Eurasia Specialized Veterinary Publication

International Journal of Veterinary Research and Allied Science

ISSN:3062-357X

2026, Volume 6, Issue 1, Page No: 100-111

Available online at: www.esvpub.com/

Climate-Sensitive Animal Disease Surveillance Must Anticipate Ecological Change Before Outbreak Counts Rise: A Veterinary Early-Warning Architecture Integrating Vectors, Hosts, Weather, Landscape, Movement, Exposure, and Response

Luca Ferraro[1]*, Matteo Ricci[1], Giulia Moretti[2], Paolo Greco[3]

[1]Department of Veterinary Climate-Sensitive Disease Surveillance, Faculty of Veterinary Medicine, University of Bologna, Bologna, Italy.
[2]Department of Veterinary Vector-Borne Disease Ecology, Faculty of Veterinary Medicine, University of Turin, Turin, Italy.
[3]Department of Veterinary Early-Warning Systems and Response, Faculty of Veterinary Medicine, Sapienza University of Rome, Rome, Italy.

***E-mail** ✉ luca.ferraro@unibo.it

ABSTRACT

Climate-sensitive animal diseases can shift before conventional surveillance registers a rise in diagnosed cases, creating a mismatch between ecological change and outbreak recognition. This article develops an original non-empirical veterinary early-warning architecture for interpreting upstream changes in weather, vector biology, host distribution, landscape, animal movement, exposure, and response capacity before they are converted into confirmed disease counts. The synthesis distinguishes climate as a disease-system modifier from climate as a sufficient cause; pathogen development from vector biology; precipitation from hydrological state; vector abundance from competence; ecological suitability from demonstrated transmission; and exposure opportunity from confirmed infection. The central contribution is a proposed architecture in which heterogeneous environmental and veterinary signals are interpreted as trajectories, concordant or discordant evidence, and decision-relevant warning states rather than as a universal numerical score. The framework is designed to preserve uncertainty and to link escalating ecological concern with proportionate surveillance and cross-sector response while maintaining explicit boundaries between evidence-supported relationships and proposed decision logic. Its utility will depend on local vectors, pathogens, host populations, production systems, movement networks, surveillance intensity, and data completeness. It does not establish prospective predictive performance, causal attribution, or intervention effectiveness. Climate-sensitive surveillance should therefore be judged by whether it detects meaningful ecological transition early enough to improve targeted observation and preparedness without converting uncertain environmental signals into premature outbreak claims.

Keywords: Climate-sensitive surveillance, Veterinary epidemiology, Vector-borne disease, Early warning, One Health, Ecological change

Received: 29 January 2026
Revised: 17 April 2026
Accepted: 19 April 2026

How to Cite This Article: Ferraro L, Ricci M, Moretti G, Greco P. Climate-Sensitive Animal Disease Surveillance Must Anticipate Ecological Change Before Outbreak Counts Rise: A Veterinary Early-Warning Architecture Integrating Vectors, Hosts, Weather, Landscape, Movement, Exposure, and Response. Int J Vet Res Allied Sci. 2026;6(1):100-11.
https://doi.org/10.51847/B08Ezg6kpx

Introduction

Veterinary surveillance often becomes most visible when laboratory confirmations, syndromic reports, mortality, or outbreak counts increase. For climate-sensitive infections, however, relevant ecological conditions may change

earlier. Temperature, rainfall, humidity, land use, host mobility, and human activity can alter vector reproduction, survival, distribution, and contact opportunities, but these effects occur within coupled ecological and anthropogenic systems rather than through climate alone [1]. European West Nile virus (WNV) modeling further indicates that climatic change can shift modeled spatial suitability even when land-use and population changes are considered, although suitability is not equivalent to observed transmission [2].

The surveillance problem is therefore interpretive: which upstream changes warrant greater attention before disease counts rise? Climate-driven early-warning methods require appropriate spatiotemporal models, adequate data, validation, and explicit uncertainty handling rather than uncritical use of meteorological associations [3]. Longitudinal canine leishmaniasis evidence also shows why intervention context matters: infection patterns were associated with winter relative humidity while preventive measures were associated in the opposite direction in one endemic region [4]. This article consequently develops a proposed veterinary architecture that separates ecological modification, exposure, surveillance evidence, warning interpretation, and response, while preserving disease-, species-, place-, and time-specific limits.

Climate as a disease-system modifier

Climate should be treated as a modifier of a disease system, not a stand-alone exposure that maps directly onto future case counts. Temperature, precipitation, and humidity can reshape vector survival, reproduction, distribution, biting opportunity, and seasonality, while land use, mobility, host availability, and behavior can amplify, suppress, or redirect those effects [1]. This framing matters operationally because the same meteorological anomaly may have different consequences when habitat, vector populations, susceptible hosts, or preventive practices differ.

Attribution also depends on the counterfactual being considered. In European WNV modeling, climate trends contributed materially to expansion of modeled suitability after accounting for land-use and population change, but the model does not exclude all ecological alternatives or establish that climate alone caused observed transmission [2]. For surveillance, the proposed implication is that climate signals should initiate ecological interpretation rather than function as diagnoses. A weather anomaly becomes more informative when it aligns with vector, host, landscape, movement, or exposure changes; discordance should remain visible rather than being forced into a single climate-risk label.

Temperature and pathogen development

Temperature can alter pathogen development inside vectors before surveillance records additional infections. For WNV in Europe, temperature-dependent transmission modeling shows that thermal conditions can shift the modeled boundary under which circulation becomes feasible, conditional on vector-host ratios and other ecological parameters [5]. Such a boundary is a mechanistic suitability construct, not a universal outbreak threshold.

