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Genomic Surveillance Should Change Veterinary Action Rather Than Merely Describe Pathogen Diversity: A Decision Architecture for Variant Detection, Transmission Inference, Resistance Signals, Risk Escalation, Targeted Control, and Data Governance

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ABSTRACT

Genomic surveillance can reveal pathogen diversity at a resolution that conventional typing cannot provide, yet sequence resolution alone does not determine what veterinary services should investigate, escalate, control, or communicate. This article develops an original non-empirical decision architecture for converting genomic observations into bounded veterinary surveillance evidence. The analysis separates sampling design and representativeness from sequencing and assembly quality; distinguishes variant or lineage detection from transmission inference; and treats antimicrobial-resistance, virulence, and host-adaptation findings as signal classes whose action relevance depends on analytical validity, phenotype, epidemiological context, and consequences of error. The proposed architecture positions genomic quality control as an upstream interpretation boundary and treats actionability as a revisable confidence state rather than an intrinsic property of a sequence. It further requires contextual reconciliation of genomic findings with host, place, time, movement, exposure, clinical or production phenotype, and surveillance-system constraints before risk escalation or targeted response. This structure is intended to make uncertainty operational: incomplete sampling, low genetic diversity, recombination, unusual mutation dynamics, genotype–phenotype discordance, pipeline changes, and missing metadata can prevent apparently precise genomic outputs from supporting precise decisions. The architecture does not provide universal distance thresholds, validated escalation rules, or proof that genomics improves veterinary outcomes. Its principal contribution is a testable way to separate description, evidence interpretation, decision confidence, and response selection so that genomic surveillance can be evaluated by whether it supports proportionate, traceable, and revisable action.

Keywords: Veterinary genomic surveillance, Genomic epidemiology, Antimicrobial resistance, Phylogenetics, Outbreak investigation, Data governance

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Introduction

Veterinary genomic surveillance is most useful when sequence evidence changes a justified investigation or control decision rather than merely increasing taxonomic resolution. During the H5N1 outbreak in dairy cattle,

genomic findings became more informative when interpreted with animal, farm, movement, and cross-species evidence, supporting plausible spread reconstruction while leaving unsampled transmission events possible [1].
High-resolution genomic similarity is therefore not inherently actionable. Surveillance design, metadata quality, source coverage, and interpretive rules influence whether related genomes support outbreak recognition, source attribution, or only descriptive clustering; fixed distance rules can mislead when those conditions are ignored [2].
Routine veterinary use also exposes pathogen-specific limits. Whole-genome sequencing refined *Mycobacterium bovis* epidemiology when combined with cattle movement and location data, but low diversity and incomplete sampling constrained fine-scale transmission reconstruction [3]. The relevant question is not whether genomics is informative, but what degree of inference it can defensibly support.
Antimicrobial-resistance surveillance broadens the problem further because genes, mobile elements, phenotypes, hosts, sectors, and data infrastructures interact across One Health systems [4]. This article therefore proposes a decision architecture that separates analytical validity, genomic signal detection, epidemiological interpretation, confidence, escalation, targeted response, governance, and reassessment without assuming that any genomic signal is self-executing.

Genomic description versus actionable surveillance

A genome can be accurately sequenced and still remain decision-poor. Across veterinary outbreak investigation, foodborne surveillance, and routine bovine tuberculosis epidemiology, action-relevant interpretation depends on linking genomic observations to sampling design, metadata, epidemiological plausibility, and the question being decided [1–3]. We therefore distinguish genomic description from actionable surveillance: description records diversity or relatedness, whereas actionability is a proposed property of evidence-in-context that can justify a proportionate next step.
This distinction is deliberately consequence-sensitive. A novel lineage may justify intensified observation before it justifies movement restrictions; a resistance determinant may justify confirmatory testing before it supports a population-level inference. One Health AMR experience shows why genomic findings require representative sampling, standardized analysis, and interoperable contextual information [4]. Accordingly, the architecture proposed here treats actionability as graded and revisable confidence, not a binary label attached to a sequence. These confidence states remain conceptual until evaluated prospectively across pathogens, species, production systems, and decision consequences.

