



Emerging Vector-Borne Pathogens at the Wildlife–Human Interface: Ecological Drivers, Surveillance Gaps, and Public Health Implications

Moshood Ayinde^{1*}, Kazeem Abdulrazaq², Habeeb Damilola Yusuf³

¹Department of Biological Sciences, Western Illinois University, Macomb, Illinois, USA

²Department of Public Health Sciences, New Mexico State University, Las Cruces, New Mexico, USA

³University of Ilorin Teaching Hospital, Ilorin, Nigeria

*Corresponding Author: Moshood Ayinde, moshodayinde35@gmail.com

Abstract

Vector-borne pathogens occupy a structurally awkward position in global health. They are transmitted by arthropods whose populations are governed by climate, hydrology and land cover; they are maintained in vertebrate reservoirs that belong to no ministry's mandate; and they reach human beings through contact processes that sit outside the clinical encounter where surveillance systems are designed to detect disease. This review synthesises the evidence on emergence of vector-borne pathogens at the wildlife–human interface, organised around three questions: what ecological processes generate emergence, where surveillance systems fail to see it, and what the resulting public health exposure amounts to. The evidence supports four propositions. First, emergence is driven less by pathogen novelty than by the reorganisation of host and vector communities under land-use change, with zoonotic reservoir hosts systematically over-represented in human-modified landscapes and spillover risk maximised at intermediate levels of land conversion rather than in intact or fully converted habitat. Second, climate change is redistributing rather than simply expanding transmission, with vector range shifts measurable in observed occurrence records and transmission suitability governed by unimodal thermal relationships that can reduce as well as increase risk. Third, transmission in multi-host systems is dominated by heterogeneity: a small number of highly competent and preferentially fed-upon reservoir species carry most amplification, which makes mean host density a poor predictor of risk and makes the biodiversity–disease relationship mechanism-specific rather than general. Fourth, the surveillance architecture that should detect all of this has eroded. Entomological capacity in the United States declined substantially in the decade after West Nile virus became endemic; fewer than half of surveyed national veterinary authorities operate wildlife disease surveillance; and in much of sub-Saharan Africa and Asia, arboviral and bacterial zoonotic febrile illness is presumptively misclassified as malaria and never enters any surveillance stream. The public health implication is that the vector-borne disease



burden recorded in official data is a function of detection capacity as much as of transmission, and that primary prevention directed at ecological drivers is a necessary complement to detection-and-response preparedness rather than an alternative to it.

Keywords: vector-borne disease; zoonotic spillover; wildlife–human interface; One Health; entomological surveillance; reservoir competence; land-use change; arbovirus

1. Introduction

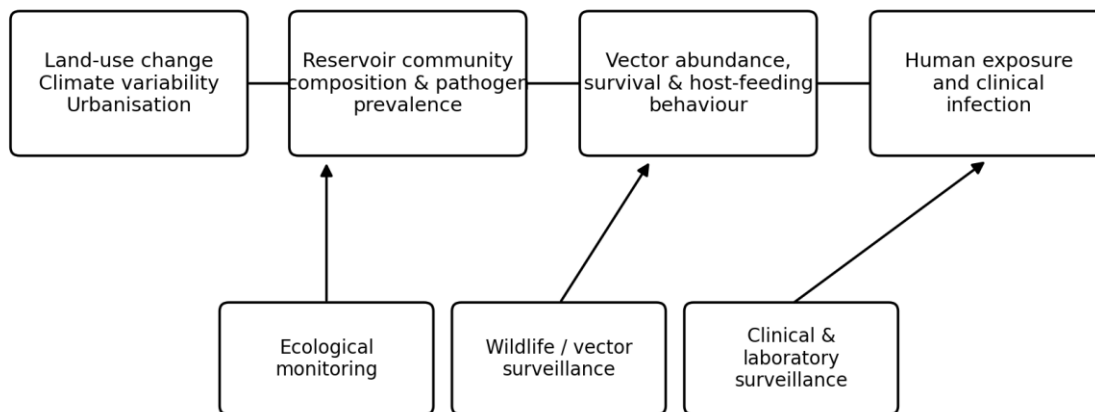
Vector-borne diseases account for more than 17% of all infectious diseases and cause over 700,000 deaths each year (World Health Organization [WHO], 2020). Malaria alone accounted for an estimated 247 million cases and 619,000 deaths in 2021 across 84 endemic countries (WHO, 2022), while dengue — the fastest-expanding arbovirus in recorded history — was estimated at 390 million infections annually, of which some 96 million are clinically apparent, with 3.9 billion people living in areas of transmission risk (Bhatt et al., 2013). Global Burden of Disease analysis confirms that incident dengue cases rose steeply between 1990 and 2017, with the greatest burden in South and Southeast Asia (Zeng et al., 2021), and mapping work projects that by 2080 more than six billion people — some 60% of the world’s population — will live in areas suitable for dengue transmission (Messina et al., 2019). The dominant driver of this redistribution is not any single factor but the interaction of climate, land use, urbanisation and human mobility (Franklin et al., 2019). These aggregate figures, however, obscure the process that generates them. The great majority of the vector-borne disease burden is carried by a small number of long-established pathogens in well-characterised transmission systems. The epidemiologically consequential events of the past three decades have instead been emergences: West Nile virus establishing across a continent in three years, chikungunya crossing the Indian Ocean, Zika producing a congenital syndrome no one had anticipated, Japanese encephalitis virus genotype IV appearing in temperate Australia, and Crimean-Congo haemorrhagic fever virus becoming autochthonous in western Europe.