Experimental evidence provides a complementary distinction. In *Culex pipiens pipiens*, WNV transmission competence increased under warmer tested conditions, demonstrating that thermal effects can act within the vector-pathogen interaction rather than only through vector abundance [6]. Bayesian modeling of experimental WNV data likewise indicates that warmer temperature shortens the extrinsic incubation period, potentially reducing the time required for an infected mosquito to become capable of transmission [7].

For early warning, these findings support monitoring thermal trajectories and biologically meaningful lags. They do not justify transferring one temperature cutoff, competence estimate, or incubation period across vector populations, viral strains, or regions. Temperature-dependent pathogen development is therefore an upstream component whose interpretation requires concurrent information about vector survival, abundance, hosts, and exposure.

Temperature and vector biology

Temperature also acts on the vector itself, and that process should remain analytically distinct from temperature-dependent pathogen development. Across vector-borne disease systems, thermal conditions can influence vector reproduction, survival, activity, geographic distribution, and the duration of seasonal suitability [1]. A warmer period can therefore alter the number, location, or behavior of vectors even when the pathogen-development effect is uncertain.

The two thermal pathways can interact without being interchangeable. Experimental WNV work in *Culex pipiens* demonstrates temperature-sensitive transmission competence under defined laboratory conditions [6], but field transmission additionally depends on whether vectors survive long enough, occur at sufficient abundance, encounter susceptible hosts, and participate in the relevant feeding network. The proposed early-warning architecture therefore treats "thermal vector state" and "thermal pathogen state" as separate evidence lanes. Concordance between them may strengthen concern; discordance may identify an incomplete transmission pathway. Neither lane is converted into a universal score, because species-specific thermal limits, acclimation, microclimate, habitat, and local seasonal dynamics can change what the same ambient temperature means biologically.

Precipitation and hydrological change

Precipitation is an imperfect surrogate for the water conditions that vectors actually experience. In California, irrigation was associated with higher and more stable mosquito abundance and with higher WNV incidence, illustrating how anthropogenic water supply can weaken a simple rainfall-to-vector interpretation [8]. Hydrological surveillance should therefore distinguish rainfall events from persistent, managed, or redistributed water availability.

Local interface conditions further modify that relationship. At wildlife-livestock interfaces in Spain, environmental determinants of WNV-vector abundance differed across sampling settings, supporting ecological calibration at the scale where hosts and vectors interact [9]. In a managed United Kingdom wetland, vegetation structure strongly shaped mosquito community composition, showing that water presence alone does not specify which vector community develops [10].

For decision-making, the relevant question is not merely whether rainfall increased, but whether precipitation or water management changed breeding habitat, habitat persistence, community composition, or host-vector contact in a direction consistent with the focal disease system. **Table 1** presents from rainfall to vector habitat: hydrological translations that alter surveillance priority.

Table 1. From rainfall to vector habitat: hydrological translations that alter surveillance priority

Weather input	Water or habitat response	Local controller	Entomological or environmental confirmation	Surveillance shift	Transmission claim withheld	Basis
Rain increase	No demonstrated habitat response	Drainage, retention, land cover	Rain anomaly only	Do not escalate on rainfall alone	**Limit:** Water may remain unavailable to vectors **Next evidence:** Check habitat persistence and vectors	Proposed
Weak/inconsistent rain	Irrigation-sustained water	Agricultural water management	Persistent water; stable/rising mosquitoes	Raise ecological attention	**Limit:** Association is setting-specific **Next evidence:** Recheck irrigation, vectors, infection	Evidence-bounded
Local water change	Interface-specific habitat response	Host presence, vegetation	Environmental shift plus vector increase	Prioritize interface surveillance	**Limit:** Abundance is not competence **Next evidence:** Add host-contact and pathogen evidence	Evidence-bounded
Variable rainfall	Managed wetland restructuring	Vegetation and management	Altered larval habitat/community	Revise expected vector assemblage	**Limit:** Community change is not transmission **Next evidence:** Confirm adult vectors and circulation	Evidence-bounded

Humidity and environmental persistence

Humidity can influence vector ecology, but the evidence base does not justify treating it as a universal mechanism of pathogen persistence. Reviews of vector-borne disease ecology identify humidity among climatic factors that can modify vector survival, activity, and distribution, with effects that vary among vectors and settings [1]. The surveillance meaning of humidity therefore depends on the biological pathway under consideration.

The canine leishmaniasis series from northwestern Spain provides a useful bounded example: maximum winter relative humidity was positively associated with new infection patterns, while preventive measures were negatively associated in the same long-term system [4]. That observation supports humidity as a potentially

informative ecological covariate, but it does not establish that humidity directly caused infection, prolonged environmental survival of *Leishmania*, or would behave similarly in another host-vector system.
Accordingly, the proposed architecture places humidity in an "ecological condition" lane unless pathogen-specific evidence supports a direct persistence mechanism. Reassessment should ask whether humidity aligns with vector survival, habitat persistence, activity, or exposure observations. Where those pathways are unmeasured, uncertainty should remain explicit rather than being converted into a stronger mechanistic claim.

