

Vulnerability to flood-sensitive environmentally persistent zoonoses: a scoping review

Authors

Ellie A. Delight^{a*}, Kelly Moon^b, Wendy Sharpe^a, Joseph Wells^a, Emilia Johnson^c, Lara G. Moussa^a, Aidan Desjardins^b, Bárbara I. A. Xavier^d, Fabiana Almerinda G. Palma^d, Sara Macbride-Stewart^e, Federico Costa^d, Robert Dreibelbis^{a,^}, Max T. Eyre^{a,^}

Affiliations

^a Department of Disease Control, London School of Hygiene and Tropical Medicine, London, United Kingdom

^b School of Biological Sciences, Queen's University Belfast, Belfast, United Kingdom
School of Civil Engineering, University of Leeds, Leeds, United Kingdom

^c School of Biodiversity, One Health and Veterinary Medicine, University of Glasgow, Glasgow, United Kingdom

^d Collective Health Institute, Federal University of Bahia, Salvador, Brazil

^e School of Social Sciences, Cardiff University, Cardiff, United Kingdom

*Corresponding author: ellie.delight@lshtm.ac.uk, London School of Hygiene and Tropical Medicine, Keppel St, London, United Kingdom, WC1E 7HT

[^]Contributed equally

Abstract

Background: Environmentally persistent zoonoses (EPZs), such as leptospirosis and melioidosis, are caused by pathogens that can survive outside of a host for over 48

hours. Their transmission is sensitive to climate hazards such as flooding, which can disperse pathogens and increase opportunities for spillover. Risk of exposure to EPZs during flooding is influenced by the vulnerability of exposed populations, which is shaped by social, economic, infrastructural, and environmental conditions. Understanding vulnerability to EPZs is therefore important for identifying populations most at risk during flooding and informing targeted disease control and equitable climate adaptation. We aimed to map how vulnerability is defined and measured in studies of flood-sensitive EPZs, characterise the vulnerability and flooding-related hazard indicators used, and assess how vulnerability and hazard were studied together.

Methods: This scoping review followed PRISMA-ScR guidance. Eleven bibliographic databases were searched (May 2025, updated January 2026) for epidemiological studies quantifying associations between vulnerability or hazard indicators and human health outcomes for eight EPZs. Data on study characteristics, definitions of vulnerability, vulnerability and hazard indicators, and methods used to study vulnerability and hazard together were extracted and synthesised.

Results: Seventy-eight studies were included. Only 16 explicitly used the terms "vulnerability" or "vulnerable," with heterogeneous usage. Vulnerability indicators (n=444) clustered into six domains but were dominated by sociodemographic characteristics (n=215), particularly sex/gender and age, while housing, environmental, infrastructural and health-service domains were less frequently represented. Hazard indicators (n=138) were dominated by broad meteorological (n=52) and seasonal (n=38) proxies. Most studies were cross-sectional, ecological or case-control, with relatively few time-series or cohort studies. Evidence was heavily concentrated on leptospirosis (41/78 studies). Vulnerability and hazard were generally

treated as separate determinants of risk, with limited conceptual or analytical integration; only four studies formally tested interactions between them.

Conclusion: Vulnerability in EPZ research is inconsistently conceptualised and narrowly measured, with limited consideration of the environmental, infrastructural and service-level conditions that are particularly important for diseases transmitted at the human–animal–environment interface. Overall, the literature remains largely focused on identifying separate environmental and population risk factors rather than explaining how flooding produces unequal risks across populations. Flooding-related hazards are often characterised using broad proxies, while study designs and limited consideration of vulnerability and hazard together constrain understanding of who is most at-risk during flooding and why. Future research should use clearer vulnerability frameworks, measure relevant domains more comprehensively, improve characterisation of flooding-related hazards, and use interdisciplinary study designs capable of examining vulnerability and hazard together. This will strengthen evidence for targeted disease control and equitable climate adaptation.

Introduction

Environmentally persistent zoonoses (EPZs) are zoonotic diseases caused by pathogens that can survive outside of a host for more than 48 hours (1). Despite causing substantial public health burdens, many EPZs remain neglected in research, surveillance and control efforts (1-6). Leptospirosis is reported to be the most widespread zoonosis, with an estimated 1 million cases and 60,000 deaths per year (2, 7). The causative agent, *Leptospira* spp., can persist in soil for at least 2 weeks and in water for up to 4 weeks (2). Melioidosis is less widespread at an estimated 165,000 cases per year, but a high case fatality rate results in an estimated 89,000

annual deaths (3, 8). The causative agent, *Burkholderia pseudomallei*, persists in soil and water for up to 7 days (9).

EPZs can be transmitted to humans both directly from infected animals and indirectly through environmentally mediated transmission, whereby infected animals shed pathogens into the environment and humans become infected through contact with, ingestion of, or inhalation of contaminated environmental sources (1, 10). The environmentally mediated transmission pathway can often predominate direct animal-human transmission (11, 12). This complicates disease control, as environmental reservoirs maintain opportunities for human infection independently of direct animal contact (1, 10).

Climate hazards can alter the persistence and distribution of pathogens in the environment, increasing opportunities for human transmission (1, 13). Flooding is particularly important for several EPZs, such as leptospirosis, as heavy rainfall and flooding can mobilise pathogens from soil and contaminated environments and disperse them into surrounding areas (10-19).

However, climate hazards alone do not determine risk of human infection with EPZs. According to the Intergovernmental Panel on Climate Change (IPCC) risk framework (Table 1), risk arises from the interaction of hazard with the exposure and vulnerability of the affected population (20). Here, vulnerability is defined as the sensitivity, susceptibility, or lack of capacity of individuals and populations to cope and adapt (20), and is shaped by local social, economic, environmental and infrastructural conditions (21-24). For EPZs, these conditions can influence risk at multiple points along the human–animal–environment interface. Inadequate water, sanitation and drainage can increase contact with contaminated floodwater (25-27), poor refuse collection and degraded local environments can support animal reservoirs such as rodents (28), and

housing conditions can increase contact with contaminated soil or water (29, 30). Limited access to healthcare can also increase the consequences of infection (23). These pathways illustrate the multidimensional nature of vulnerability to EPZs and how it can contribute to unequal health risks during flooding.

Table 1. IPCC framework definitions of hazard, exposure, and vulnerability (20)

Component of risk	Definitions
Hazard	The potential occurrence of a natural or human-induced physical event or trend that may cause loss of life, injury, or other health impacts, as well as damage and loss to property, infrastructure, livelihoods, service provision, ecosystems and environmental resources.
Exposure	The presence of people; livelihoods; species or ecosystems; environmental functions, services, and resources; infrastructure; or economic, social, or cultural assets in places and settings that could be adversely affected.
Vulnerability	The propensity or predisposition to be adversely affected. Vulnerability encompasses a variety of concepts and elements, including sensitivity or susceptibility to harm and lack of capacity to cope and adapt.