These events share a structural feature. Each involved a pathogen maintained in non-human vertebrates and transmitted by arthropods, crossing into human populations along a pathway that no single surveillance system was designed to observe. Of 335 emerging infectious disease events recorded between 1940 and 2004, 60.3% were zoonoses, 71.8% of those originated in wildlife, and vector-borne diseases accounted for 22.8% of all events, with a distinct peak in the 1980s associated with climatic anomalies (Jones et al., 2008). Host-trait analysis across 754 mammal species and 586 viruses established that the proportion of zoonotic viruses per host species is approximately constant at 0.14 across mammalian orders, so that total viral richness rather than taxonomic identity predicts zoonotic potential (Olival et al., 2017). The implication is uncomfortable: the reservoir of potential emergences is not a discrete list of candidate pathogens but a continuous function of how much wildlife there is, how much contact humans have with it, and which arthropods bridge the two.

This review synthesises the ecological, epidemiological and surveillance literature on that

process. It is organised in three parts, corresponding to the three claims in its title. Sections 2 and 3 address ecological drivers: the conceptual structure of vector-mediated spillover, and the empirical evidence on land use, biodiversity, urbanisation and climate as drivers of emergence. Section 4 presents case studies, selected to span temperate and tropical systems and to illustrate the range of mechanisms involved. Section 5 addresses surveillance gaps, drawing on capacity assessments, wildlife surveillance surveys and the febrile-illness aetiology literature. Sections 6 and 7 address public health implications, intervention options and a research and policy agenda. The argument throughout is that emergence at the wildlife–human interface is a predictable consequence of measurable ecological processes, that current surveillance is poorly configured to observe those processes, and that the gap between the two is where the public health risk actually sits.

2. The Wildlife–Human Interface: Conceptual Foundations of Vector-Mediated Spillover



Risk emerges where ecological change, host–vector interaction, and surveillance blind spots intersect.

Figure 1. Conceptual pathway linking ecological change, host–vector dynamics, human exposure, and surveillance blind spots.

Plowright et al. (2017) formalised spillover as a hierarchical sequence of barriers that every zoonotic pathogen must pass: the distribution and density of reservoir hosts; pathogen prevalence and shedding intensity within those hosts; pathogen survival and dissemination outside the host; human exposure behaviour; and finally within-host genetic, physiological and immunological compatibility. Because the barriers are multiplicative rather than additive, spillover remains rare even where reservoir infection is common, and an intervention acting on any single layer can interrupt transmission entirely. The framework also explains a persistent empirical frustration: reservoir prevalence is a weak predictor of human incidence, because it addresses only one term in a product.



For vector-borne pathogens the framework requires an additional layer, because the arthropod is simultaneously part of the pathogen's dissemination stage and part of the human exposure stage. The "bridge vector" concept was developed to capture this — a mosquito species that feeds on both the amplifying vertebrate reservoir and on humans, and so carries the pathogen across the host barrier. Kilpatrick et al. (2005) formalised the paradigm for West Nile virus and then showed its limits: where a single species such as *Culex pipiens* acts as both enzootic and bridge vector, risk assessments built on a separation between enzootic and bridge transmission will mislead. The practical consequence is that for many of the most important emerging systems, there is no separable "spillover step" to target; the amplification cycle and the human exposure cycle run through the same insect population.

Two further features distinguish vector-borne spillover from direct zoonotic transmission. The first is that the vector imposes its own set of environmental dependencies. Pathogen replication inside an arthropod is temperature-dependent, arthropod survival is governed by humidity and microclimate, and arthropod abundance is governed by larval habitat availability — so vector-borne spillover is sensitive to climate and hydrology in ways that direct-contact zoonoses are not. The second is that vector feeding behaviour is a behavioural trait subject to seasonal and ecological modulation, which means the human exposure barrier is not fixed. Kilpatrick, Kramer, Jones, Marra and Daszak (2006) demonstrated this directly: the probability that *Cx. pipiens* fed on humans rather than birds rose roughly sevenfold between mid-June and mid-September in the north-eastern United States, tracking the post-breeding dispersal of American robins. Human epidemics in that system are generated not by a change in viral prevalence but by a change in mosquito feeding preference.

This matters for how surveillance should be conceived. If the exposure barrier is dynamic and behaviourally mediated, then entomological surveillance that measures vector abundance without measuring host-feeding patterns will not capture the variable that determines human risk. If the amplification cycle runs through wildlife populations, then human case surveillance is structurally lagging — it observes the system only after the barriers have already been crossed. And if the whole process is governed by land cover, host community composition and climate, then the leading indicators of emergence are ecological variables that health systems do not collect. Sections 3 through 5 take these three problems in turn.

3. Ecological Drivers of Emergence

3.1 Land-use change and the reassembly of host communities

The single most robust finding in the emergence literature is that human land use systematically restructures host communities in favour of reservoir species. Gibb et al. (2020) analysed 6,801 ecological assemblages and 376 host species from the PREDICTS database and found that species known to host human-shared pathogens are over-represented in human-managed landscapes: zoonotic host species richness was 18–72% higher and total zoonotic host abundance 21–144% higher in secondary, agricultural and urban sites than in undisturbed



habitat, with the effect concentrated in rodents, bats and passerine birds. This is not a claim that disturbed landscapes contain more species; it is the stronger and more useful claim that they contain a different and more competent set of species. Land-use monitoring therefore carries surveillance-relevant information about host community composition, independent of any pathogen measurement.

The geography of emergence follows the same logic. Allen et al. (2017) modelled 147 wildlife-origin zoonotic emergence events from 1970 to 2008 while correcting for reporting bias, and identified forested tropical regions undergoing land-use change as the dominant risk landscape, with evergreen broadleaf tree cover, human population density and mammal species richness the top-ranked predictors. Critically, the relationship between land conversion and spillover is non-monotonic. Faust et al. (2018), using a coupled habitat–epidemiological model, showed that spillover risk is maximised at intermediate levels of conversion: complete conversion eliminates the reservoir, while intact habitat minimises human contact. This result reconciles a body of apparently contradictory field observations and carries an awkward policy implication — that partial habitat protection can generate more spillover than either full protection or full conversion.