Vector abundance and competence

An increase in vectors is not equivalent to an increase in transmission capability. During the Dutch BTV-3 epidemic, extensive *Culicoides* sampling identified abundant species and BTV-3-positive pools, supporting field involvement but not, by itself, proving transmission competence [11]. Pathogen detection in a trapped vector can reflect exposure, infection, or carriage without demonstrating successful transmission to another host.
Competence requires a different evidential standard. Experimental work with field-derived *Aedes japonicus japonicus* and *Culex pipiens* showed that WNV competence can vary across mosquito-virus combinations under controlled conditions [12]. Host-feeding data add a third dimension: molecular identification of blood meals in native United Kingdom mosquitoes demonstrated contact with multiple vertebrate hosts, informing potential bridge-vector pathways without proving pathogen transmission [13].
The architecture therefore separates vector abundance, field pathogen positivity, intrinsic competence, and host contact. These domains can converge, but they should not substitute for one another. A rising vector count may justify intensified sampling; a positive vector pool may justify pathogen-focused investigation; demonstrated competence may strengthen biological plausibility; and host-feeding evidence may identify who is exposed. Their combined interpretation remains pathogen-, vector-, and place-specific.

Wildlife host redistribution

Climate-sensitive surveillance must also consider whether susceptible, amplifying, or reservoir hosts are changing where and when they overlap with vectors. European WNV modeling indicates that climatic change can alter the geography of ecological suitability [2]. That evidence does not directly measure wildlife redistribution, but it makes host presence within newly suitable areas a relevant question rather than an assumed constant.
Interface studies show why this distinction matters. At wildlife-livestock interfaces, mosquito abundance varied with local environmental conditions [9]. If wildlife distributions, seasonal concentrations, or habitat use shift, the epidemiologically relevant effect may arise through changed contact structure rather than through weather alone. Conversely, a climate anomaly without host availability may produce little transmission opportunity.
The proposed architecture therefore treats wildlife redistribution as a conditional host-state signal. Useful observations include altered seasonal occupancy, aggregation near water or feed, changes in migratory timing, and emergence of new overlap zones with vectors or domestic animals, but these require disease-specific evidence before being interpreted as transmission. Wildlife movement should be paired with vector and pathogen surveillance, and absence of direct host data should be recorded as uncertainty rather than inferred from climate suitability.

Livestock exposure

Livestock exposure sits between ecological suitability and observed infection. In Turkish small ruminants, BTV seroprevalence and associated risk factors varied across animal and flock conditions, while insecticide use showed a protective association, demonstrating that exposure is partly structured by management rather than climate alone [14]. The same ecological hazard can therefore produce different contact opportunities across holdings.
Exposure can also be modified physically. In a United Kingdom field study, closed-door vector-proof accommodation with door brushes markedly reduced indoor *Culicoides* catches, whereas additional fine mesh did not clearly improve the strongest configuration [15]. Reduced vector entry is not equivalent to proven disease prevention, and prolonged housing may create practical or welfare constraints.
Surveillance must also recognize that first detection may lag spatial spread. When BTV-12 was identified in the Netherlands in 2024, follow-up and retrospective testing identified additional affected farms, while the introduction route remained uncertain [16]. **Table 2** presents livestock exposure configurations and the evidence needed to reconstruct infection extent.

Table 2. Livestock exposure configurations and the evidence needed to reconstruct infection extent

Livestock configuration	Exposure mechanism and controller	Evidence available	Spatial or temporal use	Unknown separating exposure from infection	Basis
Heterogeneous flock exposure	**Meaning:** Contact opportunity varies among holdings **Controller:** Husbandry, insect control, species	Vector season plus flock risk conditions	Target higher-exposure holdings	**Unknown:** Risk factors are not climate thresholds **Reconstruction:** Recheck vectors, management, diagnostics	Evidence-bounded
Physical vector exclusion	**Meaning:** Contact may be reduced **Controller:** Barrier integrity, housing feasibility	Lower indoor vector entry	Adjust exposure interpretation	**Unknown:** Catch reduction is not disease prevention **Reconstruction:** Recheck barriers and animal constraints	Evidence-bounded
First confirmed emerging-serotype detection	**Meaning:** Recognized infection may underestimate extent **Controller:** Diagnostic coverage, submission patterns	Typed positive premises	Expand investigation	**Unknown:** Introduction route may remain unknown **Reconstruction:** Test backward and outward	Evidence-bounded
Retrospective additional positives	**Meaning:** Earlier unrecognized spread becomes plausible **Controller:** Sampling history, coverage	Newly recognized stored/follow-up positives	Revise spatial-temporal extent	**Unknown:** Detection does not identify cause **Reconstruction:** Reconstruct exposure and movement history	Proposed

Companion-animal exposure

Companion animals can indicate changing exposure where they share vector environments with people, wildlife, or livestock, but their surveillance value depends on disease ecology and prevention. The long-term canine leishmaniasis evidence demonstrates that repeated testing can reveal temporal infection patterns while prevention modifies the observed signal [4]. A decline in detected infection need not imply declining ecological suitability if prevention simultaneously reduces contact.

Within the proposed architecture, companion-animal data are interpreted as an exposure-sensitive veterinary stream rather than a direct climate indicator. Useful changes include altered seasonality of diagnoses, expansion into previously low-exposure areas, or divergence between environmental suitability and animal infection. Such signals require vector, diagnostic, and contextual confirmation. Companion-animal surveillance is vulnerable to uneven veterinary attendance, prevention, travel, diagnostic practice, and owner constraints. These factors should remain explicit because they can generate apparent geographic or temporal changes unrelated to local ecological transmission.

Land-use and landscape change

Climate operates within landscapes that determine where hosts, vectors, water, and production systems meet. In Bangladesh, projected avian-influenza suitability changed when climate and land-use trajectories were modeled jointly, illustrating that future ecological risk cannot be inferred from climate variables in isolation [17]. The projection remains a suitability model rather than a validated forecast of future outbreaks.

