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Disease Control Fails When Herd-Level Risk Is Treated as an Individual-Animal Problem: A Population-Based Veterinary Architecture for Integrating Surveillance, Biosecurity, Vaccination, Treatment, Environmental Control, and Reassessment

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ABSTRACT

Veterinary disease control often begins with the affected animal, yet the determinants of persistence, spread, and recurrence frequently reside at herd and population levels. This article develops an original non-empirical population-health architecture for integrating surveillance, biosecurity, vaccination, treatment, environmental control, and reassessment without treating any component as sufficient in isolation. The analysis distinguishes pathogen introduction from within-herd amplification, individual susceptibility from realized population protection, clinical signals from laboratory confirmation, environmental contamination from infectious transmission, and individual treatment outcomes from herd-level control. It further treats animal movement, contact structure, implementation capacity, and temporal change as conditions that can redirect control decisions. The principal contribution is a proposed architecture in which disease-control actions are selected against a changing configuration of exposure pressure, transmission opportunity, susceptibility, detection evidence, environmental persistence, and implementation constraints, followed by explicit reassessment rather than closure after intervention. The framework is intended as an analytical structure for organizing evidence and decisions, not as a validated prediction tool or universally applicable protocol. Evidence across livestock systems indicates that network structure, diagnostic limitations, vaccination context, treatment strategy, biosecurity implementation, and surveillance participation can all modify interpretation, but their effects remain disease- and setting-dependent. The architecture therefore emphasizes conditional escalation, evidence boundaries, and validation across pathogens, production systems, and operational contexts.

Keywords: Population health, Herd disease control, Veterinary surveillance, Biosecurity, Vaccination, Reassessment

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Introduction

Disease control becomes analytically incomplete when the clinically affected animal is treated as the primary unit of both diagnosis and prevention. Population outcomes depend not only on whether an infected animal is detected or treated, but also on how farmers implement controls, how animals and holdings are connected, how signals are recognized, and whether interventions persist over time. Empirically informed modelling has shown that heterogeneity in farmer control behaviour can change predicted livestock-disease outcomes [1]. Contact-network structure can likewise alter transmission dynamics even when the pathogen and host population are otherwise held constant [2]. Passive surveillance adds another layer because recognition, willingness to report, trust, and system design can determine whether a disease signal becomes visible [3]. Longitudinal paratuberculosis control further

illustrates that herd improvement can require repeated testing, management changes, and biosecurity over years rather than a single animal-level action [4].

This article therefore treats herd disease control as a multilevel problem. It separates introduction, amplification, susceptibility, movement, environmental persistence, surveillance, intervention, and reassessment while preserving the distinct evidential meaning of each. The resulting architecture is proposed as a decision-organizing synthesis, not as a validated causal model or universal control protocol.

Individual disease management versus population risk

Individual disease management answers questions about a particular animal: whether disease is present, whether treatment is indicated, and whether the animal improves. Population risk asks a different question: what conditions allow infection to enter, circulate, persist, remain undetected, or recur across the herd. These levels overlap but are not interchangeable. Heterogeneity in farmer control behaviour can alter population-level model outcomes even when disease biology is unchanged [1]. Likewise, a multiyear paratuberculosis programme relied on repeated herd-level testing and control measures rather than only management of detected animals [4].

The distinction matters because an animal can recover while the herd remains vulnerable to reintroduction, environmental persistence, susceptible replacements, incomplete detection, or continued contact pathways. Conversely, a herd can lower population risk while individual cases still require clinical care. The synthesis proposed here therefore treats individual treatment as one function nested within a broader herd-risk system. Population risk is not reduced to prevalence or case count; it is interpreted as the evolving configuration of introduction pressure, transmission opportunity, host susceptibility, environmental conditions, detection capacity, and control implementation.

Pathogen introduction pathways

Before within-herd transmission can occur, a pathogen must cross an epidemiological boundary. In swine breeding herds, potentially relevant contacts include animals, people, vehicles, and supplies, and their frequencies do not map simply onto a single biosecurity score [5]. In Dutch dairy herds, incident *Salmonella* serogroup status was associated with specific management and exposure factors, supporting the view that introduction risk is conditional rather than uniform across farms [6]. Animal-transfer networks add another route because movements connect holdings that would otherwise be epidemiologically separated [7].