Empirical work on specific systems is consistent with the model. MacDonald and Mordecai (2019) demonstrated a bidirectional coupling in the Brazilian Amazon, in which forest loss increased malaria incidence while malaria burden in turn suppressed subsequent forest clearing, and Rulli et al. (2017) linked Ebola virus disease outbreak locations in Central and West Africa to recent forest fragmentation within dense-forest areas. Burkett-Cadena and Vittor (2018) showed the mechanism operating at the level of vector communities: forest conversion disproportionately favours mosquito species that are competent vectors of human pathogens, so land conversion changes not just the number of mosquitoes but the identity of the dominant species. Habitat fragmentation increases edge contact and is a general driver of novel pathogen emergence (Wilkinson et al., 2018), and Kilpatrick and Randolph (2012) provide the integrative account for vector-borne zoonoses specifically.

A parallel body of work in land and environmental monitoring supplies the measurement methods this argument depends on. Geostatistical characterisation of agricultural soils in south-western Nigeria, for example, demonstrates how spatially explicit environmental sampling can resolve land-use gradients at the resolution at which host and vector communities actually respond (Kianguebene-Koussingounina et al., 2023), while predictive analytic approaches developed for monitoring urban emissions and infrastructure risk illustrate how continuously collected environmental indicators can be converted into risk signals rather than retrospective descriptions (Okojie et al., 2023). The relevance is methodological: the ecological drivers identified above are measurable with existing environmental monitoring infrastructure, and the obstacle to using them in health surveillance is institutional rather than technical.

3.2 Biodiversity, host community composition and the dilution debate

If land use restructures host communities, the next question is how community structure maps onto transmission. LoGiudice et al. (2003) provided the foundational empirical case in the



Lyme disease system: the white-footed mouse (*Peromyscus leucopus*) infects a large majority of the larval ticks that feed on it, whereas opossums, squirrels and most birds are poor reservoirs and additionally groom off large numbers of larvae, so that adding host species to the community reduces nymphal infection prevalence. Keesing et al. (2006) set out the general mechanisms by which diversity can reduce risk — encounter reduction, transmission reduction, susceptible host regulation — or amplify it, and Ostfeld and Keesing (2012) synthesised the evidence that dilution is most likely where the most competent reservoirs are also the species that persist in degraded, species-poor communities. Civitello et al. (2015) reported broad meta-analytic support for a general dilution effect across a wide range of parasite species.

The counter-literature is substantive and should not be treated as marginal. Randolph and Dobson (2012) argued that “biodiversity buffers disease” is a Panglossian over-generalisation unsupported in tick-borne systems; Wood and Lafferty (2013) showed that for Lyme disease the direction of the biodiversity–risk relationship depends on which axis of diversity is measured and at what spatial scale; and Salkeld et al. (2013) found in meta-analysis that biodiversity–zoonotic-risk relationships are idiosyncratic rather than consistently dilutive. Halsey (2019) proposed the resolution that this review adopts: abandon the binary dilution/amplification framing in favour of mechanism-specific, host-community-explicit models tied to a defined spatial scale and a defined risk endpoint. On that framing, the question is not whether biodiversity reduces disease but which species are gained or lost, how competent they are, and how vectors distribute their bites among them.

West Nile virus illustrates why this reframing matters. Komar et al. (2003) experimentally infected 25 North American bird species and showed that corvids and several passerines — blue jays, common grackles, house finches and American crows — develop viraemias sufficient to infect feeding mosquitoes, with American crow infection almost uniformly fatal. Kilpatrick, Daszak, Jones, Marra and Kramer (2006) then showed that host heterogeneity rather than vector abundance dominates transmission: American robins contributed disproportionately to the basic reproduction number despite being a small fraction of the avian community, while the numerically dominant house sparrow contributed little. Robins supplied roughly half of early-summer *Cx. pipiens* blood meals while comprising under 5% of the bird community. In such a system, the community-level mean of host competence is an almost meaningless quantity, and risk is determined by the abundance and accessibility of a handful of species.

3.3 Urbanisation

Urbanisation restructures transmission through three partly independent channels: container-breeding vector species that exploit artificial larval habitat, urban heat-island effects that accelerate vector and pathogen development, and socio-ecological heterogeneity that concentrates exposure in particular neighbourhoods and housing types (LaDeau et al., 2015). The result is that urban vector-borne disease risk is characteristically fine-grained, with variation across a few hundred metres that regional models cannot represent. It also means that the most important urban vector species — *Aedes aegypti* above all — are commensal rather than wild, so that the wildlife–human interface framing applies to them only historically. Brady



and Hay (2020) trace how the domestication of *Ae. aegypti* enabled dengue to become the first pandemic arbovirus, a trajectory in which the relevant ecological driver was human settlement rather than wildlife contact.

3.4 Climate change and vector range redistribution

Climate change is frequently described as expanding vector-borne disease risk. The evidence supports a more specific claim: it is redistributing risk, with expansions at higher latitudes and altitudes, contractions in some equatorial lowlands, and seasonal lengthening across much of the tropics.