Sampling architecture

Genomic surveillance begins before sequencing. The sampling unit—animal, herd, farm, diagnostic submission, wildlife location, food interface, or environmental source—determines what population process can be observed. In modeled regional swine surveillance, farm-selection strategy changed early-detection performance, demonstrating that sampling design can alter surveillance sensitivity independently of sequencing resolution [5]. The transferable principle is design dependence, not superiority of one universal selection method.
A decision-oriented sampling architecture should therefore specify the surveillance objective before choosing where, when, and from whom to sample. Early detection may favor sentinel or risk-enriched coverage, whereas prevalence estimation, lineage tracking, source attribution, and transmission inference require different denominators and sampling frames. Phylogeny-aware subsampling methods can explicitly preserve genomic diversity and have been demonstrated with swine influenza surveillance data [6]. However, phylogenetic coverage is only one dimension of representation: a sequence set can span a tree while systematically missing farms, regions, seasons, host classes, or epidemiological interfaces. The proposed architecture consequently records both the sampling mechanism and the decision it is intended to support, allowing downstream confidence to be reduced when the sample-generating process is misaligned with that decision.

Sample representativeness

Representativeness should be judged against the intended decision rather than by sequence count alone. Surveillance designed to detect an unusual pathogen can tolerate enrichment that would bias prevalence estimation; source attribution requires source populations that are sufficiently sampled to make absence or similarity interpretable. Because farm-selection methods produce different detection behavior under different surveillance assumptions, representativeness is proposed here as objective-specific rather than a single dataset property [5].

Four dimensions should remain analytically separate: host or production-system coverage, spatial coverage, temporal coverage, and genomic or phylogenetic coverage. Phylogeny-aware selection can improve representation of sequence diversity [6]. It cannot, by itself, repair epidemiological undercoverage. Reviews of WGS surveillance similarly identify incomplete sampling and contextual-data limitations as constraints on inference [2]. Thus, adding more genomes from the same accessible source cannot compensate for systematic exclusion of another host, region, season, or interface. In the proposed architecture, missing strata are carried forward as uncertainty rather than being erased by dataset size, and claims are restricted to the sampled surveillance frame.

Sequencing quality

Sequencing quality is not a generic technical precondition; it is part of evidential validity. Multilaboratory AMR proficiency testing showed that quality distributions and problematic outputs can vary by species and laboratory workflow, supporting organism- and purpose-aware quality assessment rather than a universal pass threshold [7]. Contamination, coverage irregularity, read quality, and genome biology can have different consequences depending on whether the downstream task is species identification, resistance detection, assembly, or fine-scale clustering.

Platform choice is likewise conditional on intended inference. Oxford Nanopore R10 sequencing produced results comparable with Illumina for tested SNP-based bacterial outbreak applications under validated workflows, but that finding does not make platforms, organisms, or pipelines interchangeable [8]. A surveillance system should therefore record platform, chemistry, library method, relevant quality metrics, and intended use, while treating substantial workflow changes as analytical events requiring verification. High throughput cannot compensate for unrecognized quality failure, and conservative quality control should not automatically discard biologically unusual genomes without investigating whether the apparent anomaly is technical or real.

Assembly and genomic quality control

Assembly converts reads into the genomic representation on which many downstream labels depend. In this architecture, assembly and genomic quality control form an explicit interpretation boundary: genomes that fail purpose-specific checks should enter a hold, reanalysis, or resequencing state rather than flowing silently into lineage, resistance, virulence, or transmission decisions. Species-specific proficiency-testing evidence supports this boundary because contamination and assembly-quality outliers can be detected only when expectations are appropriate to the organism and use case [7].