Robust evidence is therefore needed on how vulnerability and flooding-related hazards jointly shape risk to identify which populations are disproportionately affected and understand the pathways that generate this risk. Better understanding can inform equitable disease-control and climate adaptation interventions aimed at reducing vulnerability to flood-related health impacts and strengthening resilience to climate change (31). However, across the wider climate-health and infectious disease literature, vulnerability is often under-represented or poorly distinguished from other components of risk (32, 33), and when measured, is often reduced to a limited set of individual-level characteristics (34-36). How vulnerability is conceptualised and measured specifically in research on flood-sensitive EPZs, and how it is considered alongside flooding-related hazards, has not been systematically characterised.

Without this understanding, it is difficult to assess whether the existing evidence base can explain why flooding generates unequal EPZ risks across populations or identify where disease-control and adaptation measures should be targeted.

In this study, we aimed to map how vulnerability is conceptualised and measured in EPZ research, characterise the vulnerability and flooding-related hazard indicators used, and assess the extent to which their joint effects have been examined, including the conceptual and analytical approaches used. In doing so, we sought to identify evidence gaps and priorities for research to support flood-sensitive EPZ prevention and control in the context of climate change.

Methods

Protocol and registration

The scoping review was informed by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) (37) and is registered on the Open Science Framework (<https://osf.io/e8tmg>).

EPZ identification

Flood-sensitive EPZs were identified based on three criteria derived from previous reviews (1, 13). First, the disease must be zoonotic (transmissible from animals to humans). Second, the causative pathogen must persist in the environment for at least 48 hours after being shed and remain capable of causing subsequent human infection (1). Third, transmission must be “flood-sensitive” whereby transmission is exacerbated by flooding or precipitation (13). We included precipitation as it is a key driver of flooding and often used as a proxy measure for flood-generating conditions (38). Eight target EPZs were identified (Table 2).

136 **Table 2. Flood-sensitive environmentally persistent zoonoses**

Environmentally persistent zoonoses	Zoonotic pathogen*	Evidence of exacerbation by climate hazard†	Environmental transmission pathway**
Campylobacteriosis	<i>Campylobacter jejuni</i> and <i>C. fetus</i>	Flooding and precipitation	Ingestion of contaminated water
Cryptosporidiosis	<i>Cryptosporidium parvum</i> (most common zoonotic species, but many others exist)	Flooding and precipitation	Ingestion of contaminated food or water
E. coli	<i>Escherichia coli</i>	Flooding and precipitation	Ingestion of contaminated food or water
Giardiasis	<i>Giardia duodenalis</i>	Flooding and precipitation	Ingestion of contaminated food or water
Leptospirosis	<i>Leptospira</i> spp.	Flooding and precipitation	Ingestion or contact of cuts in skin with contaminated water or soil
Melioidosis	<i>Burkholderia pseudomallei</i>	Flooding and precipitation	Ingestion, inhalation, or contact of cuts in skin with contaminated water or soil
Salmonellosis	<i>Salmonella enterica</i> Dublin, <i>S. enterica</i> Enteritidis, <i>S. enterica</i> Typhimurium, <i>S. enterica</i> choleraesuis	Flooding and precipitation	Ingestion of contaminated food or water
Toxoplasmosis ¹	<i>Toxoplasma gondii</i>	Precipitation	Ingestion of contaminated food, water, or soil

137 Based on evidence from *(1) and †(13). *Relationship between flooding-related hazard and indirect transmission
138 pathway. ¹ Toxoplasmosis was retained as while direct evidence of sensitivity to flooding is lacking, *T. gondii*
139 oocysts can persist in soil for 12–18 months (39) and are transmitted by ingestion of contaminated soil and water,
140 a route similar to indirect transmission of other flood-sensitive EPZs.

Study eligibility criteria

The eligibility criteria are outlined in Table 3.

Table 3. Eligibility criteria

Criteria	Included	Excluded
Publication period	Studies published over any period	NA
Location and scale	Studies based within all countries and settings (i.e., urban and rural)	Studies in which the unit of analysis was the country or larger (not allowing assessment of within-population vulnerability)
Language	Studies published in English, Portuguese, Spanish, and French	Articles published in other languages
Study type	Empirical quantitative studies using primary or secondary data	- Literature reviews, conference abstracts, opinions, editorials, commentaries, or other non-empirical studies - Qualitative studies - Prediction, forecasting, simulation, or modelling studies without empirical human outcome data
Flooding or rainfall hazard	- Studies including variables related to flooding or precipitation, including: - Meteorological indicators (e.g., rainfall in mm/day) - Environmental indicators linked to flooding or precipitation exposure (e.g., wet season, digital elevation models, household elevation, or proximity to rivers where explicitly linked to flooding risk)	- Studies focused exclusively on other meteorological or climate hazards (e.g., landslides, storms, droughts, temperature, fires, heatwaves, natural cover change, ocean climate change, sea level, warming) - Studies referring to seasonality without explicit relevance to flooding or precipitation - Studies in which flooding or precipitation was mentioned only in context for study

	Self-reported indicators of flooding exposure (e.g., frequency of contact with floodwater, household flooding)	setting or data collection and not analysed as a risk factor
Human Vulnerability*	- Studies quantifying associations between vulnerability indicators and EPZ outcomes - Vulnerability indicators included, but were not limited to: - Sociodemographic factors (e.g., age, gender, race, disability, income, occupation, religion) - Services-related factors (e.g., WASH services, waste disposal, public transport) - Physical, built or living environment characteristics (e.g., type of household construction material, number of pets, proximity to sewage or drainage systems) - Policy and governance factors (e.g., governance, health policies, infrastructure investments) - Studies were not required to explicitly use the terms <i>vulnerability</i> or <i>vulnerable</i>, provided included variables aligned with the review's operational definition of vulnerability	- Studies not involving human populations - Studies describing geographical areas as vulnerable without assessing vulnerability characteristics of human populations - Studies in which vulnerability indicators were included solely as adjustment variables, and no results were reported for those indicators
Human infection from EPZ	Studies reporting human EPZ outcomes, including but not limited to: incidence,	Laboratory or experimental studies unrelated to population-level human health outcomes

	prevalence, seroprevalence, morbidity, mortality, or disease severity. - Human EPZ outcomes must be related to target EPZ: - Campylobacteriosis - Cryptosporidiosis - E coli - Giardiasis - Leptospirosis - Melioidosis - Salmonellosis - Toxoplasmosis	- Studies focused exclusively on animal, agricultural, or wildlife populations - Studies reporting effect estimates for any disease outside of 8 target EPZ - Studies which treated EPZ as exposures or risk factors rather than outcomes - Studies reporting non-specific diarrhoeal disease outcomes where the target EPZ was not reported separately
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Information sources and search strategy

The search strategy was developed around three core concepts: i) flooding or participation, ii) human vulnerability, and iii) environmental persistent zoonoses. The literature search was conducted in May 2025 and updated in January 2026 across 11 bibliographic databases: Ovid MEDLINE, Scopus, Web of Science, SciELO, Global Index Medicus (inclusive of LILACS, WPRIM, IMSEAR, IMEMR, and AIM), Ovid Global Health, and African-Wide Information. Search terms were developed and piloted in Ovid MEDLINE before being adapted for the remaining databases. Search strategies can be found in S1 Table. No manual search from reference lists was conducted.