Observed invasion velocities are now measurable directly from occurrence records. Kraemer et al. (2019) estimated that *Ae. aegypti* spread through the United States at approximately 250 km per year and *Ae. albopictus* at approximately 100 km per year in Europe, accelerating to roughly 150 km per year in the most recent five-year window, and projected that 159 and 197 countries respectively would report the two species by 2080. Ryan et al. (2019) projected that under high-emissions scenarios nearly a billion people face novel exposure to *Aedes*-borne virus transmission by the end of the century, with risk rising in Europe and at higher latitudes while some equatorial lowlands become too hot for optimal *Ae. aegypti* transmission. Colón-González et al. (2021), in a multi-model multi-scenario intercomparison, estimated that under the highest emissions and population pathway an additional 4.7 billion people relative to a 1970–1999 baseline would be at risk of malaria and dengue by the late 2070s, with the malaria transmission season lengthening by up to 1.6 months in tropical highlands and the dengue season by up to four months in tropical lowlands. The mechanism is captured by unimodal thermal-performance models. Transmission of dengue, Zika and chikungunya by *Ae. aegypti* peaks near 29 °C and by *Ae. albopictus* near 26°C, with the extrinsic incubation period shortening sharply as temperature rises toward the optimum before declining biting rates and survival reverse the trend (Mordecai et al., 2017, 2019). Because these relationships are humped rather than monotonic, warming increases transmission in currently cool regions and decreases it in currently hot ones — which is why aggregate statements about climate and vector-borne disease are unreliable without specifying the baseline temperature.

Ticks respond through a different set of mechanisms, governed by development and diapause rather than by the short generation times that dominate mosquito dynamics. Jore et al. (2011) documented both latitudinal and altitudinal range shifts of *Ixodes ricinus* at its northern Norwegian limit; Jaenson et al. (2012) showed northward expansion and increased abundance in Sweden over 30 years, linked to milder winters and longer growing seasons; and Medlock et al. (2013) catalogued the multi-factor drivers — climate, deer abundance, habitat structure and land use — operating across Europe. Ogden et al. (2008) produced the risk maps projecting northward expansion of *Ixodes scapularis* into Canada, and Eisen et al. (2016) documented the United States county-scale picture, with *I. scapularis* established in 45.7% of counties against 30.1% in a 1998 survey. Gilbert (2021) cautions that tick-borne disease responses to warming are frequently non-linear and host-mediated, and Randolph's earlier work on tick-borne



encephalitis established that transmission cycles requiring synchronous larval and nymphal co-feeding may be disrupted rather than expanded by warming. Semenza and Suk (2018), Rocklöv and Dubrow (2020) and Caminade et al. (2019) synthesise the policy implications, and Romanello et al. (2022) place vector-borne disease within the broader climate–health indicator framework.

4. Case Studies in Emergence and Re-emergence

4.1 *West Nile virus: a fully documented amplification system*

West Nile virus is the best-characterised emerging vector-borne zoonosis available, and the only one in which the full chain from reservoir competence through vector feeding preference to human epidemic timing has been quantified. Komar et al. (2003) established the competence hierarchy; Kilpatrick, Daszak, et al. (2006) established that heterogeneity in that hierarchy dominates the basic reproduction number; and Kilpatrick, Kramer, et al. (2006) established that seasonal shifts in mosquito host choice drive the timing of human cases. Kilpatrick (2011) synthesised how globalisation and land use enabled the invasion, and Ronca et al. (2021) reviewed two decades of North American experience, arguing that West Nile virus has effectively become a neglected disease in its country of introduction.

The European trajectory has been different in form but not in kind. Young et al. (2021) reported 3,849 human infections and 379 deaths across the European Union and enlargement countries from 2010 to 2018, with the 2018 season alone accounting for 1,993 cases — more than all preceding years combined — and 45 newly affected areas. Usutu virus, which shares *Culex* vectors and avian hosts, has established alongside it: Angeloni et al. (2023) documented 105 autochthonous human infections across eight European countries between 2012 and 2021, of which 85 were asymptomatic detections in blood donors and only 12 presented with neurological disease, with evidence of circulation in birds or other vertebrates reported from 15 EU countries. The Usutu case is instructive precisely because its human burden is small: it was detected largely through blood-donor screening and veterinary investigation rather than clinical surveillance, which is a fair description of how a low-virulence emerging arbovirus becomes visible at all.

4.2 *Tick-borne emergence in North America*

The North American tick-borne disease picture has changed faster than the mosquito-borne picture, and with less public attention. Kugeler et al. (2021), using commercial insurance claims, estimated that approximately 476,000 patients were diagnosed and treated for Lyme disease annually in the United States during 2010–2018 — roughly an order of magnitude above notifiable case counts, a discrepancy that is itself a surveillance finding. Beyond *Borrelia burgdorferi*, the same vector transmits *Babesia microti* (Vannier & Krause, 2012) and *Anaplasma phagocytophilum* (Bakken & Dumler, 2015), producing frequent co-infection, and *Borrelia miyamotoi*, first recognised as a human pathogen in 46 Russian patients with relapsing fever (Platonov et al., 2011) and subsequently found across the *Ixodes* range (Wagemakers et



al., 2015). Powassan virus, the lineage II deer tick virus, is transmitted within roughly 15 minutes of tick attachment and carries a case-fatality rate near 10–15% with neurological sequelae in about half of survivors (Ebel, 2010; Hermance & Thangamani, 2017).

Steere et al. (2016) provide the definitive clinical and microbiological account of Lyme borreliosis against which these co-circulating agents must be distinguished, and the routine difficulty of doing so — overlapping seasonality, overlapping vector, overlapping presentation — is itself a reason why co-infections are systematically under-recognised.

Two genuinely novel pathogens were described from the same region within three years. Heartland virus, a phlebovirus related to severe fever with thrombocytopenia syndrome virus, was identified in two north-western Missouri farmers (McMullan et al., 2012) and subsequently detected in field-collected *Amblyomma americanum* nymphs (Savage et al., 2013); Bourbon virus, a novel thogotovirus, was described from a fatal Kansas case (Kosoy et al., 2015) and later detected in field-collected *A. americanum* in Missouri (Savage et al., 2017). In both cases the pathogen was discovered because a severely ill patient was investigated with agnostic molecular methods, not because surveillance was looking for it.