The boundary must also be version-aware. Demonstrated cross-platform concordance is empirical and use-specific, so a change in sequencing platform, assembler, mapping reference, allele scheme, database, or cluster pipeline should not be assumed to preserve decision equivalence [8]. The proposed rule is not that every change invalidates previous results, but that material analytical changes trigger documented comparison before old and new outputs are pooled for consequential inference. Exceptions may be justified when a downstream task tolerates a known imperfection, provided the exception, rationale, and resulting uncertainty remain visible.

Table 1 presents purpose-specific genome quality gates and downstream analyses permitted at each gate.

Table 1. Purpose-specific genome quality gates and downstream analyses permitted at each gate

Genome QC gate	Technical condition	Deployment constraint	Downstream use allowed	Failure or version check
Decision-eligible	**Assembly:** Assembly supports intended analysis **QC:** Purpose-specific checks acceptable	Organism and workflow within validated scope	**Evidence:** Internally coherent reads, assembly, identity, and contamination profile **Use:** Proceed to signal interpretation	**Unresolved:** Does not establish epidemiological meaning **Check:** Reassess if context or analytic version changes
Conditional-use	**Assembly:** Local imperfection with preserved task suitability **QC:** Exception documented	Urgent or limited-sample setting	**Evidence:** Limitation identifiable and bounded **Use:** Permit restricted downstream use	**Unresolved:** Confidence reduced for affected inference **Check:** Confirm with alternate analysis or new sample

Hold/reanalyse	**Assembly:** Assembly or QC problem may alter result **QC:** Failure source unresolved	Consequential decision depends on affected feature	**Evidence:** Contamination, missing target, unstable assembly, or discordant output **Use:** Do not escalate from genome alone	**Unresolved:** Technical versus biological explanation unresolved **Check:** Reprocess, resequence, or recollect
Version-change verification	**Assembly:** Output generated after material analytic change **QC:** Prior equivalence not established	Longitudinal or cross-laboratory comparison required	**Evidence:** Cluster, allele, or variant assignment may shift **Use:** Avoid unqualified pooling	**Unresolved:** Difference may reflect method rather than biology **Check:** Perform documented concordance check

Variant and lineage detection

Variant and lineage labels compress genomic change into surveillance categories, but the category is not the hazard. Longitudinal PRRSV-2 surveillance in China demonstrated lineage turnover, recombination, and temporal evolutionary change across a major production disruption, while not supporting a simple inference that every lineage shift represented increased clinical risk [9]. Apparent emergence can also reflect altered sampling intensity, geography, host population structure, or recombination.

Dynamic tools can update phylogenetic and geographic views as sequences accumulate, making near-real-time lineage tracking technically feasible [10]. Their output remains conditional on the samples and metadata entering the system. A surveillance dashboard can therefore identify a change worth examining without validating the action that should follow.

Low-diversity pathogens create a different hard case. Complete-genome analysis of African swine fever virus genotype II helped reconstruct broad dispersal patterns in Europe, yet limited genetic variability left uncertainty in ancestral locations and pathways [11]. The proposed architecture consequently separates “lineage detected” from “hazard inferred” and retains an explicit low-resolution state when genomic change is insufficient to resolve origin, direction, or fine-scale transmission.

Phylogenetic and transmission inference

Phylogenies organize genetic relatedness and can constrain plausible transmission histories, but they do not observe transmission events. In routine *M. bovis* epidemiology, genomic information gained interpretive value when combined with cattle movements and locations, while slow evolution and incomplete sampling left multiple transmission explanations compatible with similar genomes [3]. The architecture therefore separates confidence in relatedness from confidence in a common source, transmission chain, and direct transmission event.

That separation is especially important for slowly evolving pathogens. ASFV phylogeography can narrow dispersal histories, yet low diversity and uneven sampling preserve uncertainty about ancestral locations and unsampled intermediates [11]. Directional arrows in a reconstruction should consequently represent inference rather than observation unless exposure evidence independently resolves direction.

Genomic distance should also not substitute for epidemiological plausibility. WGS surveillance experience shows that similar genomes can reflect recent transmission, persistent clones, a common source, or unsampled chains depending on the organism and setting [2]. Transmission claims should therefore be calibrated to evolutionary rate, within-host diversity, recombination, sampling density, temporal spacing, and contact evidence rather than a context-free distance rule.