Study records

Data management

Rayyan was used for de-duplication, reference management, and screening (40).

Selection

Studies were subjected to a three-stage screening process across titles, abstracts, and full texts based on the eligibility criteria. Abstract and full-text screening was performed in duplicate. Reasons for exclusions were documented and disagreements were resolved through discussion. Records from the updated search were imported and screened using the same process. Supplementary texts for included studies were sought for data extraction when relevant results were provided as supplementary documents.

Articles published in Portuguese, Spanish, and French were translated using DeepL (41). Extracted data from Portuguese-language studies ($n = 2$) were subsequently checked against the original articles by a bilingual researcher fluent in Portuguese and English to verify translation accuracy. No discrepancies were identified.

Data extraction

A data extraction form was developed in Microsoft Excel by one reviewer and checked by two reviewers. The data extraction form was piloted on 10 studies and refined before use on the remaining studies.

Study-level data included publication year, EPZ, country of study, study design, and conceptualisation of vulnerability (including whether the terms “vulnerability” or “vulnerable” were explicitly used and, if so, in what context), and approaches used to consider vulnerability and hazard together. This included whether vulnerability and hazard indicators co-occurred within studies, whether they were conceptually distinguished through frameworks, directed acyclic graphs (DAGs) or presentation of results, and whether this distinction was carried into the analysis through stratification or interaction terms.

Indicator-level data included indicator name, associated EPZ, and health outcome category. Health outcomes were categorised as one from the following: (i) population exposure/infection, defined as infection or exposure identified in a population not selected based on symptoms (e.g., community serosurveys or household sampling); (ii) clinical case, defined as infection identified among individuals who sought care or were ascertained through clinical surveillance for illness (e.g., hospital-based cohorts or notified case series), where severity or morbidity was not the health outcome; or (iii) severe clinical outcome, defined as complications or severe manifestations among clinical cases (e.g., ICU admission, organ failure, or mortality).

Notably, sex and gender were treated as a single indicator. We use "sex" to refer to biological attributes and "gender" to socially constructed roles and identities, but included studies rarely specified which their measure captured, most commonly reporting a binary male/female variable without stating whether it reflected sex assigned at birth or gender identity. These measures were therefore coded as a combined "sex/gender" indicator, and that term is used throughout.

Data analysis and synthesis

Data analysis was conducted in Microsoft Excel and R version 4.5.0 using dplyr, gtsummary, and ggplot2 packages (42-45).

Study selection was reported using a PRISMA 2020 flow diagram (37, 46). Publication trends were summarised by year of publication and EPZ. Study characteristics were summarised by study design, country of study, use of the terms vulnerability or vulnerable, and health outcome category. Studies explicitly using the terms vulnerability or vulnerable were subjected to inductive thematic analysis (47), whereby

themes were developed through discussion between reviewers as patterns emerged from the extracted data. Themes were summarised narratively.

Vulnerability and hazard indicators were categorised into domains using an inductive coding approach (47), whereby domains were developed through discussion between reviewers as patterns emerged from the extracted data. Domains were quantitatively summarised. Vulnerability indicator domains were further summarised by EPZ and health outcome category.

Co-occurrence of vulnerability and hazard indicator domains within studies was summarised quantitatively (42). The presentation and analytical approaches used to distinguish vulnerability and hazard indicators were summarised descriptively.

Critical appraisal

Consistent with scoping review methodology, a critical appraisal was not conducted as the aim was to map the extent and nature of the evidence rather than evaluate study quality.

Results

Selection of sources of evidence

In total, 13,073 records were retrieved across 11 databases (Fig 1). Following deduplication, 5,586 records remained for screening. During screening, 3,782 and 1,555 records were excluded during title and abstract screening, respectively. A total of 249 reports were sought for retrieval, of which 14 could not be obtained. These reports were not accessible through institutional subscriptions and corresponding authors did not respond when contacted. After full text screening, during which 157 studies were excluded, 78 studies were included in the review.

Fig 1. PRISMA-ScR flow diagram of study selection. *Excluded as despite using indicators of vulnerability and hazards relevant to EPZs they did not incorporate vulnerability within models of risk or present statistical comparisons or effect estimates. Therefore, these studies were not eligible for indicator extraction and were excluded. †One preprint report was excluded because the peer-reviewed version of the same study was identified in the updated search and included in the review. Source: Page MJ, et al. *BMJ* 2021;372:n71. doi: 10.1136/bmj.n71

Publication trends and study characteristics

The publication of relevant studies has increased in recent years, with over half of included studies published between 2018 – 2025 (Fig 2). The most notable increase in publications was of leptospirosis studies, which peaked at 6 publications in 2025.

Fig 2. Number of included studies by publication year and environmentally persistent zoonosis. Bars are coloured according to the EPZ investigated; studies examining more than one EPZ are grouped as “>1 EPZ”. Of these multi-EPZ studies, four investigated both cryptosporidiosis and giardiasis, and two investigated three or more enteric EPZ, including *E. coli*.

Despite the increasing frequency of publications, the evidence base remains narrow across EPZs (Table 4). Over half of all studies were based on leptospirosis, whereas *E. coli* was included in only two studies; we did not identify any studies on toxoplasmosis that met our inclusion criteria. Study designs were largely limited to cross sectional (n = 28), case control (n = 8) or aggregated ecological studies (n = 18), with a small number of time series (n = 6), and cohort studies (n = 4). Most health outcomes were based on clinical case (n = 58), defined as suspected or confirmed cases among individuals who sought healthcare. See S2 Table for a summary of all included studies.

251 **Table 4. Characteristics of the 78 included studies.** *Counts are not mutually exclusive; studies may*
252 *contribute to more than one EPZ category and health outcome category.*

Characteristic	Number of studies
EPZ	
Leptospirosis	41
Cryptosporidiosis	11
Giardiasis	11
Melioidosis	8
Salmonellosis	8
Campylobacteriosis	7
E.coli	2
Country	
Brazil	14
Australia	5
Indonesia	4
Argentina	3
Malaysia	3
Mexico	3
Canada	2
China	2
Colombia	2
Croatia	2
Ethiopia	2
Fiji	2
India	2
Nicaragua	2
Thailand	2
Other ¹	28
Study design	
Cross sectional	28
Ecological	18

Case series	11
Case control	8
Time series	6
Cohort	4
Other ²	2
Randomized controlled trial	1
Were the terms “vulnerable” or “vulnerability” used?	
No	62
Yes	16
Health outcome	
Clinical case	58
Population exposure/infection	16
Severity/sequelae	13

¹ Includes countries represented by one study only

² Includes spatial cluster/time-series analysis and ecological niche modelling studies with a cross-sectional demographic component

Of the 78 included studies, only 16 explicitly used the terms "vulnerable" or "vulnerability". Among these, usage was heterogeneous and rarely defined. The term was applied across at least four overlapping themes: a property of place, where social conditions and hazard exposure were spatially co-located (15, 48, 49); socioeconomic disadvantage, describing poverty, poor living conditions, or marginalisation (50-53); biological or demographic susceptibility, particularly among age-defined groups such as children or the elderly (54-57); and a general synonym for elevated disease risk without further description (58, 59). One study used vulnerability as a keyword only, without further reference in the text (60). This heterogeneity across themes illustrates the absence of shared terminology or definitions of vulnerability across EPZ research.