Landscape also structures movement. Livestock-network research in East Africa shows that contact patterns differ among production systems and change with seasonal resource availability [18]. At a broader scale, European WNV phylogeography associates viral spread with bird movement, wetlands, migratory pathways, and agricultural intensity [19]. These studies support a surveillance distinction between background landscape, changing landscape state, and movement through that landscape. The architecture treats land-use conversion, irrigation, wetland alteration, farm density, habitat, and resource distribution as contact modifiers. Their importance is conditional: a landscape change becomes more warning-relevant when it converges with suitable vectors, susceptible hosts, pathogen evidence, or changing movement.

Animal movement

Animal movement can transport susceptible, infected, or vector-exposed animals across ecological boundaries faster than local case surveillance reveals changing risk. Seasonal livestock networks demonstrate that connectivity is not fixed: resource availability, production system, trade, and congregation can alter who contacts

whom and when [18]. Wildlife-associated pathogen spread likewise reflects movement through heterogeneous landscapes rather than a simple function of geographic distance [19].

Movement should therefore be represented as a dynamic exposure structure. For livestock, signals may include market flows, transhumance, border crossings, or aggregation. For wildlife, migratory timing and redistribution can modify interface contact. The architecture does not assume that high network centrality means infection; movement becomes actionable only when connected to pathogen, exposure, or ecological evidence. **Table 3** presents movement processes that relocate climate-sensitive surveillance targets.

Table 3. Movement processes that relocate climate-sensitive surveillance targets

Movement process	Interface reshaped	Movement record or observation	Sampling relocation	Unsafe network inference	Refresh interval and basis
Seasonal livestock reconfiguration	**Meaning:** Contact structure may shift before cases **Driver:** Resource scarcity, production system, markets	Changing routes, aggregation, trade intensity	Reprioritize sampling locations	Connectivity is not infection	**Refresh:** Reassess by season and pathogen **Basis:** Evidence-bounded
Wildlife dispersal or migration shift	**Meaning:** Interface opportunity may change **Driver:** Habitat, flyways, wetlands, food/water	Changed occupancy or movement corridors	Intensify interface observation	Association does not prove transmission	**Refresh:** Pair with vector/pathogen data **Basis:** Evidence-bounded
Cross-boundary livestock movement	**Meaning:** Exposure can be imported or redistributed **Driver:** Border controls, quarantine, immunity	Movement records and destination links	Target entry points and recipient networks	Recorded movement may be incomplete	**Refresh:** Audit movement-data coverage **Basis:** Proposed
Static network with changing ecology	**Meaning:** Historical centrality may become misleading **Driver:** Season, land use, movement restrictions	Discordance between current flows and old network	Avoid fixed sentinel assumptions	Network topology is time-sensitive	**Refresh:** Rebuild or update network **Basis:** Proposed

Integrating meteorological and veterinary signals

Early warning becomes more informative when independent signal types converge. Sentinel chickens in the Netherlands detected WNV circulation that had not been recognized by other surveillance streams, demonstrating the value of complementary animal surveillance [20]. Long-term corvid surveillance in northern Italy also showed that early-detection performance depends on sampling intensity; sparse surveillance can create apparent absence rather than true absence [21].

Integration should be organized around decisions rather than data accumulation. One Health surveillance scholarship emphasizes cross-sector linkage between information and intervention [22]. Spatial modeling in the United Kingdom further illustrates how vector distributions, host distributions, climate, and exposed populations can be combined to identify potential WNV risk areas while remaining distinct from confirmed transmission [23]. The proposed architecture consequently asks three questions of each signal: what process does it represent, how independent is it from other signals, and what decision could reasonably follow? Concordance can justify escalation of surveillance; discordance can identify missing data or alternative explanations. No signal is automatically privileged because of technological sophistication or temporal proximity.

A proposed climate-sensitive early-warning architecture

The central contribution is a proposed architecture that separates four functions: ecological state detection, exposure translation, veterinary surveillance confirmation, and response selection. Climate and landscape variables first indicate changing ecological conditions; vector, host, and movement evidence then determine whether those conditions plausibly alter contact. Veterinary observations establish whether exposure or circulation is becoming detectable, while response decisions remain downstream of an explicit interpretation boundary. This organization extends coupled-system reasoning [1], climate-EWS methodological principles [3], complementary sentinel evidence [20], and decision-linked One Health surveillance [22] without claiming that the combined architecture has been prospectively validated.

Escalation is proposed to depend on trajectory, concordance, persistence, and decision relevance rather than one universal threshold. Sampling adequacy must modify confidence because weak surveillance can mimic absence

[21]. Potential-risk surfaces should likewise remain upstream of confirmation because integrated ecological models can identify plausible risk without demonstrating transmission [23].

The architecture keeps discordance visible rather than averaging it into a score, triggering reassessment of missing biological links, data quality, or competing explanations.

The figure is needed to show how multiple signal lanes converge without collapsing ecological suitability, exposure, detection, and response into the same evidential state.

