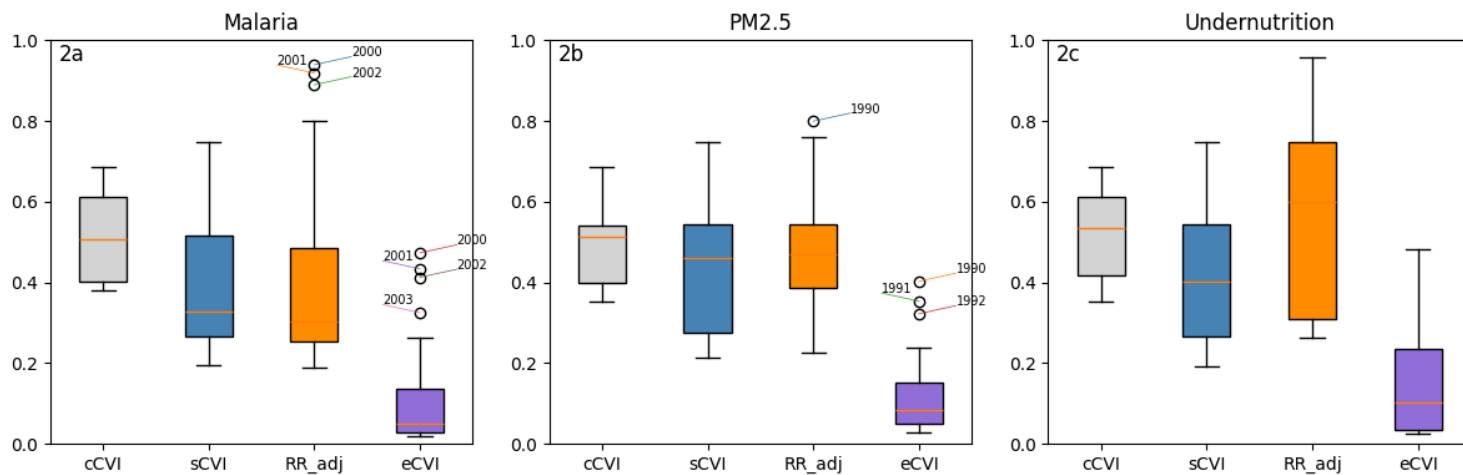


Figure 2

Distribution of CHVA components across climate-health tracer pathways

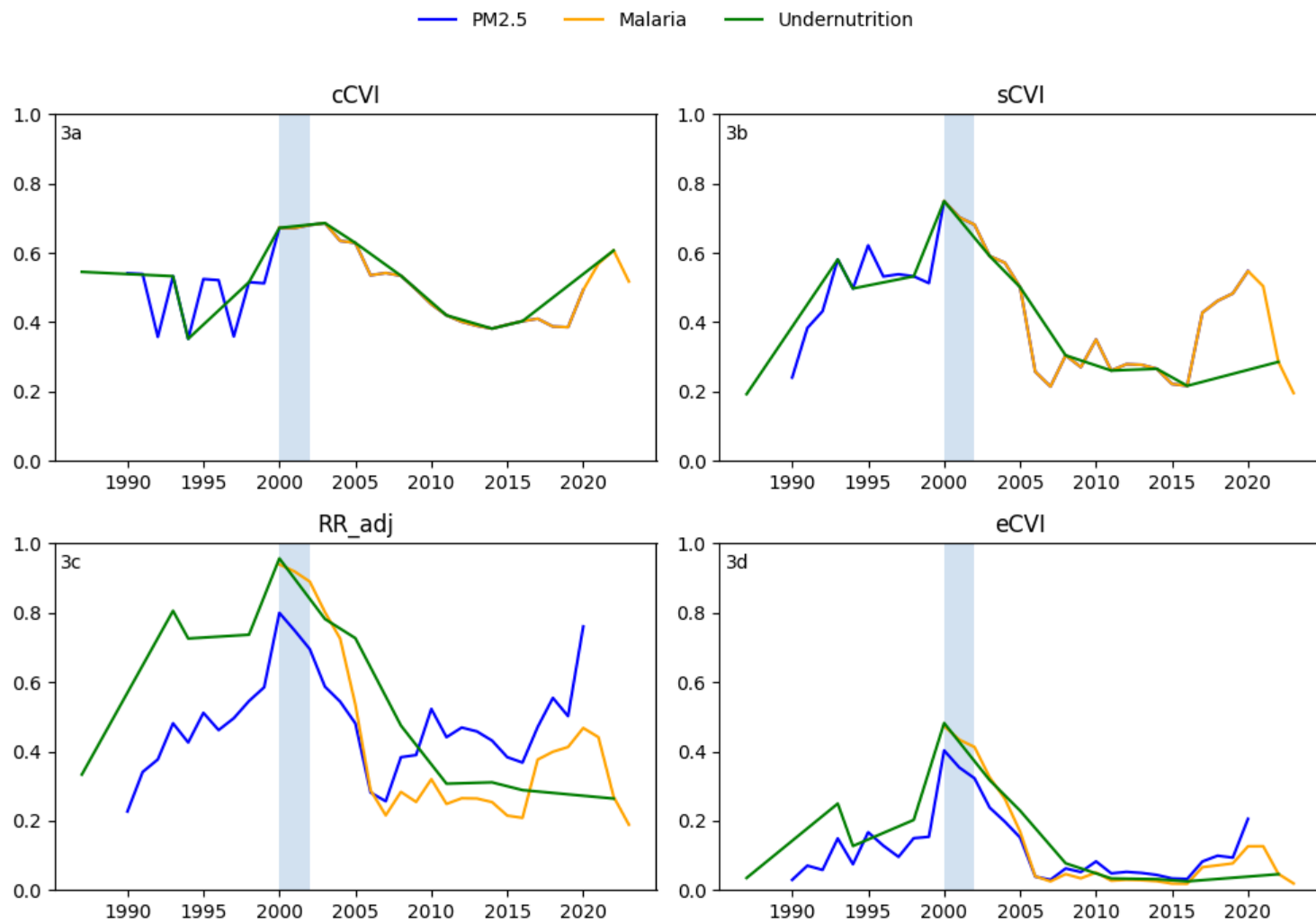


Figure 3