Vulnerability indicators

Six vulnerability domains emerged from 444 vulnerability indicators extracted across studies (Table 5). Sociodemographic indicators (n = 215) included sex/gender, age, and socioeconomic position or poverty. Biological susceptibility (n = 75) included chronic conditions, and nutritional status. Housing (n = 21) included household building material and housing type. Local environment (n = 65) included urbanicity, built environment, and infrastructure quality. WASH services (n = 63) included solid waste service provision and water supply. Finally, health and social services (n = 5) included healthcare access, coverage and type of healthcare facility.

Table 5. Vulnerability domains and indicators used across included studies.

Domain	Indicators (number of studies)
Sociodemographic (n = 215)	Sex/gender (n = 60)
	Age (n = 57)
	Socioeconomic position and poverty (n = 22)
	Education & literacy (n = 21)
	Household occupancy & crowding (n = 14)
	Race, ethnicity & ancestry (n = 13)
	Parental/caregiver characteristic (n = 12)
	Childcare & family composition (n = 10)
	Employment status (n = 2)
	Population movement & mobility (n = 2)
	Marital status (n = 1)
	Social vulnerability (n = 1)
Biological susceptibility (n = 75)	Chronic conditions & comorbidities (n = 43)
	Coinfection (n = 16)
	Nutritional status (n = 10)
	Skin injury (n = 6)

Domain	Indicators (number of studies)
Local environment (n = 65)	Settlement context & density (n = 44)
	Water, drainage & sanitation infrastructure (n = 9)
	Solid waste & environmental cleanliness (n = 7)
	Built environment & infrastructure quality (n = 5)
Housing (n = 21)	Housing structure & materials (n = 14)
	Household type (n = 4)
	Household space (n = 3)
WASH services (n = 63)	Water supply & drinking water (n = 30)
	Sanitation (disposal & toilets) (n = 20)
	Solid waste service (n = 11)
	Electricity or gas supply (n = 2)
H&S services ¹ (n = 5)	Healthcare access & coverage (n = 4)
	Type of healthcare facility (n = 1)

¹ H&S services: health and social services

Across EPZs, indicators from the sociodemographic domain were used most frequently measured, accounting for 41 – 55% of total vulnerability indicators within each EPZ (Fig 3). Use of other domains varied by EPZ, although housing and health and social service indicators were consistently the least frequently measured.

The focus on the sociodemographic domain was consistent across health outcome types (S3 Table). Sociodemographic domain indicators accounted for 53% of all indicators measured within population exposure/infection-outcome models, 48% in clinical case-outcome studies, and 43% in severity/sequelae case-outcome models. Use of other domains varied significantly by health outcome, with comorbidity and biological susceptibility domain indicators accounting for 45% of indicators within

severity/sequelae-case outcome models, but only 13% or 6% of clinical case-outcome or population exposure/infection-outcome models.

Hazard indicators

Four hazard domains emerged from 138 hazard indicators extracted across studies (Table 6). Hazards were commonly represented by meteorological and broad seasonal proxy indicators rather than direct measures of flooding events (Table 6). Meteorological (n = 52) and seasonal indicators (n = 38) together accounted for almost two-thirds of all hazard indicators. Meteorological indicators most commonly captured precipitation over a specified period (n = 27), including monthly, weekly, cumulative, and average daily measures, while a further 15 indicators measured long-term precipitation averages. Seasonal indicators were predominantly binary wet/dry season classifications (n = 28).

Table 6. Hazard domains and indicators used across included studies

Domain	Indicators (number of studies)
Meteorological conditions (n = 52)	Precipitation amount over specified period (n = 27)
	Long-term averages (n = 15)
	Threshold-based precipitation measures (n = 4)
	Derived precipitation indices (n = 3)
	Precipitation frequency (n = 2)
	Precipitation intensity/extreme (n = 1)
Seasonal patterns (n = 38)	Binary wet/dry season (n = 28)
	Multi rainfall seasons (n = 5)
	Calendar seasons (n = 3)
	Binary monsoon/non-monsoon season (n = 2)

Domain	Indicators (number of studies)
Flood susceptibility & landscape (n = 27)	Proximity to water bodies (n = 10)
	Topography & elevation (n = 7)
	Flood-prone areas & flood zones (n = 5)
	Drainage & flow pathways (n = 3)
	Direct flood exposure/flood-prone areas (n = 2)
Flood events & hydrological hazards (n = 21)	Individual contact with flood water (n = 9)
	Community & area flood events (n = 8)
	Household flooding events (n = 4)

¹ Precipitation is used as an overarching term for measures reported in included studies as either rainfall or precipitation.

How vulnerability and hazard were studied together

We first examined which vulnerability and hazard domain were represented within the same studies (Fig 4). Across all EPZs, sociodemographic vulnerability indicators were most frequently paired with meteorological condition (n = 39) and seasonal patterns (n = 29) (Fig 4A). Pairing patterns varied between diseases, with leptospirosis accounting for most of the observed diversity (Fig 4B). Flood event and flood susceptibility domains were rarely represented in studies of EPZs other than leptospirosis, with leptospirosis studies pairing these hazard domains with a wide range of vulnerability domains.

Fig 3. Co-occurrence of vulnerability and hazard domains across included studies. Heat maps show the number of distinct studies in which a given vulnerability domain (y-axis) and hazard domain (x-axis) were both included. (A) Counts pooled across all included EPZs. (B) Counts stratified by EPZ. Cell shading and numeric labels indicate the number of studies reporting each vulnerability–hazard domain pairing. H&S services: health and social services

Hazards and vulnerabilities were generally treated as separate determinants of risk, with little explicit conceptualisation of how they might act together. Only three studies used conceptual frameworks or directed acyclic graphs (DAGs) that explicitly represented hazards and vulnerabilities as distinct but related determinants of disease risk (15, 61, 62). However, none translated this conceptual framing into statistical analyses assessing effect modification between hazard and vulnerability indicators.

Six studies examined vulnerability and hazard together using stratified analyses, providing separate estimates of association across hazard or vulnerability groups (48, 50, 63-66). For example, Azevedo et al., fitted separate regression models for flood-related and non-flood-related leptospirosis cases across vulnerability indicators, but did not formally test whether flooding modified the association between vulnerability and disease risk (50). The remaining studies similarly estimated associations separately within strata, without formally testing for effect modification.