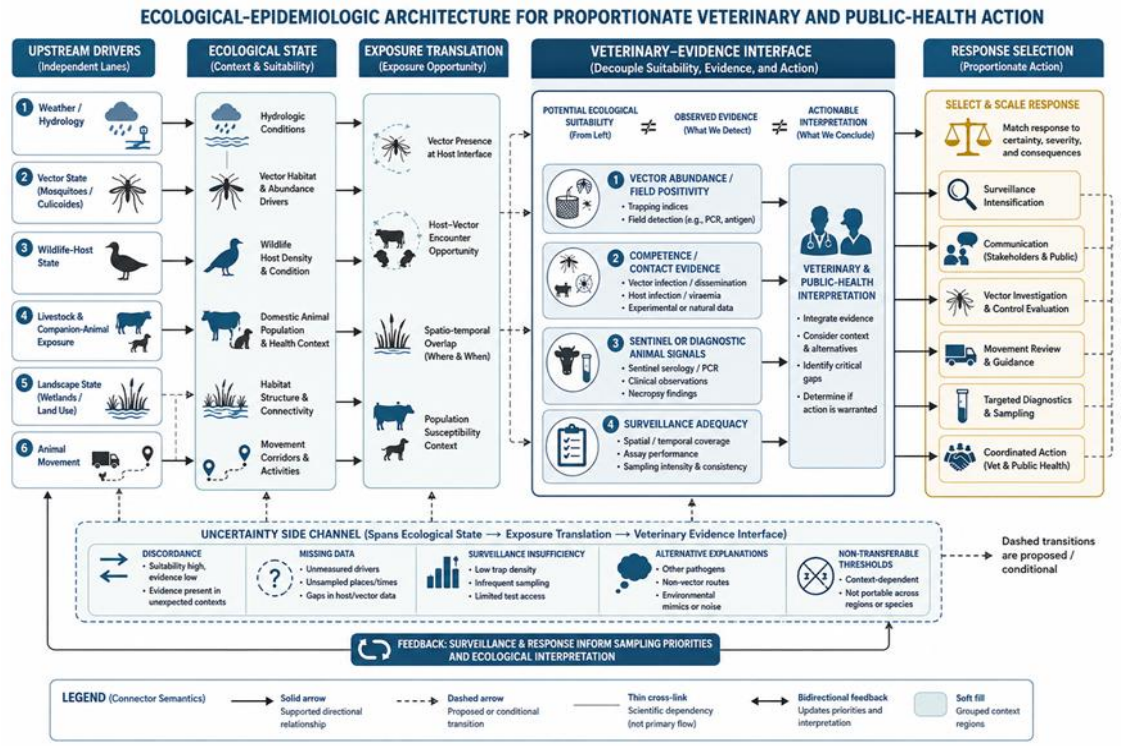

Figure 1. Ecological-to-action interfaces that separate climate signal, exposure opportunity, veterinary detection, and response

Warning states and alert thresholds

Warning states should track changing surveillance objectives rather than fixed cutoffs. During the emergence of Schmallenberg virus, the relative value of passive, syndromic, and targeted surveillance changed as objectives moved from early detection toward estimating extent and subsequent monitoring [24]. This supports phase-sensitive interpretation rather than one permanent alert rule.

Thresholds also carry resource consequences. Scenario-tree work on bluetongue surveillance shows that sensitivity, timeliness, and cost depend on assumptions about design prevalence, herd coverage, and surveillance components [25]. Passive signals are additionally non-specific: evaluation of cattle excess-mortality notification found variation in reporting and statistical performance, including failure of herd-level models to converge [26]. Network-based targeted testing can improve simulated early detection, but such optimized strategies remain model-dependent until prospectively tested [27].

The architecture therefore proposes qualitative warning states—ecological change, exposure convergence, surveillance suspicion, and confirmed circulation—whose operational thresholds must be locally calibrated. Movement between states should require decision-appropriate evidence, not accumulated points.

Uncertainty and false signals

Early-warning systems fail in both directions. A meteorological anomaly can generate concern without a competent vector, susceptible host, or transmission pathway, while inadequate surveillance can conceal real circulation. Model misspecification and nonstationarity can distort climate-derived warnings [3]. Sparse sentinel sampling can also convert insufficient observation into apparent absence [21], whereas excess-mortality signals may respond to non-infectious causes or reporting behavior [26].

A second error arises when modeled suitability is mistaken for observed transmission. Temperature-dependent reproduction models represent biological potential under assumptions [5], while integrated vector-host risk maps identify plausible areas rather than confirmed circulation [23]. The proposed architecture therefore requires each alert to carry an uncertainty statement identifying which link is observed, inferred, or unmeasured.
Table 4 presents differential diagnosis of climate, suitability, non-detection, and syndromic warning discordance.

Table 4. Differential diagnosis of climate, suitability, non-detection, and syndromic warning discordance

Observed warning combination	Inferential error	Missing biological stream	Surveillance experiment that discriminates	Basis
State: Climate signal without biological corroboration **Pattern:** Weather anomaly only	Ecological alarm overinterpreted	**Condition:** Vector absence, unsuitable hosts **Limit:** Climate is not diagnosis	**Action:** Maintain observation; avoid outbreak claim **Test:** Seek vector/host evidence	Proposed
State: Modeled suitability without circulation evidence **Pattern:** High suitability; negative/absent detection	Risk surface treated as infection	**Condition:** Model assumptions, surveillance coverage **Limit:** Suitability is not transmission	**Action:** Increase targeted surveillance **Test:** Test predicted high-risk areas	Evidence-bounded
State: Weak surveillance with no detections **Pattern:** Low effort plus zero detections	False reassurance	**Condition:** Sparse sampling, reporting gaps **Limit:** Non-detection is effort-dependent	**Action:** Lower confidence in apparent absence **Test:** Increase coverage or alternate stream	Evidence-bounded
State: Non-specific veterinary signal **Pattern:** Mortality/syndromic rise without etiology	False pathogen attribution	**Condition:** Other diseases, management, weather stress **Limit:** Syndrome lacks specificity	**Action:** Trigger diagnostics, not pathogen declaration **Test:** Resolve cause before escalation	Evidence-bounded

Veterinary and public-health response

Early warning has value only if surveillance outputs can be translated into proportionate action without overstating what the signal proves. Integrated vector-management evaluation demonstrates that response systems can be examined across management, implementation, and cross-sector integration rather than presumed effective because an intervention exists [28]. A broader One Health implementation review found recurrent weaknesses in environmental-sector participation and formal monitoring and evaluation [29].