Antimicrobial-resistance signals

AMR genomics can identify high-concern determinants before their population significance is known. A *bla*NDM-1-positive *Proteus mirabilis* isolate from a companion dog combined a clinically relevant resistance genotype with phenotypic susceptibility testing, supporting confirmatory and epidemiological attention at the isolate level [12]. It did not establish population prevalence, transmission source, or treatment-outcome probability.

At surveillance scale, longitudinal genomics can distinguish persistent from changing resistance patterns. UK poultry surveillance of extended-spectrum cephalosporin-resistant *Escherichia coli* documented temporal changes in resistance determinants and genomic lineages, showing the value of repeated sampling while leaving production

and sampling changes as alternative explanations for observed trends [13]. Trend detection should therefore trigger denominator and context review before causal attribution.

Cross-sector relatedness adds a One Health signal without resolving direction. Large-scale sequencing of animal clinical *Salmonella* isolates identified resistance mechanisms and close genomic relationships to human clinical strains, supporting prioritization of cross-sector investigation while remaining compatible with shared ancestry, common sources, or unsampled routes [14]. The proposed architecture accordingly keeps four questions separate: what resistance determinant is detected, whether phenotype corroborates it, how the signal is distributed across animal populations, and whether epidemiological evidence supports a transmission pathway.

Virulence and host-adaptation signals

Host association is not synonymous with host adaptation. Population-genomic structure can arise through selection, recombination, ecological filtering, drift, founder effects, or repeated exposure, and these alternatives must be considered before sequence association is assigned adaptive function [15]. The proposed architecture therefore treats host-associated genomic structure as hypothesis-generating evidence rather than a direct escalation trigger.

Large livestock datasets can strengthen an association without resolving its mechanism. In bovine and swine enterotoxigenic *Escherichia coli*, host species was associated with differences in genotype, antimicrobial-resistance, and virulence profiles, but the observational design leaves lineage, era, geography, and production ecology as plausible contributors [16]. Association should therefore increase surveillance attention without being relabelled as demonstrated adaptation.

Functional evidence can raise confidence for a specific mutation. Experimental work on cattle-derived H5N1 PB2 substitutions linked selected mutations to increased polymerase activity and, for a tested mutation, increased pathogenicity in a mouse model [17]. Such evidence supports a stronger mechanistic interpretation than sequence association alone, but it does not establish field transmission, cattle-level severity, or population risk. **Table 2** presents genomic-to-field evidence stack for host adaptation and virulence signals.

Table 2. Genomic-to-field evidence stack for host adaptation and virulence signals

Evidence layer	Host-associated pattern	Convergent/recurrent signal	Functionally supported signal	Field-concordant signal
Biological support	Genomic feature differs by host or lineage	Similar candidate change appears independently	Candidate alters a measured biological function	Genomic signal aligns with observed phenotype and epidemiology
Population-structure or model dependency	Sampling frame and population structure	Evolutionary background matters	Assay, host model, and genetic background	Host, production system, and exposure context
Observed genomic pattern	Repeated association in surveillance data	Recurrence across relevant genomes	Experimental functional effect	Reproducible genomic–phenotypic concordance
Response authorized	Investigate, do not infer mechanism	Raise biological-priority status	Increase confidence in biological relevance	Consider escalation within defined scope
Threat to inference	Confounding or shared ancestry remains plausible	Recurrence is not functional proof	Laboratory effect may not transfer to field risk	Association can still be non-causal
Evidence needed to advance	Replicate across settings and lineages	Seek functional and epidemiological corroboration	Test in relevant host and epidemiological context	External prospective validation

Integration with epidemiological data

Genomic evidence becomes more decision-relevant when reconciled with who was affected, where and when detection occurred, how animals or products moved, which exposures were plausible, and what clinical or production phenotype accompanied the signal. In dairy-cattle H5N1 investigation, movement and multispecies context materially shaped interpretation of genomic relationships [1]. Missing contact or movement information should therefore remain visible as uncertainty rather than being implicitly treated as absence of exposure.