A further four studies formally tested for effect modification using statistical interaction terms. Arsenault et al. reported that the interaction between seasonal period and campylobacteriosis incidence differed by age group (67), while Britton et al. found that the association between rainfall and cryptosporidiosis risk differed according to water supply quality (59). Lau et al. and Obels et al. tested whether associations between rainfall and leptospirosis varied by age or sex, respectively, but found no statistically significant evidence of effect modification (68, 69). Overall, few studies explicitly conceptualised or analysed how vulnerability might modify the effects of flooding-related hazards on EPZ risk.

Discussion

This scoping review identified substantial gaps in how vulnerability and flooding-related hazards are conceptualised, measured and analysed in research on flood-sensitive EPZs. More than half of the included studies were published since 2018, indicating growing attention to this area, yet the evidence was heavily concentrated on leptospirosis and remained sparse for other flood-sensitive EPZs. Vulnerability was inconsistently conceptualised and measured predominantly through a narrow range of sociodemographic indicators, while hazards were commonly represented using broad meteorological or seasonal proxies. The frequent use of cross-sectional, ecological and case-control designs also limited the extent to which temporally varying hazards could be examined alongside detailed, multilevel vulnerability. Although all included studies assessed both vulnerability and flooding-related hazard indicators, few explicitly conceptualised or analysed how the two acted together. These findings suggest that much of the existing research is not yet designed to fully characterise vulnerability to EPZs or explain why flooding-related hazards generate unequal health risks across populations.

Vulnerability is a complex and multidimensional concept, and its inconsistent use across the included literature reflects broader variation in how vulnerability has been defined within climate and health research (70). Definitions have evolved across IPCC Assessment Reports, with reports that researchers have been slow to adopt updated definitions (24). Adoption of a shared definition, such as that used in the IPCC Sixth Assessment Report (AR6), could encourage a more holistic consideration of vulnerability across multiple domains and improve comparability and accumulation of evidence across EPZ research and maintain relevance for climate adaptation (20).

Vulnerability was most often characterised through individual-level sociodemographic indicators, reflecting a relatively narrow conceptualisation that overlooks important drivers of EPZ transmission. The predominance of sex/gender and age as vulnerability indicators is consistent with findings from wider vulnerability and infectious disease research reviews (34-36) and reflects their routine inclusion in observational studies (36), and relevance to exposure pathways and biological mechanisms of disease (71, 72). However, this emphasis on individual-level characteristics risks framing vulnerability primarily in biological or demographic terms while overlooking the structural social, environmental, infrastructural, and service-level processes that generate unequal health risks among individuals (20, 21, 23, 73-76). In the context of EPZs, this is particularly limiting. Broader vulnerability domains, such as housing quality, WASH services, waste management, and the condition of the local built and natural environment all shape where pathogens are shed and persist and the opportunities for people to encounter contaminated soil or water (77-79). These domains are particularly important for understanding risk of EPZs as many disproportionately affect populations experiencing poverty and inadequate services (1-6, 30, 80). The six domains identified in this review provide a practical organising structure for considering these broader domains when studying vulnerability to EPZs. Greater consistency in the definition and reporting of indicators within these domains, while retaining context-specific measures where necessary, would improve comparability and support future evidence synthesis.

Flooding-related hazards were commonly represented by broad seasonal or meteorological proxies, which can make it difficult to determine the specific contribution of flooding to EPZ risk. This is likely to reflect the greater availability and routine collection of these data compared with direct measures of flooding. Rainfall

measures can still be informative for capturing flood-generating conditions, such as intensity, frequency, duration, antecedent wet or dry conditions or thresholds for extreme rainfall (81-83). However, more direct measures such as flood susceptibility and occurrence can characterise where and when flooding is likely or observed, while individual or household contact with floodwater is closer to direct human exposure. Two studies included in this review incorporating meteorological and flood susceptibility or event indicators suggest that this distinction can matter: Azevedo et al. reported stronger associations between leptospirosis and flood susceptibility than with rainfall frequency, while Soni et al., found household flooding, but not heavy rainfall, was associated with increased leptospirosis risk (50, 84). Future studies should therefore select hazard indicators according to the part of the flooding pathway relevant to the research question and, where feasible, combine meteorological data with flood susceptibility, hydrological, or direct exposure measures to distinguish broader rainfall-related environmental effects from those associated with flooding itself or with direct contact with floodwater. Flood susceptibility may be particularly useful for characterising longer-term flood risk where study designs or available data do not permit measurement of individual flood events.

One of the clearest gaps identified by this review was the limited extent to which studies were explicitly designed to understand how vulnerability changes the health effects of flooding-related hazards. Few studies used conceptual frameworks or DAGs to structure these relationships; six examined vulnerability and hazard together using stratified analyses, and a further four formally tested effect modification using interaction terms. Treating vulnerability and hazard as separate determinants can identify populations with high underlying disease risk but does not establish whose risk increases most when a climate hazard occurs or why. Future research should

therefore begin with an explicit conceptual model and research question about how vulnerability is expected to shape hazard-related risk. This can then guide the choice of study design, vulnerability and hazard indicators, and analytical approach.

The study designs used across the literature also constrained the extent to which these questions could be addressed. Most studies were cross-sectional, ecological or case-control, with relatively few time-series or cohort studies. Cross-sectional and case-control studies can capture vulnerability in greater detail but often rely on retrospective or relatively coarse measures of hazard exposure, while time-series studies can characterise temporal variation in hazards but usually contain less information on within-population vulnerability. Cohort studies are better suited to linking time-varying hazards with detailed individual- and household-level vulnerability information over time, but their cost and logistical demands may be prohibitive for many EPZs, many of which are neglected diseases disproportionately affecting marginalised populations and often studied in resource-constrained settings (78, 85). Where cohort studies are not feasible, other observational designs could make greater use of linked meteorological, hydrological and spatial data, including flood susceptibility when event-level flooding cannot be measured. Small-area time-series analyses using fine geographic units, such as census tracts, may also allow time-varying hazards to be linked with area-level measures of social and environmental vulnerability (86). Interdisciplinary approaches bringing epidemiological and social expertise together with climate, hydrological and environmental science could help make better use of these complementary data sources (87).

These gaps may reflect a broader challenge in infectious-disease research rather than one unique to EPZs, with research on vulnerability to climate-sensitive infectious

diseases still developing compared with some other areas of climate and health (83, 88). A 2016 review of meteorological exposures and diarrhoeal disease similarly called for research to move beyond simple climate hazard–disease associations and examine how demographic and WASH factors modify climate-related risks (83). Heat-health research provides a more established example of examining how climate-related health effects vary across populations and translating this evidence into adaptation. Studies have identified elevated health risks among older people, pregnant women and other groups, informing targeted adaptation measures such as modification of work schedules and strengthened community support during heatwaves (89-91). Although the evidence for flooding is less developed, a multi-country study of 761 cities found stronger flood-related mortality effects in low-income countries, communities with lower human development, and populations with a greater proportion of older people (92). Vector-borne disease studies have similarly shown modification of flood-related risks by socioeconomic and infrastructural factors, such as displacement and preventative resource access for malaria (93) and urbanisation and water-supply shortages for dengue (94). Such evidence can identify whose risk is amplified by climate hazards and provide a stronger basis for targeted preparedness, disease-control measures and equitable adaptation. Our findings suggest that this shift towards explicitly examining how vulnerability shapes hazard-related risk has yet to occur widely in EPZ research.