The proposed architecture therefore treats introduction pressure as a set of heterogeneous interfaces rather than a single external-risk variable. The practical implication is not that every contact is hazardous, but that route identity, frequency, timing, and the receiving herd's susceptibility can alter the meaning of an exposure. Introduction should also remain analytically separate from amplification: a factor that increases the chance of entry may not determine subsequent within-herd spread. Evidence from different species and production systems cannot justify universal pathway weights, so any future quantitative implementation would require disease-specific measurement and external validation.

Within-herd transmission

Once a pathogen enters a herd, the relevant question shifts from entry to amplification. Transmission opportunity depends on how animals, pens, management groups, workers, equipment, and husbandry routines connect susceptible and infectious units. Network modelling demonstrates that changing contact structure can alter epidemic behaviour [2]. Longitudinal herd-control evidence likewise suggests that repeated testing and management changes may be required to reduce persistence after infection is established [4]. Animal-transfer network analyses further show that connectivity influences how infection can be redistributed between epidemiological units [7].

This section therefore distinguishes pathogen presence from transmission potential. Detection of one infected animal does not reveal whether the herd is in a self-limiting, expanding, persistent, or repeatedly re-seeded state. The proposed architecture interprets within-herd transmission as a dynamic interaction between pathogen biology and contact opportunity, constrained by the available evidence for the specific disease and production system. It does not assign a universal reproduction threshold, nor does it assume homogeneous mixing. Where contact data are incomplete, transmission-state judgments should remain provisional and be revised as surveillance, movement, or management information changes.

Host susceptibility and population immunity

Population protection cannot be inferred from a binary record of vaccination. In naturally infected Danish finisher herds, vaccination against *Lawsonia intracellularis* altered selected production, clinical, treatment, and shedding outcomes, but the effects were outcome- and herd-specific [8]. Real-world dairy vaccination protocols also vary substantially among herds, demonstrating that "vaccinated" can represent different schedules, products, target groups, and implementation practices [9]. Timing matters as well: prepartum vaccination relative to management transition altered measured serum and colostral immunoglobulin responses in Holstein cows [10].

The proposed architecture therefore separates host susceptibility, vaccine delivery, measurable immune response, and realized population protection. These states can diverge. A herd may have high vaccine coverage but heterogeneous protocol execution; measurable immune responses may not translate directly into reduced transmission; and protection may vary across age, physiological state, prior exposure, or pathogen strain. Decisions should consequently use the strongest available evidence for the relevant host-pathogen context while retaining uncertainty when only surrogate immune markers or administrative vaccination records are available.

Table 1 follows vaccination evidence from delivery records to field outcomes, showing where apparent coverage can fail to establish realized population protection.

Table 1. The vaccination-to-protection cascade and the evidence gap at each transition

Evidence Layer	What is Observed	Why the Next Layer is Not Guaranteed	Population-Protection Question
Vaccine Delivery Record	Product, schedule, target group, and nominal coverage	Recorded delivery may conceal uneven execution or subgroup omissions	Which animals were actually reached under the intended protocol?
Protocol Execution by Subgroup	Timing and delivery across age, physiological stage, or management group	Correct delivery does not guarantee a measurable or uniform response	Does susceptibility remain concentrated in a particular subgroup?
Measurable Immune Response	Serological or other surrogate immune evidence	A surrogate response is not equivalent to reduced infection, shedding, or transmission	Does the biological response correspond to field protection?
Field Clinical or Shedding Outcome	Observed changes in disease, production, treatment, or shedding	Effects can remain pathogen-, outcome-, and herd-specific	Is complementary control still required under the current exposure pressure?

Note: The cascade organizes evidence needed to infer population protection; it does not define a universal coverage threshold or validated protection score.