The relative informational contribution of context also varies by pathogen. For slowly evolving *M. bovis*, movement and location data can discriminate among hypotheses that genomes alone cannot separate [3]. The

architecture accordingly proposes qualitative evidence weighting according to evolutionary rate and surveillance context, without assigning universal numerical weights.

Cross-sector similarity requires the same restraint. Closely related animal and human *Salmonella* genomes can prioritize epidemiological reconciliation but do not independently establish route or direction [14]. Integration therefore precedes source attribution: genomic relatedness, time, place, movement/contact, host, phenotype, exposure, and metadata completeness are jointly examined, and discordance can lower confidence as legitimately as concordance can raise it.

Evidence thresholds for action

Genomic thresholds should be calibrated to the biological and decision context rather than treated as universal constants. Modeling of common-source bacterial outbreaks showed that useful genomic-distance thresholds vary with mutation rate and outbreak timescale, with reduced specificity expected for slowly evolving organisms [18]. A distance that is informative in one organism, timescale, or question may therefore be inappropriate in another.

Biological exceptions further weaken rigid threshold logic. A DNA-repair-deficient *Listeria monocytogenes* strain accumulated variation rapidly enough to challenge conventional cgMLST cluster expectations, illustrating how unusual evolutionary dynamics can separate epidemiologically linked isolates beyond an otherwise useful rule [19]. Such cases justify an exception and reassessment pathway, not abandonment of genomic clustering.

One Health inference introduces another boundary: cross-compartment linkage depends on which source populations were sampled and on analytical definitions. Australian multisector *E. coli* genomics demonstrated sensitivity of inferred linkage to parameter choices and source coverage [20]. The proposed decision rule is therefore an evidence bundle: analytical validity, representativeness, genomic relatedness, biological signal, epidemiological plausibility, phenotype, and consequence of error are considered together before action confidence is assigned.

A proposed genomic-surveillance decision architecture

The proposed architecture separates four non-equivalent questions: is the genomic result analytically valid; what biological or epidemiological signal does it support; how confident is the surveillance interpretation after contextual reconciliation; and what proportionate response follows? Purpose-specific quality control forms a noncompensatory upstream boundary because a technically unreliable genome should not gain authority merely through alarming annotation [7].

No single genomic distance should control veterinary action across pathogens, evolutionary states, or One Health compartments. Threshold modeling, hypermutation as a hard case, and cross-source parameter sensitivity each demonstrate why context can change the meaning of the same apparent genomic proximity [18–20]. The architecture instead uses proposed revisable decision states: descriptive signal, investigation signal, escalation-supporting evidence, and action-supporting evidence. These are not validated scores or mandatory tiers; they identify how much interpretive work separates detection from intervention.

The architecture is recursive. New samples, phenotypic evidence, movement data, contradictory exposures, or an analytical-version change may strengthen or weaken the interpretation [2]. **Figure 1** compresses this logic while retaining quality, context, uncertainty, and reassessment as visible boundaries rather than hiding them in a linear pipeline.