This review has limitations. Non-English studies (n = 6) were translated for screening and data extraction, which broadened inclusion but may have introduced some inaccuracies in interpretation. The search string itself was not translated, so some non-English literature may have been omitted. Grey literature was not searched. No formal critical appraisal was undertaken, consistent with the mapping purpose of a scoping

review. Sex and gender were combined into a single indicator because studies seldom specified whether a binary male/female variable reflected biological sex or gender. This conflation limits interpretation, since the two constructs imply different pathways to vulnerability with biological susceptibility on the one hand, and socially patterned exposure and care responsibilities on the other, which have different implications for intervention. Classification of some flooding-related indicators was also challenging, particularly measures such as proximity to rivers or drainage channels, flood-prone areas, and topography, which capture susceptibility to flooding rather than flood occurrence itself. These indicators were retained within the hazard domains in accordance with the review protocol but illustrate the practical difficulties of separating components of climate-health risk frameworks.

In conclusion, this review highlights important gaps in how vulnerability to flood-sensitive EPZs is conceptualised, measured and studied alongside flooding-related hazards. Vulnerability was often narrowly and inconsistently characterized, hazard measures relied on broad meteorological or seasonal proxies, and few studies were explicitly designed to examine how vulnerability shapes hazard-related risk. Future research should use clearer conceptual frameworks, measure vulnerability across relevant domains and multiple levels, select hazard indicators that more accurately measure the flooding process and adopt study designs and analytical approaches capable of examining their interaction directly. Strengthening this evidence base will help identify which populations are more at-risk during flooding, explain why risks are unequal, and support more targeted disease control and equitable climate adaptation.

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Supporting information

S1 Table. Search strategies. Full search strategies for all 11 electronic databases, including platform, date range searched, and number of records retrieved. Searches were run in May 2025 and updated in January 2026. Search strings were developed in MEDLINE and adapted for each database's syntax. No language or date limits were applied.

S2 Table. Data charted from included studies. Data items comprised bibliographic information (authors, year, title, journal), study design, country of data collection, study aim, EPZ(s) studied, whether the terms "vulnerability" or "vulnerable" were used in the study, and any evidence of interaction between vulnerability factors and hazards.

S3 Table. Vulnerability indicators by domain, EPZ and model outcome.

Separate sub-tables are presented for each EPZ. Values are n (%), where n represents the number of times a vulnerability indicator was included in a model assessing the given outcome. Within each health outcome column, percentages are calculated from the total number of indicator occurrences recorded for that outcome only. Percentages in the Overall column are calculated from the total number of indicator occurrences across all outcomes for that EPZ. Percentages therefore sum down columns.