Animal movement and contact structure

Herds are embedded in contact systems rather than isolated production units. Livestock movement and contact-network studies show that transmission opportunities are distributed unevenly across connected holdings [2, 7]. This heterogeneity means that two herds with similar apparent biosecurity or clinical status may face different exposure pressure because their movement patterns, trading relationships, service contacts, or network positions differ. Swine breeding-herd data also show that introduction-relevant contacts span several categories, including animals, people, vehicles, and supplies [5].

The architecture therefore treats contact structure as a population property that modifies both introduction and onward dissemination. Movement restrictions or contact reduction may be important in some settings, but network position alone should not be interpreted as infection probability. Data completeness, temporal change, informal movements, and disease-specific modes of transmission can all alter inference. A useful control assessment must therefore ask not only whether contacts occur, but which contacts connect otherwise separated epidemiological units, whether they are recurring, and whether the receiving population is susceptible. Network evidence is consequently treated as a targeting aid and explanatory layer, not as a deterministic control rule.

Environmental persistence and contamination

The environment can preserve or redistribute infectious material, but environmental detection must not be conflated with viable pathogen or successful transmission. Repeated surveillance in Bangladeshi live-bird markets identified spatial and climatic variation in environmental avian-influenza occurrence, indicating that environmental risk can change with place and time [11]. Experimental work in artificial streams showed that viral RNA may remain detectable after recoverable infectious virus has declined, establishing a critical measurement boundary between molecular signal and infectivity [12]. Conversely, experimental African swine fever

contamination produced inefficient transmission to sentinel pigs under the conditions tested, showing that contamination alone does not guarantee infection [13].

The proposed architecture therefore separates three states: environmental evidence, infectious viability, and realized exposure sufficient for transmission. Environmental control can be justified without assuming that every positive sample represents the same hazard. Interpretation should consider matrix, temperature, persistence time, cleaning practices, animal access, dose, and sampling method where evidence exists. This distinction prevents both overreaction to persistent molecular signals and underestimation of environmental reservoirs when viable pathogen and exposure pathways remain plausible.

Clinical and syndromic surveillance

Clinical and syndromic surveillance represents the earliest observable layer of herd-level detection, but signals are filtered before they become actionable evidence. Farmer-based passive surveillance depends on recognition, motivation, trust, and reporting arrangements; absence of a report therefore cannot be treated as absence of disease [3]. Surveillance signals are also temporally unstable. Repeated environmental avian-influenza surveillance demonstrated that detectable risk patterns can vary across locations and climatic conditions [11], reinforcing the broader need to interpret any single observation within time and context rather than as a fixed herd state.

In the proposed architecture, a clinical or syndromic signal is an alert state, not a diagnosis. Changes in morbidity, mortality, production, behaviour, treatment demand, or other herd-level patterns may justify intensified observation or laboratory testing, but their specificity depends on disease and system context. The operational question is therefore whether a signal is isolated, repeated, clustered, escalating, or discordant with expected herd performance. The architecture also recognizes a reporting boundary: biologically meaningful change can exist without entering the formal surveillance system.

Table 2 maps surveillance blind spots across the path from herd change to laboratory confirmation, making non-detection distinguishable from true absence.

Table 2. Where a herd signal can disappear between biological change, recognition, reporting, and confirmation

Surveillance Layer	How The Signal Can Be Lost or Distorted	What May Still be Visible	Proportionate Recovery Action
biological change in the herd	Early, mild, dispersed, or nonspecific expression	Subtle morbidity, mortality, production, behavior, or treatment-demand changes	Increase attention when exposure pressure or clustering makes a weak signal consequential
observer recognition	Background disease, limited observation, or uncertain pattern recognition	Informal or isolated observations	Verify the pattern across animals, groups, and time
formal reporting	Trust, incentives, workflow, or delayed communication	Unreported knowledge held by workers or farmers	Repair the reporting pathway while preserving biological evidence
laboratory confirmation	Sampling quality, timing, test performance, target analyte, or diagnostic access	A reported syndrome with incomplete etiological evidence	Use targeted testing and interpret positive or negative results within their measurement limits
herd-state update	One signal is allowed to dominate or the alert is treated as a confirmed outbreak	Clinical, laboratory, environmental, and epidemiological evidence may disagree	Retain the alert state until the evidence supports resolution, escalation, or a competing explanation

Note: Surveillance layers identify where visibility can fail; they are not diagnostic definitions or universal escalation thresholds.