The invasion of *Haemaphysalis longicornis* demonstrates the vector side of the same problem. Rainey et al. (2018) reported the first established United States population, on a sheep in Hunterdon County, New Jersey. Within roughly a year, Beard et al. (2018) documented detections in 45 counties across nine states, on six domestic species (dogs accounting for 52% of domestic detections), six wildlife species (white-tailed deer accounting for 54% of wildlife detections) and two humans. The species reproduces parthenogenetically, so a single female can found a massive infestation, and in its native range it transmits severe fever with thrombocytopenia syndrome virus and *Rickettsia japonica*. An invasive tick of substantial vectorial potential established across nine states before anyone noticed, which is a reasonably precise measure of passive surveillance sensitivity.

4.3 Emergence in temperate Europe and Australia

Negredo et al. (2017) reported the first autochthonous Crimean-Congo haemorrhagic fever cases in western Europe: an index case acquired from a tick bite in Ávila province, Spain, in 2016, and a nosocomially infected nurse. The finding confirmed establishment of the virus in *Hyalomma lusitanicum* in Iberia, far from the classical endemic range. Türkiye remains the largest reported outbreak setting globally, with roughly 9,000 confirmed cases and a case-fatality rate near 5% since the 2002 emergence (Leblebicioglu et al., 2016), and Hawman and Feldmann (2023) emphasise continuing *Hyalomma* range expansion in the absence of any licensed vaccine or antiviral. Tick-borne encephalitis shows the same pattern of altitudinal and latitudinal expansion: Beauté et al. (2018) analysed EU/EEA surveillance for 2012–2016, reporting 12,500 cases with 98% concentrated in ten endemic countries and documented spread into previously unaffected areas (Ruzek et al., 2019).

Japanese encephalitis virus in Australia in 2022 is the clearest recent demonstration of how quickly a temperate system can be surprised. Waller et al. (2022) reported the sentinel fatal case



in the Northern Territory, and Mackenzie et al. (2022) analysed the emergence of genotype IV across south-eastern Australia — the first detection of that genotype outside Indonesia — spreading through piggeries and *Culex annulirostris* populations under La Niña flooding, with waterbirds and feral pigs as amplifying hosts. The outbreak was detected through veterinary investigation of reproductive losses in piggeries before the human dimension was recognised, which is an argument for animal-sector surveillance that no amount of clinical capacity would have substituted for.

4.4 Pandemic arboviral expansion in commensal vector systems

Two events of the past decade demonstrate what happens when an arbovirus escapes a wildlife-maintained cycle into a fully commensal urban vector system. Weaver and Lecuit (2015) document the global expansion of chikungunya, enabled in part by the E1-A226V substitution that improved viral fitness in *Aedes albopictus* and so extended transmission into temperate regions where that species, not *Ae. aegypti*, is the dominant vector. Musso and Gubler (2016) review the Zika pandemic, including the congenital syndrome and Guillain-Barré associations that were unrecognised in five decades of sporadic African and Asian circulation and became apparent only when the virus reached immunologically naive populations at scale. Both cases illustrate a general point: a pathogen's clinical profile is not fully characterised until it has been transmitted at epidemic scale, and surveillance systems calibrated on historical severity will systematically under-rate emerging arboviruses.

4.5 Climate-forecastable emergence: Rift Valley fever

Rift Valley fever is the exception that proves the rule about ecological predictability. Linthicum et al. (2016) describe the coupling of epizootics to El Niño–Southern Oscillation-driven heavy rainfall and the flooding of dambo habitats, where transovarially infected *Aedes* eggs hatch and initiate amplification in livestock before *Culex* secondary vectors expand transmission. Because the initiating condition is a measurable large-scale climate anomaly with a lead time of months, Rift Valley fever is genuinely forecastable — and the fact that it remains the only vector-borne zoonosis routinely forecast in this way indicates how rarely the necessary ecological understanding and monitoring infrastructure coincide.

4.6 The tropical blind spot: febrile illness aetiology

The case studies above are drawn disproportionately from high-income temperate settings, and that bias is itself the finding. Crump et al. (2013) prospectively investigated 870 febrile inpatients in northern Tanzania and found that malaria accounted for only 1.6% of admissions, while bacterial zoonoses accounted for 26.2% — leptospirosis 8.8%, Q fever 5.0%, spotted fever group rickettsioses 8.0% — and chikungunya for 7.9%; almost none were diagnosed clinically, and most patients had been treated presumptively for malaria. D'Acremont et al. (2014) systematically investigated 1,005 febrile Tanzanian outpatient children and found malaria in only 10.5%, with 96.5% of children who received antimalarials not having malaria. Maze et al. (2018) synthesise the sub-Saharan African literature and show that the decline of

malaria incidence in many settings has unmasked a large burden of undiagnosed arboviral and bacterial zoonotic infection for which no point-of-care diagnostics exist.

The consequence for this review's subject is direct. Emerging vector-borne pathogens in high-burden tropical settings are not absent from the data because they are absent from the population; they are absent because the diagnostic pathway terminates at a malaria rapid test. Any global assessment of where vector-borne emergence is occurring is therefore also, and inseparably, a map of where diagnostic capacity exists.