796

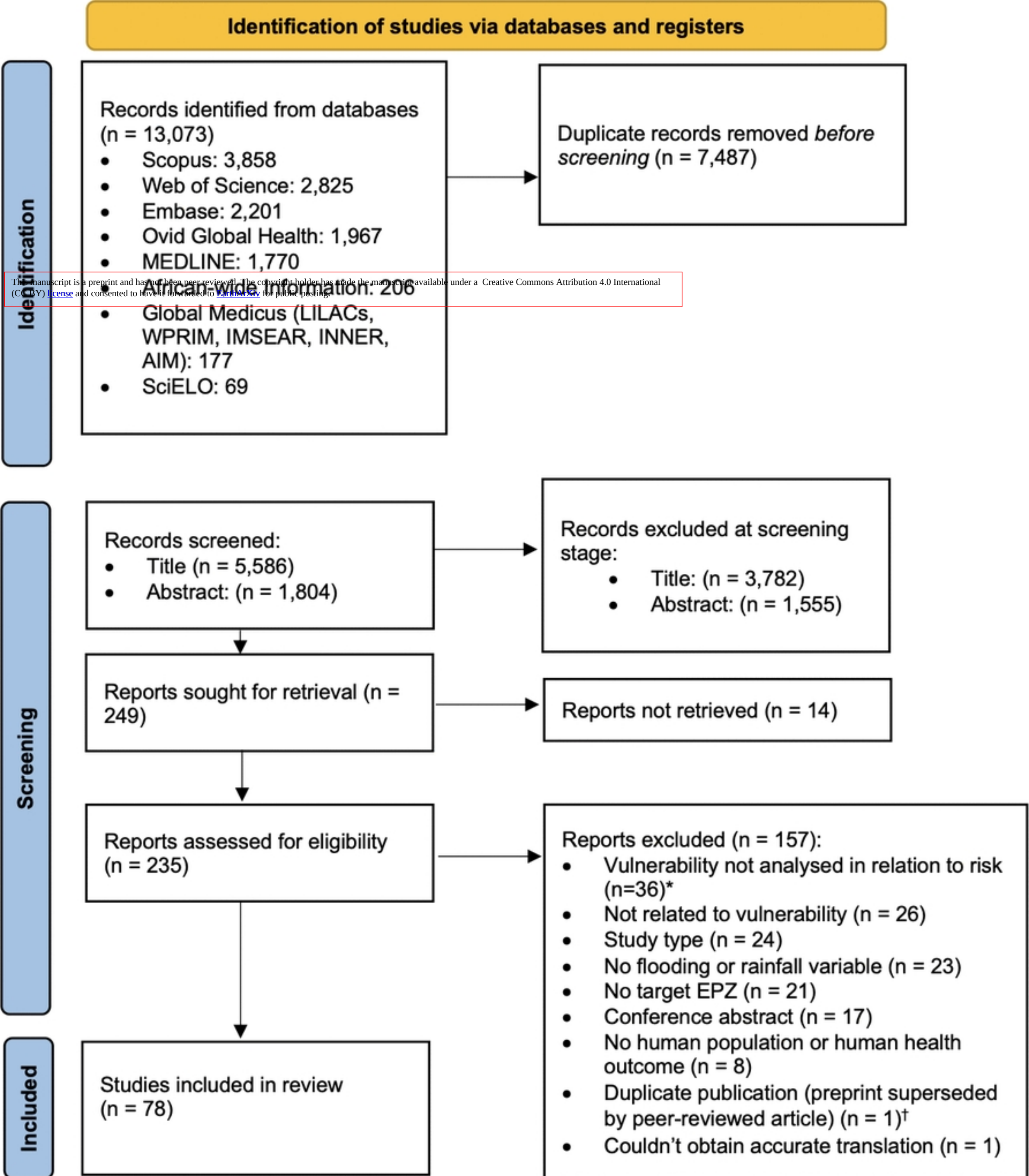


Fig1

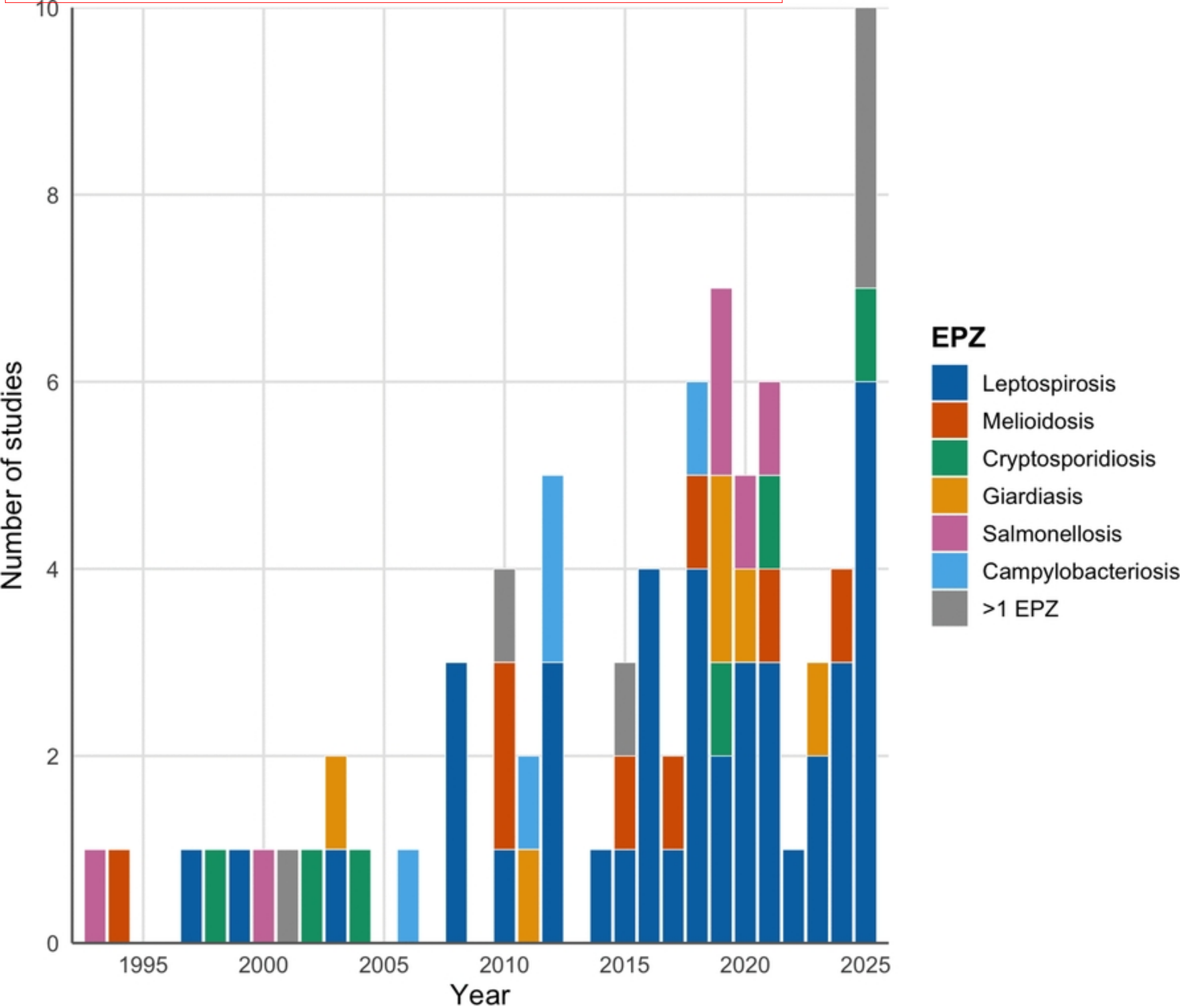


Fig2