Response also depends on movement and production context. Stochastic modeling of foot-and-mouth disease control at the Thailand-Myanmar border shows that quarantine outcomes depend on trade, compliance, vaccination, and epidemiological assumptions [30]. Bioeconomic modeling in Thailand similarly indicates that control strategies can perform differently across areas with different farm densities and production structures [31]. These studies are not evidence that the same measures control climate-sensitive vector-borne disease; they support the narrower principle that response must be context-specific.

Accordingly, the proposed architecture links each warning state to a menu of surveillance intensification, communication, vector investigation, movement review, targeted diagnostics, or coordinated response rather than prescribing a universal intervention package.

Transferability across regions

The architecture is transferable as a reasoning structure, not as fixed parameters. Climate and land-use projections for avian influenza in Bangladesh show that suitability depends on locally specific ecological and production variables [17]. Potential WNV-risk modeling in the United Kingdom similarly depends on local vector, host, demographic, and climate distributions [23]. Area-specific livestock-control modeling further demonstrates that structurally different production regions can favor different response choices [31].

Movement evidence reinforces this constraint. Livestock connectivity varies with production systems and seasonal resource availability [18], while wildlife-associated WNV spread is linked to region-specific landscape and migratory structures [19]. Transfer therefore requires recalibration of signal definitions, temporal lags, vector and host ecology, movement networks, surveillance sensitivity, and available response capacity.

A region adopting the architecture should not copy alert thresholds from another setting. Instead, it should preserve the architecture's boundaries—suitability versus transmission, exposure versus infection, signal versus decision—while rebuilding the empirical relationships that connect those states locally.

Validation priorities

The proposed architecture should be challenged prospectively rather than accepted because its components are biologically plausible. A thermal-biology-informed WNV reproduction number reproduced important seasonal timing in Europe but explained irregular interannual and geographic incidence patterns less completely, demonstrating the need to compare mechanistic indicators with observed surveillance outcomes [32]. Atmospheric modeling of windborne *Culicoides* introduction similarly shows that estimated risk is sensitive to biological and flight assumptions [33].

Movement components need equivalent testing. Temporal livestock-network analysis in Senegal indicates that candidate sentinel locations can change when temporal structure is represented rather than aggregated [34]. Combining different administrative movement-data sources in the United States materially altered network coverage and centrality, showing that surveillance targeting can be distorted by incomplete movement data [35].

Validation should test lead time, calibration, discrimination, missing-data robustness, regional transportability, and whether proposed warning states outperform simpler alternatives. Failure to outperform simpler surveillance should count as evidence against retaining unnecessary architectural complexity.

The final figure is needed to make explicit that validation must occur at several boundaries rather than as one global performance statistic.

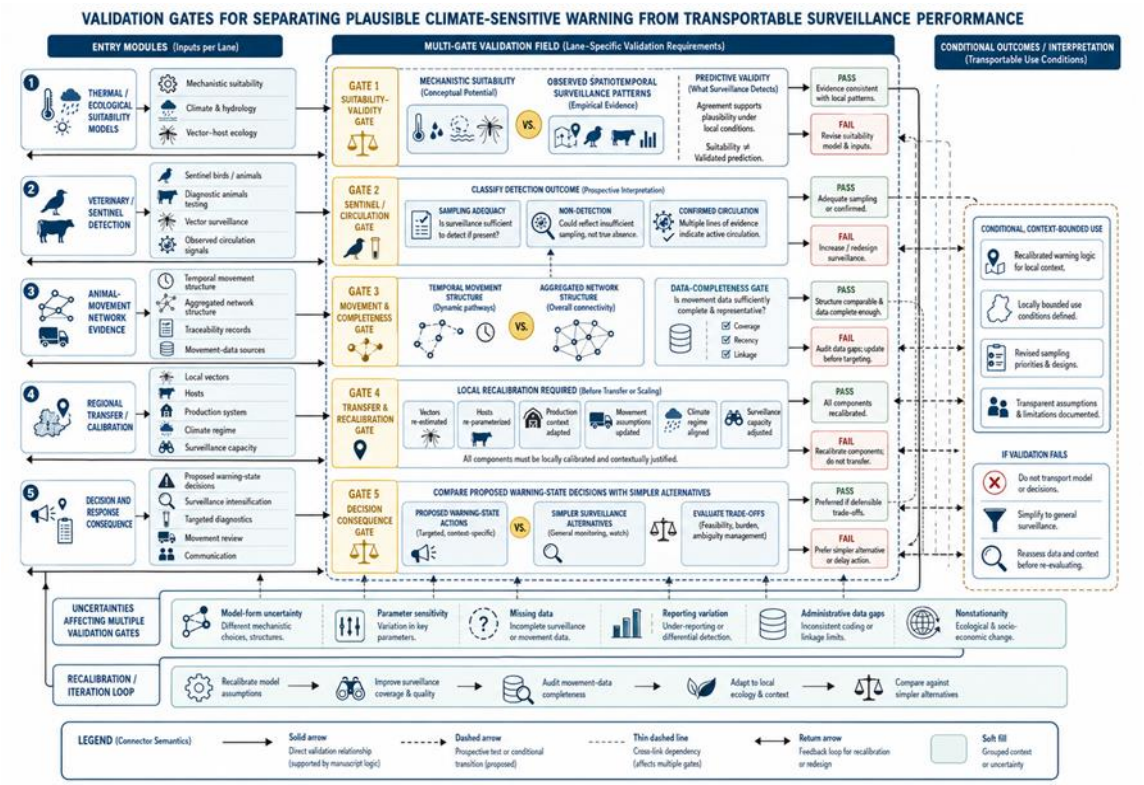

Figure 2. Validation gates for separating plausible climate-sensitive warning from transportable surveillance performance