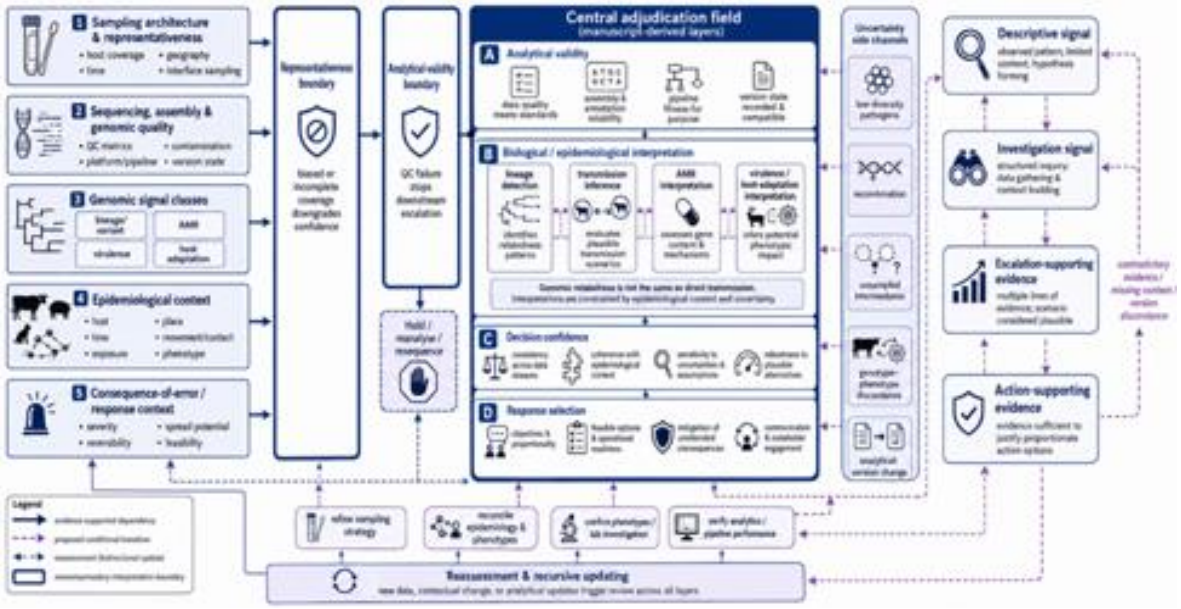

Figure 1. Decision boundaries that prevent veterinary genomic signals from becoming self-executing actions

Risk escalation

Risk escalation should reflect consequences as well as genomic novelty. In an affected Ohio dairy herd, H5N1 infection was associated with substantial clinical disease and production loss, demonstrating that observed animal-health and production burden can materially change the decision context [21]. Those herd-specific observations do not define a universal severity threshold, but they support inclusion of phenotype and burden in escalation decisions.

Model-derived evidence can also inform escalation when its conditional nature remains explicit. A movement-aware model of H5N1 spread among US dairy herds produced projections that varied with assumptions about detection, movement, and interventions [22]. Such outputs can prioritize scenarios or investigations, but they should not be represented as validated forecasts.

Escalation should ordinarily lead to proportionate epidemiological follow-up rather than maximal control solely because a genomic cluster exists. One Health foodborne surveillance experience links WGS with epidemiological investigation, traceback, and source testing [23]. **Table 3** presents non-genomic corroboration required as genomic novelty moves from observation to action.

Table 3. Non-genomic corroboration required as genomic novelty moves from observation to action

Escalation rung	Genomic input	Non-genomic context	Action window	Evidence for upgrade or de-escalation
Observe	Valid novel or changing signal	**Clinical/production meaning:** Genomic change without corroborated consequence **System:** Routine surveillance	Increase attention and contextual review	**Residual uncertainty:** Hazard significance unresolved **Next evidence:** Seek additional samples and metadata
Investigate	Cluster, AMR, adaptation, or transmission hypothesis	**Clinical/production meaning:** Signal plus plausible epidemiological relevance **System:** Connected animals, farms, sectors, or exposures	Target case finding and reconciliation	**Residual uncertainty:** Route or source still uncertain **Next evidence:** Add epidemiology, phenotype, and comparator sampling
Conditionally escalate	Valid genomics plus phenotype, movement, or model support	**Clinical/production meaning:** Multiple evidence domains concordant or consequences substantial **System:** High vulnerability or rapid spread potential	Intensify proportionate surveillance/control consideration	**Residual uncertainty:** Model and sampling uncertainty remain **Next evidence:** Reassess rapidly against observed data
Action-supporting	Contextually corroborated	**Clinical/production meaning:** Evidence sufficiently coherent	Select pathway-matched response	**Residual uncertainty:** Effectiveness remains intervention-specific

Escalation rung	Genomic input	Non-genomic context	Action window	Evidence for upgrade or de-escalation
	signal with bounded alternatives	for a defined decision context **System:** Consequences of delay exceed residual uncertainty		**Next evidence:** Monitor response and downgrade if evidence changes

Targeted control responses

Control should target the best-supported pathway, not the most visually prominent genomic result. During dairy-cattle H5N1 investigation, genomic evidence gained meaning through animal, farm, movement, and cross-species context [1]. The proposed response logic therefore links candidate actions to the transmission or exposure hypothesis they are intended to interrupt and keeps alternatives visible when several pathways remain plausible.