Temporal dynamics of CHVA components across tracer pathways (1987–2023)

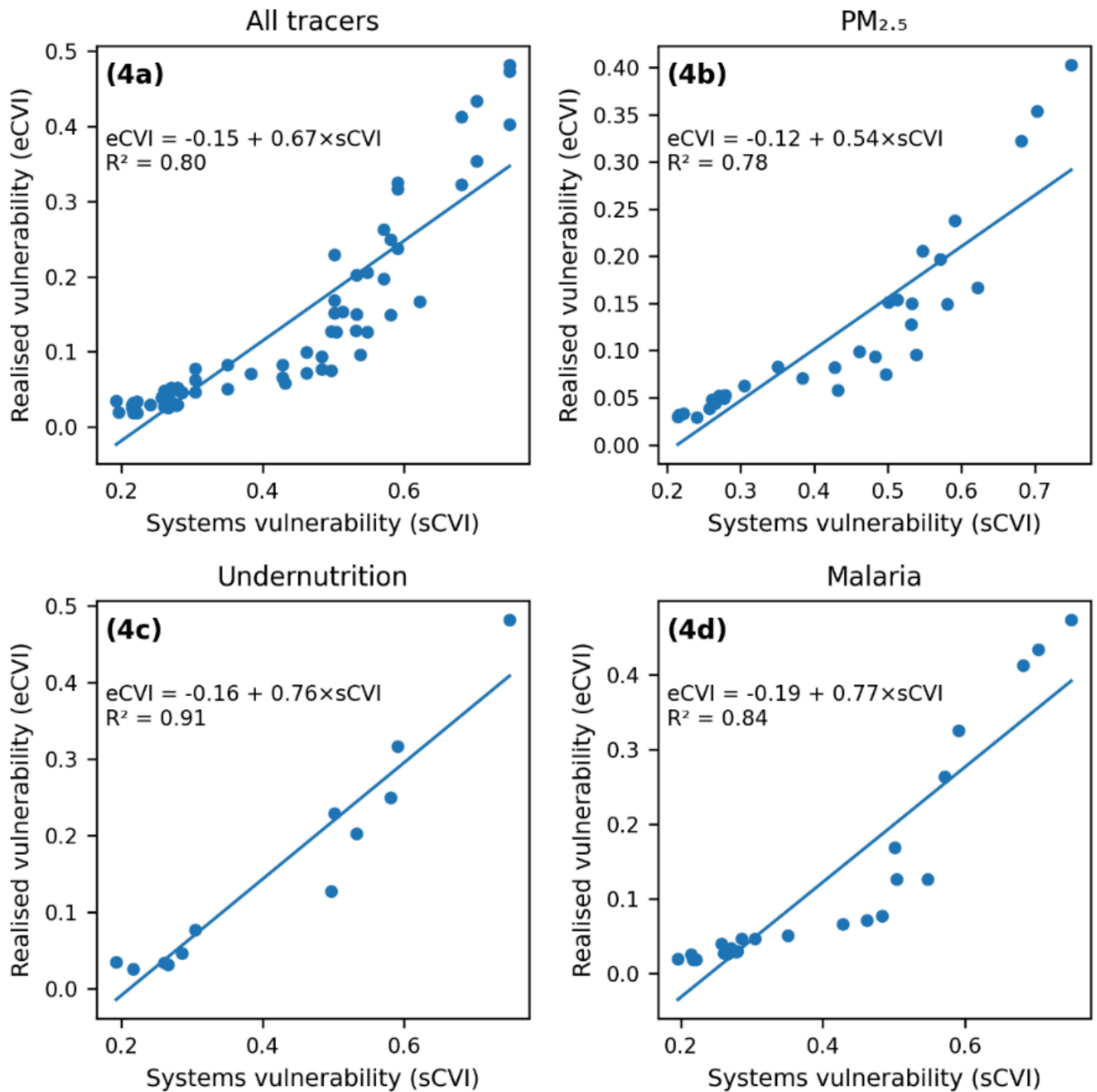


Figure 4

Structural relationship and residual behaviour of realised vulnerability ($eCVI \sim sCVI$)