Conclusion

Climate-sensitive animal disease surveillance should not wait for outbreak counts to rise before asking whether the underlying disease system has changed. Weather, hydrology, vectors, hosts, landscape, movement, animal exposure, surveillance intensity, and response capacity can change on different timescales and may produce concordant or conflicting signals. The architecture proposed here separates ecological suitability from exposure,

observed veterinary evidence, interpretation, and action so that uncertainty is preserved rather than averaged away. Its value is conceptual and testable, not established predictive performance. Regional calibration, adequate surveillance, complete movement information, explicit alternative explanations, and prospective comparison with simpler warning approaches are necessary before operational claims are justified. Earlier warning is useful only when it improves observation without turning ecological possibility into premature certainty.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

References

1. de Souza WM, Weaver SC. Effects of climate change and human activities on vector-borne diseases. Nat Rev Microbiol. 2024;22(8):476-91. doi:10.1038/s41579-024-01026-0
2. Erazo D, Grant L, Ghisbain G, Marini G, Colón-González FJ, Wint W, et al. Contribution of climate change to the spatial expansion of West Nile virus in Europe. Nat Commun. 2024;15(1):1196. doi:10.1038/s41467-024-45290-3
3. Haque S, Mengersen K, Barr I, Wang L, Yang W, Vardoulakis S, et al. Towards development of functional climate-driven early warning systems for climate-sensitive infectious diseases: Statistical models and recommendations. Environ Res. 2024;249:118568. doi:10.1016/j.envres.2024.118568
4. Olmeda P, Díaz-Regañón D, Villaescusa A, Amusategui I, García A, Herrero F, et al. Twenty-year evolution of Leishmania infantum infection in dogs in Valdeorras (Galicia, northwestern Spain): Implication of climatic factors and preventive measures. Parasit Vectors. 2024;17(1):281. doi:10.1186/s13071-024-06357-8
5. de Freitas Costa E, Streng K, Avelino de Souza Santos M, Counotte MJ. The effect of temperature on the boundary conditions of West Nile virus circulation in Europe. PLoS Negl Trop Dis. 2024;18(5):e0012162. doi:10.1371/journal.pntd.0012162
6. Vogels CBF, Göertz GP, Pijlman GP, Koenraadt CJM. Vector competence of northern and southern European Culex pipiens pipiens mosquitoes for West Nile virus across a gradient of temperatures. Med Vet Entomol. 2017;31(4):358-64. doi:10.1111/mve.12251
7. Vollans M, Day J, Cant S, Hood J, Kilpatrick AM, Kramer LD, et al. Modelling the temperature dependent extrinsic incubation period of West Nile virus using Bayesian time delay models. J Infect. 2024;89(6):106296. doi:10.1016/j.jinf.2024.106296
8. Kovach TJ, Kilpatrick AM. Irrigation increases and stabilizes mosquito populations and increases West Nile virus incidence. Sci Rep. 2024;14(1):19913. doi:10.1038/s41598-024-70592-3
9. Casades-Martí L, Peralbo-Moreno A, Delacour-Estrella S, Ruiz-Fons F. Environmental determinants of West Nile virus vector abundance at the wildlife-livestock interface. Med Vet Entomol. 2025;39(1):200-15. doi:10.1111/mve.12774
10. Smith DC, Schäfer SM, Golding N, Nunn MA, White SM, Callaghan A, et al. Vegetation structure drives mosquito community composition in UK's largest managed lowland wetland. Parasit Vectors. 2024;17(1):201. doi:10.1186/s13071-024-06280-y
11. Uiterwijk M, Jacobs F, de Boer K, Feddema AJ, Vloet RPM, Dik M, et al. Culicoides species involved in the BTV-3 epidemic, the Netherlands, 2023-2024. Med Vet Entomol. 2026;40(1):111-22. doi:10.1111/mve.70007
12. Wagner S, Mathis A, Schönenberger AC, Becker S, Schmidt-Chanasit J, Silaghi C, et al. Vector competence of field populations of the mosquito species Aedes japonicus japonicus and Culex pipiens from Switzerland for two West Nile virus strains. Med Vet Entomol. 2018;32(1):121-4. doi:10.1111/mve.12273
13. Brugman VA, Hernández-Triana LM, England ME, Medlock JM, Mertens PPC, Logan JG, et al. Blood-feeding patterns of native mosquitoes and insights into their potential role as pathogen vectors in the Thames estuary region of the United Kingdom. Parasit Vectors. 2017;10(1):163. doi:10.1186/s13071-017-2098-4