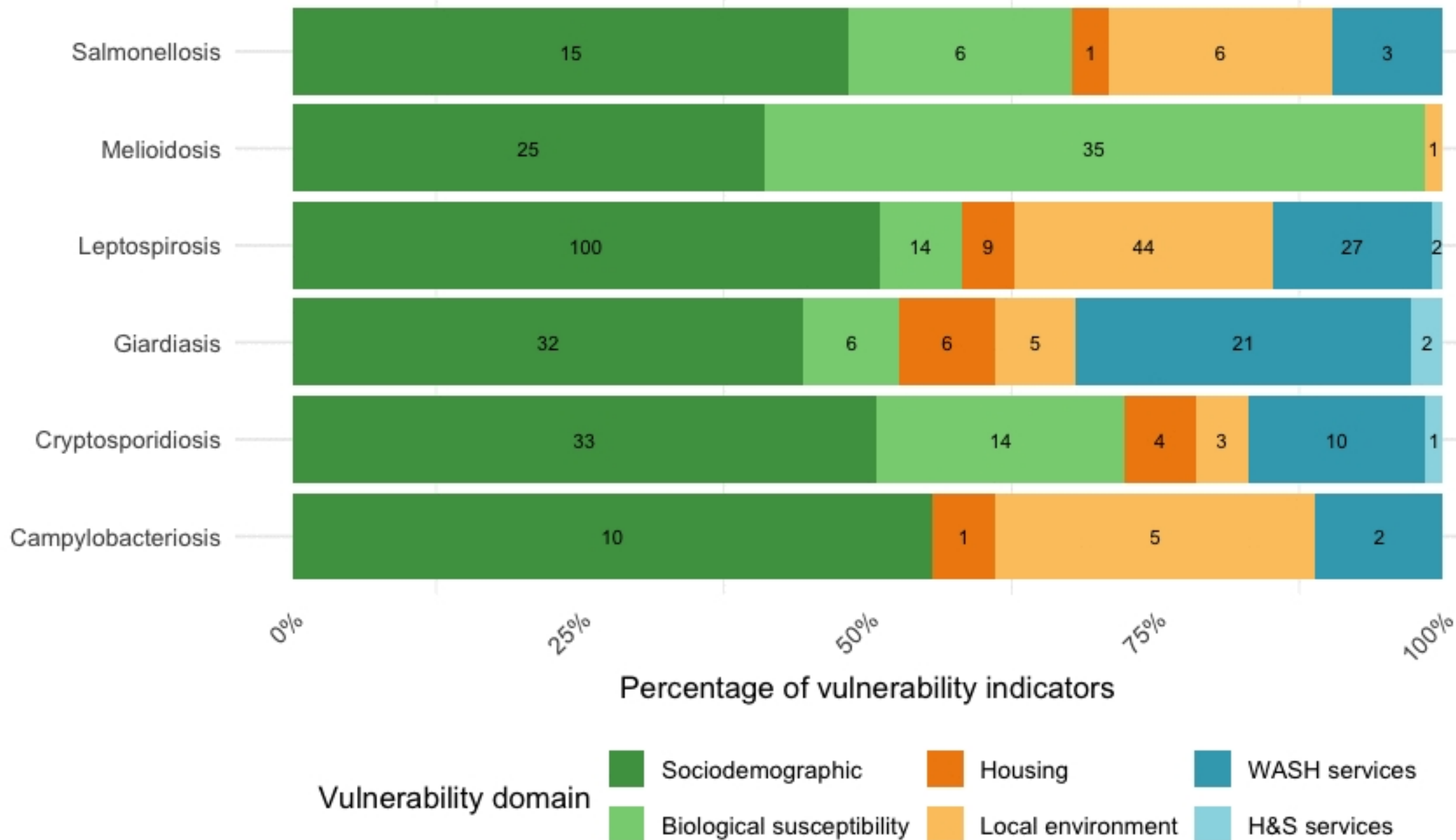


Fig3

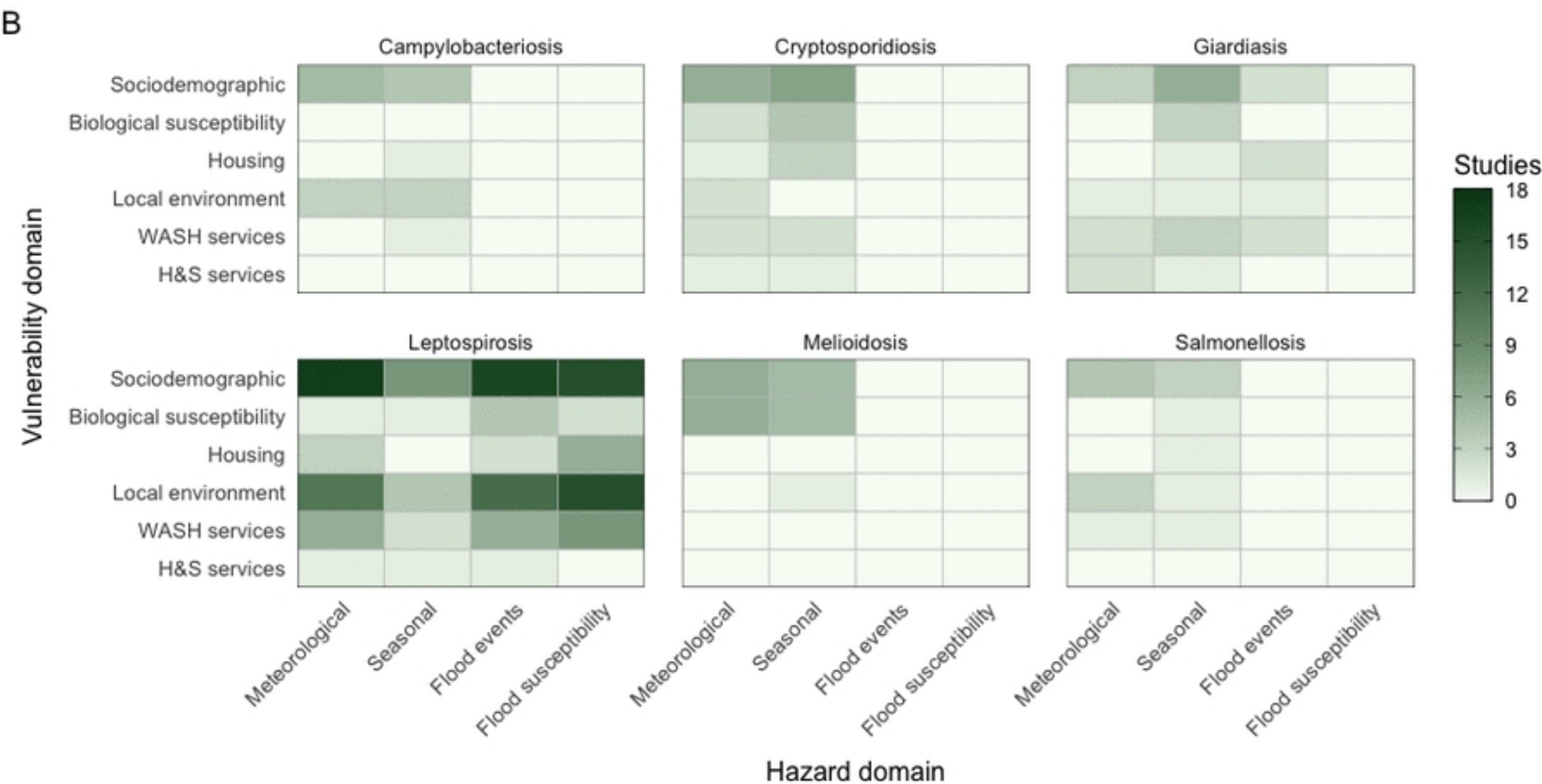
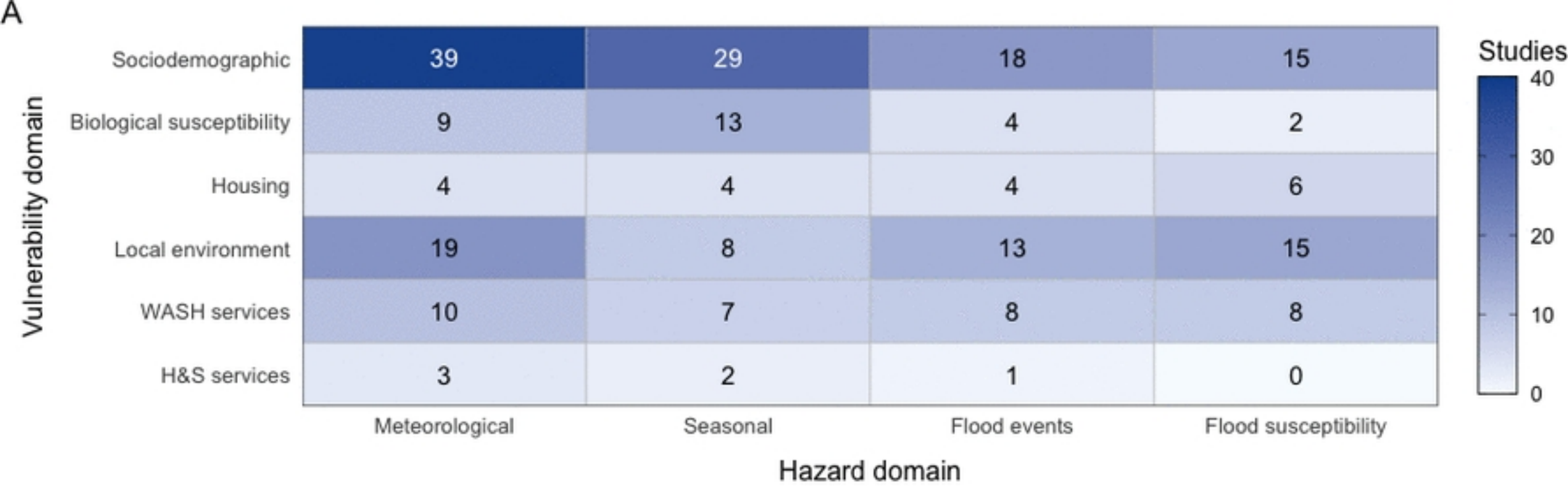


Fig4