5. Surveillance Gaps

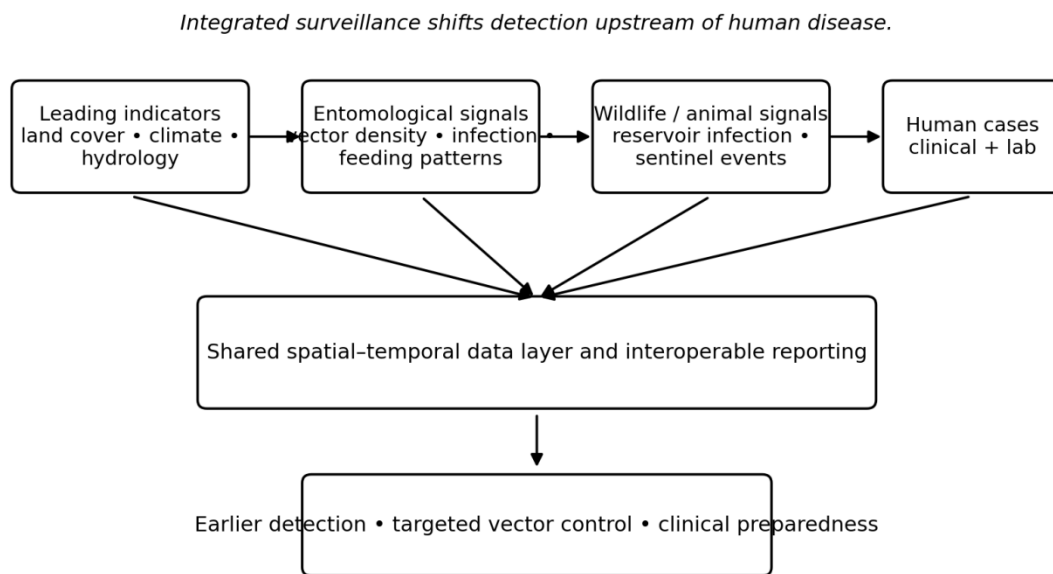


Figure 2. Integrated surveillance architecture that moves detection upstream from human clinical cases.

5.1 The erosion of entomological capacity in a high-income setting

The United States offers the best-documented case of surveillance decline, because the decline was measured. Rosenberg et al. (2018) reported 642,602 vector-borne disease cases to the Centers for Disease Control and Prevention for 2004–2016 — 491,671 tickborne (77%), 150,842 mosquitoborne (23%) — with tickborne cases more than doubling from approximately 22,000 in 2004 to 48,610 in 2016, and nine vector-borne human pathogens newly recognised or introduced during that period. Against this rising burden, Hadler et al. (2015) documented what happened to the surveillance system: federal arbovirus surveillance funding through the Epidemiology and Laboratory Capacity cooperative agreements fell to 39% of its 2004 peak, reaching \$9.3 million in 2012–2013; state arbovirus surveillance personnel fell 28% (from 702 to 503) and full-time equivalents 41% (from 348 to 206); 57% of states eliminated avian death surveillance, 58% reduced mosquito trapping and 68% reduced mosquito testing; and only 34%



of states reported that most local health departments conducted adult mosquito surveillance, down from 48% in 2004.

The capability assessment is equally stark. The National Association of County and City Health Officials (2017) surveyed 1,906 vector control organisations with a 57% response rate and found that 84% needed improvement in at least one of five core competencies, only 8% were fully capable, and 86% did not conduct pesticide-resistance testing; in 17 states, every responding programme was rated as needing improvement. Petersen et al. (2019) drew the structural conclusion, arguing that the United States has no integrated national vector-borne disease prevention and control system comparable to those maintained for other notifiable threats, and that the absence is a governance failure rather than a resource accident.

This matters beyond the United States for two reasons. First, if the country that experienced the largest arboviral invasion in modern history allowed its entomological capacity to decline by a third in the following decade, the assumption that capacity follows burden is not supported. Second, the decline is concentrated precisely in the activities — mosquito trapping, mosquito testing, avian mortality surveillance — that generate leading rather than lagging indicators. What was preserved was human case reporting, which is the component that cannot prevent anything.

5.2 Wildlife disease surveillance

If amplification occurs in wildlife, wildlife surveillance is the only place the leading signal exists. Delgado et al. (2023) surveyed 103 member countries of the World Organisation for Animal Health and found that only 54.4% operated a wildlife disease surveillance programme at all. The absence of dedicated budgets constrained outbreak investigation, sample collection and diagnostic testing; capacity was uniformly low rather than regionally clustered; and data analysis and disease risk assessment were the most commonly cited unmet needs. Kelly et al. (2017) described the PREDICT project as a proof of concept for transdisciplinary viral surveillance at the human–wild animal interface, but the proof-of-concept framing is itself telling: two decades after the One Health concept entered policy discourse, systematic wildlife pathogen surveillance remains a demonstration activity rather than a routine function.

The institutional cause is analysed by Destoumieux-Garzón et al. (2018), who trace the persistent fragmentation between human, animal and environmental health sectors. Vectors and wildlife reservoirs fall between mandates: human health ministries are funded to detect human cases, veterinary services are funded to protect livestock trade, and environment ministries are funded for conservation. None is funded to monitor the arthropod that connects them. Mackenzie and Jeggo (2019) make the argument in the specific context of vector-borne and wildlife-associated zoonoses, where the mandate gap is widest.

5.3 Data standardisation and interoperability

A surveillance system that cannot combine its own outputs cannot detect distributional change. Kraemer et al. (2015b) built the first openly available global compendium of *Ae. aegypti* and



Ae. albopictus occurrence records — 19,930 and 22,137 records respectively — explicitly to address the absence of standardised, interoperable global vector occurrence data, and the derived global maps (Kraemer et al., 2015a) remain the reference layer for a very large fraction of published risk modelling. The fact that a research group had to assemble this dataset from scattered sources, and that no comparable resource exists for most tick and sandfly vectors, is a structural indictment of vector data governance.