Observed disease or production burden may justify intensified case finding, testing, segregation, biosecurity review, or other context-appropriate measures while genomic uncertainty persists, but evidence from one affected herd cannot establish universal effectiveness of any intervention [21]. Response intensity should remain proportionate to consequence, plausibility, reversibility, and feasibility.

Model-derived movement risk can help prioritize farms, routes, or surveillance resources, yet modeled interventions remain conditional on assumptions and ascertainment [22]. Targeted control is consequently treated as a revisable state: field observations, new sequences, negative investigations, and changing epidemiology feed back into the original hypothesis. A control decision can be escalated, narrowed, redirected, or stopped when the evidential basis changes.

Data sharing and interoperability

Action-oriented surveillance requires information to move across laboratories and jurisdictions without erasing provenance or local responsibility. Australian national pathogen-genomics implementation illustrates the need for coordinated laboratory methods, shared infrastructure, workforce capability, governance, and continuing evaluation [24]. These system principles are informative for veterinary networks but require adaptation to animal-health mandates, production structures, and jurisdictional constraints.

A shared analysis environment can support distributed participation rather than requiring every laboratory to operate identically. AusTrakka demonstrated how multiple jurisdictions could contribute to a common genomic surveillance environment using standardized analytical infrastructure and controlled data exchange [25]. Emergency implementation should not be assumed to predict routine veterinary costs or capacity.

Genome files alone are insufficient for interoperability. DataHarmonizer demonstrates schema-driven standardization and validation of contextual pathogen-genomics metadata [26]. Veterinary interoperability should therefore include controlled terminology for host, sample source, location granularity, collection time, epidemiological linkage, analytical method, and data version. Schema conformance improves semantic consistency but cannot guarantee that submitted metadata are complete or factually correct.

Governance, privacy, and versioning

Interoperability should be governed as a lifecycle rather than treated as a file-transfer problem. National program architecture, shared analytic infrastructure, and standardized contextual metadata collectively support the need to coordinate access, semantics, analysis, and stewardship across a surveillance network [24–26]. The governance model proposed here keeps data access, analytic authority, interpretation authority, and response authority distinguishable because they may reside in different veterinary, laboratory, production, or public-health institutions.

Every decision-relevant output should preserve sample provenance, contextual metadata state, pipeline and database version, and the interpretation attached at that time [26]. This is important because a later database update, cluster-definition change, corrected metadata field, or newly available comparator genome may alter the meaning of an earlier result.

Privacy and commercial sensitivity cannot be solved by de-identification language alone. The architecture therefore proposes purpose-limited access, role-based visibility, auditable change histories, and rules for cross-sector release without claiming one governance design is ethically or legally sufficient across jurisdictions. Versioning is treated as scientific provenance: a consequential genomic conclusion should remain reproducible as the output of a specified data and analytical state.

Validation priorities

Analytical validity must be maintained over the workflow lifecycle. Standards-based pathogen-genomics implementation emphasizes validation, quality management, change control, and reverification when methods materially change [27]. Veterinary systems should test how these principles map to different pathogens, laboratories, regulatory settings, and consequences of error rather than equating accreditation with surveillance utility.

External validation must also address computational reproducibility. A multicountry, intersectoral comparison showed that genomic pipelines can differ in cluster assignments across foodborne pathogens, making inter-pipeline congruence a practical requirement for networks that pool or compare outputs [28]. Disagreement should be characterized by its decision consequence rather than assuming that one pipeline is automatically correct.