14. Şevik M. Epidemiology of bluetongue virus infection among small ruminants in Turkey: Seroprevalence and associated risk factors. Prev Vet Med. 2023;213:105871. doi:10.1016/j.prevetmed.2023.105871
15. King S, Nicholls M, Scales J, Gubbins S, Pearce-Kelly P, Saverimuttu S, et al. The efficacy of vector-proof accommodation for the protection of livestock against Culicoides biting midges. Parasit Vectors. 2025;18(1):108. doi:10.1186/s13071-025-06736-9
16. van den Brom R, Santman-Berends I, van der Heijden MG, Harders F, Engelsma M, van Gennip RGP, et al. Bluetongue virus serotype 12 in sheep and cattle in the Netherlands in 2024 - a BTV serotype reported in Europe for the first time. Vet Microbiol. 2025;301:110365. doi:10.1016/j.vetmic.2024.110365
17. Castonguay AC, Chowdhury S, Shanta IS, Schrijver B, Schrijver R, Hassan MM, et al. Projecting the impacts of climate and land-use change on avian influenza suitability in Bangladesh. One Health. 2025;21:101238. doi:10.1016/j.onehlt.2025.101238
18. Ekwem D, Enright J, Hopcraft JGC, Buza J, Shirima G, Shand M, et al. Local and wide-scale livestock movement networks inform disease control strategies in East Africa. Sci Rep. 2023;13(1):9666. doi:10.1038/s41598-023-35968-x
19. Lu L, Zhang F, Oude Munnink BB, Munger E, Sikkema RS, Pappa S, et al. West Nile virus spread in Europe: Phylogeographic pattern analysis and key drivers. PLoS Pathog. 2024;20(1):e1011880. doi:10.1371/journal.ppat.1011880
20. Streng K, Atama N, Chandler F, Blom R, van der Jeugd H, Schrama M, et al. Sentinel chicken surveillance reveals previously undetected circulation of West Nile virus in the Netherlands. Emerg Microbes Infect. 2024;13(1):2406278. doi:10.1080/22221751.2024.2406278
21. Tamba M, Bonilauri P, Galletti G, Casadei G, Santi A, Rossi A, et al. West Nile virus surveillance using sentinel birds: Results of eleven years of testing in corvids in a region of northern Italy. Front Vet Sci. 2024;11:1407271. doi:10.3389/fvets.2024.1407271
22. Braks M, Giglio G, Tomassone L, Sprong H, Leslie T. Making vector-borne disease surveillance work: New opportunities from the SDG perspectives. Front Vet Sci. 2019;6:232. doi:10.3389/fvets.2019.00232
23. Withers AJ, Croft S, Budgey R, Warren DA, Johnson N. Modelling vector and host distributions to inform potential disease risk: A case study of West Nile virus in the United Kingdom. Med Vet Entomol. 2025;39(4):842-62. doi:10.1111/mve.12825
24. Veldhuis A, Mars J, Stegeman A, van Schaik G. Changing surveillance objectives during the different phases of an emerging vector-borne disease outbreak: The Schmallenberg virus example. Prev Vet Med. 2019;166:21-7. doi:10.1016/j.prevetmed.2019.03.008
25. Welby S, van Schaik G, Veldhuis A, Brouwer-Middelesch H, Peroz C, Santman-Berends IM, et al. Effectiveness and cost efficiency of different surveillance components for proving freedom and early detection of disease: Bluetongue serotype 8 in cattle as case study for Belgium, France and the Netherlands. Transbound Emerg Dis. 2017;64(6):1771-81. doi:10.1111/tbed.12564
26. Vredenberg I, van Schaik G, van der Poel WHM, Stegeman A. Evaluation of a voluntary passive surveillance component in cattle through notification of excess mortality. Prev Vet Med. 2024;233:106334. doi:10.1016/j.prevetmed.2024.106334
27. Ansari S, Heitzig J, Moosavi MR. Optimizing testing strategies for early detection of disease outbreaks in animal trade networks via MCMC. Chaos. 2023;33(4):043144. doi:10.1063/5.0125434
28. Fite J, Baldet T, Ludwig A, Manguin S, Saegerman C, Simard F, et al. A one health approach for integrated vector management monitoring and evaluation. One Health. 2025;20:100954. doi:10.1016/j.onehlt.2024.100954
29. Milazzo A, Liu J, Multani P, Steele S, Hoon E, Chaber AL. One Health implementation: A systematic scoping review using the Quadripartite One Health Joint Plan of Action. One Health. 2025;20:101008. doi:10.1016/j.onehlt.2025.101008
30. Wongnak P, Yano T, Sekiguchi S, Chalvet-Monfray K, Premashthira S, Thanapongtharm W, et al. A stochastic modeling study of quarantine strategies against foot-and-mouth disease risks through cattle trades across the Thailand-Myanmar border. Prev Vet Med. 2024;230:106282. doi:10.1016/j.prevetmed.2024.106282
31. Chanchaidechachai T, Fischer EAJ, Saatkamp HW, de Jong MCM, Hogeveen H. One-size measures do not fit all areas: Evaluation of area-specific control of foot and mouth disease in Thailand using bioeconomic modelling. Prev Vet Med. 2024;233:106359. doi:10.1016/j.prevetmed.2024.106359

32. Heidecke J, Kriit HK, Fransson P, Wallin J, Rocklöv J. Evaluating the ability of a thermal biology-informed reproduction number to explain patterns of West Nile incidence in Europe. One Health. 2026;22:101469. doi:10.1016/j.onehlt.2026.101469
33. Bibard A, Martinetti D, Giraud A, Picado A, Chalvet-Monfray K, Porphyre T. Quantitative risk assessment for the introduction of bluetongue virus into mainland Europe by long-distance wind dispersal of Culicoides spp.: A case study from Sardinia. Risk Anal. 2025;45(1):108-27. doi:10.1111/risa.14345
34. Ciss M, Giacomini A, Diouf MN, Delabouglise A, Mesdour A, Garcia Garcia K, et al. Description of the cattle and small ruminants trade network in Senegal and implication for the surveillance of animal diseases. Transbound Emerg Dis. 2023;2023:1880493. doi:10.1155/2023/1880493
35. Sequeira SC, Locke SR, Habing G, Arruda AG. Combining different sources of movement data to strengthen traceability and disease surveillance. Prev Vet Med. 2025;237:106442. doi:10.1016/j.prevetmed.2025.106442