The problem is a familiar one in other monitoring domains, and the solutions developed there are transferable. Integrated data visualisation frameworks for continuous performance monitoring demonstrate how heterogeneous instrument and process streams can be rendered into decision-relevant displays rather than archived reports (Ogbole et al., 2021); real-time machine-learning risk dashboards built for hospital operations show the same pattern applied to health-system data (Filani et al., 2022); and sensor-based fault detection systems demonstrate that continuous monitoring of a distributed physical system can reliably surface anomalies before failure, provided the data architecture is designed for it (Sunday & Omoegun, 2022). The corresponding literature on statistical methods and quality engineering establishes what is required to make such monitoring trustworthy — defined measurement systems, control limits and validated analytical procedures (Adaramola et al., 2023) — while reliability engineering and failure analysis frameworks supply the vocabulary for reasoning about how complex multi-component monitoring systems fail (Gideon et al., 2019). Vector surveillance has not generally been designed to these standards, and its failure modes are correspondingly under-analysed.

Two further transferable elements deserve mention. Predictive analytics developed for demand forecasting demonstrate the value of building forecast evaluation into operational systems from the outset rather than retrofitting it (Aifuwa et al., 2020), and privacy-centric security engineering frameworks address the data-protection constraints that in practice limit cross-sectoral sharing of surveillance data (Okoruwa et al., 2020). The latter is not a peripheral concern: much of the reluctance to share animal, human and environmental health data across institutional boundaries is framed in terms of confidentiality and legal exposure, and technical architectures that resolve that framing are a precondition for integration.

5.4 The diagnostic gap as a surveillance gap

Section 4.6 established that arboviral and bacterial zoonotic febrile illness is systematically misclassified in high-burden tropical settings. The surveillance consequence deserves stating plainly: a pathogen that cannot be diagnosed cannot be notified, and a pathogen that cannot be notified does not exist in any surveillance dataset, regardless of its true incidence. This produces a self-reinforcing distortion in which apparent emergence tracks diagnostic investment. When investigators deploy comprehensive aetiological panels in African febrile illness cohorts, they find substantial arboviral and rickettsial burden; when they do not, they find malaria. The published global distribution of emerging vector-borne disease is therefore partly an artefact, and policy built on it will systematically under-resource the settings with the least capacity.



5.5 Supply chain and resilience constraints

A final, under-discussed gap concerns the commodity systems on which surveillance and control depend. Entomological surveillance requires traps, CO₂, preservation media and reagents; molecular testing requires cold chain and consumables; vector control requires insecticide supply and application equipment. In practice these supply chains are fragile, and the resulting interruptions are recorded as capacity failures rather than logistics failures. Work on supplier relationship management and supply chain resilience in complex global systems establishes the general principle that resilience derives from supplier relationship structure and redundancy rather than from inventory alone (Ike et al., 2021), and end-to-end visibility frameworks demonstrate how traceability across multi-tier chains can be made operational (Nnabueze et al., 2021). Applying this reasoning to surveillance commodities is straightforward and has largely not been done.

6. Public Health Implications and Response Options

6.1 What the burden data can and cannot support

The preceding sections imply that recorded vector-borne disease burden is jointly determined by transmission and detection, and that the two cannot be separated with existing data. Three practical consequences follow. First, apparent increases in incidence following a diagnostic or surveillance investment should not be interpreted as emergence, and apparent stability in low-capacity settings should not be interpreted as absence. Second, trend analyses that span periods of capacity change — which, given the United States data above, includes most long time series — require explicit adjustment or explicit acknowledgement of confounding. Third, the geographic distribution of published emergence events is not a valid basis for prioritising surveillance investment, because it is partly generated by that investment.

6.2 Vector control: what works and what is failing

Vector control remains the intervention with the strongest historical record against vector-borne disease and is indispensable to elimination targets (Wilson et al., 2020). Two developments dominate the current picture.

Wolbachia deployment is the most significant new tool. Hoffmann et al. (2011) demonstrated the first successful field establishment of wMel in *Ae. aegypti*, reaching near-fixation in two Cairns suburbs within five weeks of releases ending. Utarini et al. (2021) then reported the Applying *Wolbachia* to Eliminate Dengue cluster-randomised trial in Yogyakarta, Indonesia: 12 intervention and 12 control clusters, 8,144 participants enrolled and 6,306 analysed under a test-negative design, with protective efficacy of 77.1% (95% CI 65.3–84.9) against symptomatic virologically confirmed dengue and 86.2% (95% CI 66.2–94.3) against dengue hospitalisation. Ryan et al. (2020) reported sustained establishment and reduced local transmission under programmatic deployment in northern Queensland. This is the strongest



efficacy evidence available for any vector-borne disease intervention, and it applies to a commensal urban vector rather than to a wildlife-interface system.

Insecticide resistance is the countervailing trend. Hemingway et al. (2016) warned that pyrethroid resistance in African *Anopheles* — the chemical class on which both long-lasting insecticidal nets and much indoor residual spraying depend — threatens to reverse gains that halved *Plasmodium falciparum* infection prevalence in endemic Africa between 2000 and 2015, a decline to which insecticide-treated nets contributed an estimated 68% of averted cases (Bhatt et al., 2015). Ranson and Lissenden (2016) document the spread and intensification of target-site and metabolic resistance mechanisms, and Moyes et al. (2017) show the same problem in *Aedes*, with widespread pyrethroid and organophosphate resistance across the Americas and Asia and resistance testing performed too infrequently to guide product selection. The NACCHO finding that 86% of United States vector control programmes do not conduct resistance testing (National Association of County and City Health Officials, 2017) indicates that this is not solely a low-income-country problem.

The strategic framework is WHO's *Global Vector Control Response 2017–2030* (WHO, 2017), built on four pillars — strengthened inter-sectoral action, engaged and mobilised communities, enhanced vector surveillance and monitoring and evaluation, and scaled-up integrated vector control — resting on two foundations of entomological capacity and expanded research. Roiz et al. (2018) operationalise this as integrated *Aedes* management. The framework is sound; the capacity assessments in Section 5 indicate it is not being implemented.