Prospective evaluation should then extend beyond analytical accuracy. Implementation literature identifies workforce, funding, workflow integration, infrastructure, and decision-support constraints that can prevent technically capable genomics from becoming routine action [29]. Veterinary validation should therefore examine sampling coverage, turnaround, interpretive concordance, false escalation and missed escalation, workflow burden, decision change, unintended consequences, and transfer across species and production systems. **Figure 2** organizes these validation targets as interacting boundaries rather than a checklist of isolated technical tests.

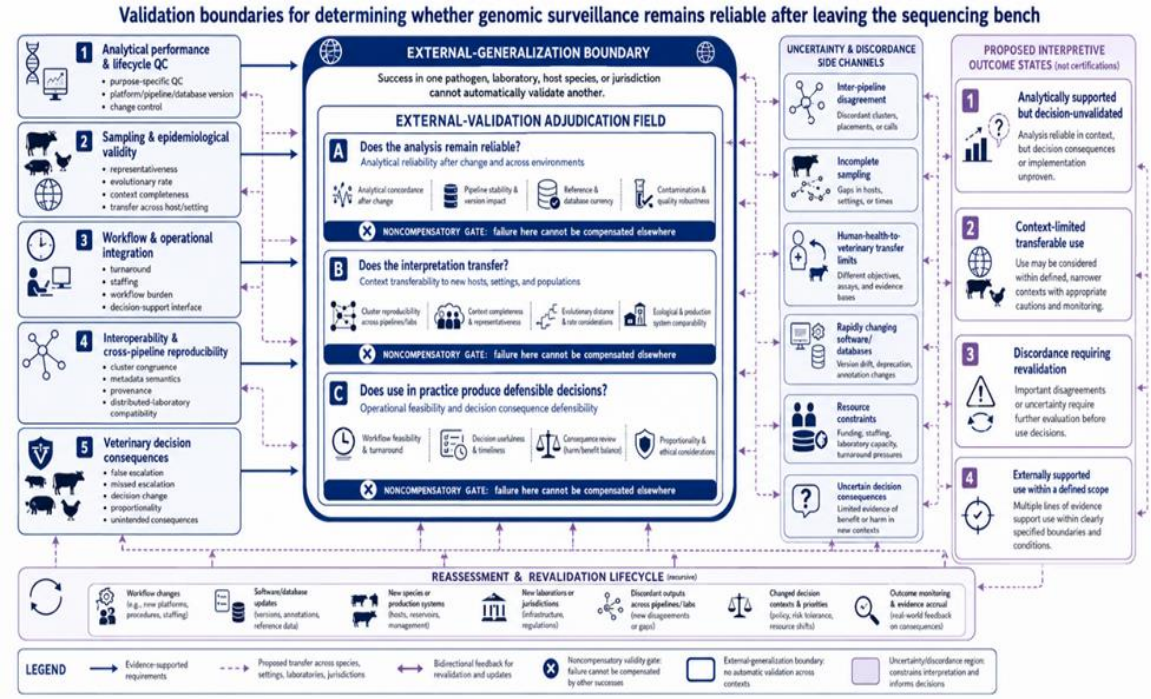

Figure 2. Validation boundaries for determining whether genomic surveillance remains reliable after leaving the sequencing bench

Conclusion

Veterinary genomic surveillance should be judged by decisions it can defensibly support, not by sequence resolution alone. The architecture proposed here separates sampling, analytical validity, genomic signals, epidemiological interpretation, confidence, escalation, response, governance, and reassessment so that uncertainty remains visible rather than being compressed into a lineage label or genomic distance. Its boundaries are deliberate: relatedness is not transmission, host association is not adaptation, resistance genotype is not treatment outcome, model output is not forecast certainty, and technical validation is not implementation effectiveness. The architecture remains unvalidated as an integrated decision system. Its value therefore depends on prospective testing across pathogens, species, production settings, laboratories, and jurisdictions, including whether it improves traceability, calibration, timeliness, and proportionality without producing avoidable false escalation or false reassurance.

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