6.3 Clinical and health system implications

For clinicians and health systems, the practical implications of emergence at the wildlife–human interface are mostly implications about index-of-suspicion and about the speed of the clinical pathway. Emerging vector-borne infections present as undifferentiated fever, and the distinguishing features are exposure history and geography rather than clinical syndrome. In settings where emergence is occurring at the edge of a known range, the pathway from presentation to specific diagnosis depends on whether a front-line clinician thinks of the possibility and whether a specimen can reach a laboratory capable of testing for it.

Evidence from quality improvement in acute care is relevant here, and under-used in the emerging infection literature. Nurse-led quality improvement in clinical decision units has been shown to be an effective mechanism for changing front-line detection and escalation behaviour, precisely because it operates at the point where undifferentiated presentations are triaged (Ayinde et al., 2023), and nurse leadership has been documented as a determinant of infection control performance during healthcare crises, when standard operating procedures are least adequate to the situation (Ohunyon et al., 2023). Both findings point the same way: the sensitivity of a surveillance system to a novel pathogen depends heavily on front-line clinical behaviour, and front-line clinical behaviour is modifiable through structured quality improvement rather than only through guideline issuance.

6.4 Governance

The One Health High-Level Expert Panel definition, adopted by the Quadripartite in December 2021 and published by Adisasmito et al. (2022), defines One Health as “an integrated, unifying



approach that aims to sustainably balance and optimize the health of people, animals and ecosystems,” explicitly bringing ecosystems into the operational definition rather than treating them as context. This is a meaningful advance for vector-borne disease, where the vector sits outside every single-sector mandate.

Implementation, however, rests on the International Health Regulations (2005), whose weaknesses Gostin and Katz (2016) analyse: no enforcement mechanism, chronic under-reporting driven by trade and travel penalties, and failure by most States Parties to meet core capacity deadlines. Kandel et al. (2020), analysing IHR annual reporting from 182 countries immediately before COVID-19, found that only 52% had strong operational readiness capacity and just 28% had strong emergency-response operations, with the weakest scores concentrated in the African and Eastern Mediterranean regions — that is, in the regions with the largest vector-borne disease burden and the thinnest entomological surveillance.

Morens and Fauci (2020) place this in the longer arc of pandemic emergence driven by human ecological disruption and argue that preparedness must extend upstream to the drivers of spillover rather than remaining a detection-and-response enterprise. Plowright et al. (2021) make the corresponding case for land-use-based primary prevention as the most cost-effective tier of preparedness. The evidence reviewed in Section 3 supports this position for vector-borne pathogens specifically: if zoonotic host abundance is systematically elevated in human-modified landscapes, and if spillover risk peaks at intermediate land conversion, then land-use planning is a vector-borne disease intervention whether or not it is administered as one.

7. A Research and Policy Agenda

Five priorities follow from the evidence reviewed.

- Restore and standardise entomological surveillance. The capacity decline documented by Hadler et al. (2015) and the capability gaps documented by NACCHO (2017) are both remediable with modest resources, and both concern the leading-indicator components of surveillance. Minimum national standards for adult vector trapping, pathogen testing and insecticide resistance monitoring — with published compliance reporting — would be a low-cost, high-yield reform. Petersen et al. (2019) set out the institutional form this should take.
- Treat host-feeding and host community data as core surveillance variables. The West Nile virus literature established that vector abundance without host-feeding data cannot predict human risk (Kilpatrick, Kramer, et al., 2006). Blood-meal analysis is now inexpensive and should be a routine component of arboviral vector surveillance rather than a research activity.
- Close the tropical diagnostic gap. The febrile-illness aetiology literature (Crump et al., 2013; D’Acremont et al., 2014; Maze et al., 2018) identifies the single largest distortion in global vector-borne disease data. Multiplexed, deployable diagnostics for the principal arboviral and bacterial zoonotic causes of acute febrile illness would change both clinical management and the global evidence base.
- Build wildlife and vector surveillance into environmental monitoring rather than beside it.



Given that only just over half of national veterinary authorities operate wildlife disease surveillance (Delgado et al., 2023), the realistic route to ecological early warning is to attach pathogen and vector sampling to existing environmental and biodiversity monitoring infrastructure, and to treat land-cover change as a surveillance data stream in its own right.

- Develop mechanism-specific models of biodiversity–disease relationships. Halsey’s (2019) proposal — mechanism, scale and endpoint specified before testing — should become the default standard for this literature, replacing the general dilution/amplification framing that has absorbed two decades of effort without resolution.

8. Conclusion

Emerging vector-borne pathogens at the wildlife–human interface are not a category of exotic accident. They are the predictable output of three measurable processes: land-use change that reassembles host and vector communities in favour of competent reservoir species and anthropophilic vectors; climate change that redistributes transmission suitability across latitude, altitude and season; and transmission dynamics dominated by heterogeneity in host competence and vector feeding preference, such that a handful of species determine risk. Each of these processes is observable with existing methods.

What is not observable, in most of the world, is the result. Entomological surveillance capacity in the highest-income setting with the most recent major arboviral invasion declined by roughly a third in the decade after that invasion. Fewer than 55% of national veterinary authorities conduct wildlife disease surveillance. There is no standardised global vector occurrence dataset for most vector taxa. And in the settings with the greatest burden, undifferentiated febrile illness is presumptively attributed to malaria, so that the arboviral and rickettsial fraction of that burden is never counted.

The public health implication is that the principal deficit in emerging vector-borne disease is not scientific but observational and institutional. The ecology is reasonably well understood; the mechanisms of emergence are documented; the interventions with the best evidence, from integrated vector management to *Wolbachia* deployment, exist and work. What is missing is a surveillance architecture configured to observe the ecological processes that generate emergence, an institutional home for the arthropod that connects the sectors, and diagnostic capacity in the places where the burden is largest. Until those gaps close, the field will continue to characterise emergences retrospectively, one surprise at a time.

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