Laboratory surveillance

Laboratory surveillance adds etiological or exposure evidence, but its apparent precision can conceal important measurement uncertainty. A meta-analysis of bovine brucellosis serology found meaningful variation in diagnostic accuracy across tests and study conditions, showing that sensitivity, specificity, and reference-standard limitations affect herd-level interpretation [14]. For *Mycobacterium avium* subsp. *paratuberculosis*, phagomagnetic separation followed by quantitative PCR was evaluated as a rapid surveillance approach capable of targeting viable organisms in bulk-tank and individual-cow milk, yet sampling scale and dilution remain relevant to inference [15]. Repeated bulk-tank-milk ELISA surveillance in Alberta dairy herds further

demonstrates the practical value of herd-level sampling while also illustrating that antibody evidence is not identical to active infection or transmission intensity [16].

The architecture therefore places a laboratory-interpretation boundary between test result and control decision. Sample source, timing, target analyte, test performance, herd prevalence, and the distinction between exposure, viability, infection, and disease must be considered before escalation. A negative result may reduce uncertainty without eliminating it; a positive result may strengthen evidence without identifying the complete transmission state. Laboratory surveillance is thus a state-updating function within herd control, not an infallible endpoint.

Biosecurity as layered risk reduction

Biosecurity is most useful when treated as a portfolio of route-specific barriers rather than as a binary property of a farm. A pig-farm risk-assessment tool developed for porcine reproductive and respiratory syndrome explicitly differentiated multiple introduction and transmission routes, illustrating why one barrier cannot represent overall vulnerability [17]. Implementation, however, is not purely technical. Dairy-farm evidence shows that communication between veterinarians and farmers can influence whether recommended measures are considered understandable, feasible, and worth sustaining [18]. Similarly, turkey producers' perceptions of avian-influenza biosecurity demonstrate that practical implementation can diverge from formal recommendations even when the biological rationale is accepted [19].

The proposed architecture therefore separates designed biosecurity from implemented biosecurity. Entry controls, internal separation, hygiene, movement management, and environmental controls may address different pathways, while labour, infrastructure, cost, risk perception, and advisory relationships determine their realized use. A highly developed protocol should consequently not be interpreted as equivalent to effective risk reduction unless implementation and the targeted pathway are both plausible. Biosecurity also cannot compensate automatically for unrecognized infection, inadequate population protection, or continuing environmental exposure.

Vaccination and population protection

Vaccination is positioned within the architecture as a susceptibility-modifying intervention rather than a stand-alone declaration that the herd is protected. Field vaccination, herd vaccination protocols, and vaccination timing each show that realized effects depend on biological and implementation context [8-10]. These observations support a distinction between vaccine administration, immune response, coverage of relevant subgroups, and the population consequences ultimately observed.

This distinction is noncompensatory. High administrative coverage does not nullify an important introduction pathway, and a strong immune marker does not establish interruption of transmission. Conversely, imperfect vaccination does not imply that vaccination lacks value; it means that its contribution must be interpreted together with pathogen characteristics, population turnover, age or physiological structure, exposure pressure, and complementary controls. The architecture therefore proposes that vaccination decisions be reassessed when herd composition, exposure conditions, circulating pathogen characteristics, or measured outcomes change. No universal coverage threshold is inferred from the selected evidence, and population protection remains disease-, vaccine-, and setting-dependent.

Treatment within herd-level control

Treatment remains essential for animals likely to benefit clinically, but treatment decisions operate at a different analytical level from control of herd exposure and transmission. Evidence on selective treatment of clinical mastitis supports using clinical and diagnostic information to avoid unnecessary antimicrobial treatment where appropriate [20]. A meta-analysis of nonsevere clinical mastitis found that selective treatment did not adversely affect the specified cure, somatic-cell-count, milk-yield, recurrence, or culling outcomes compared with blanket treatment in the included evidence [21]. Those findings concern defined individual-animal outcomes; they do not demonstrate interruption of herd-level transmission.

Treatment also sits within an antimicrobial and production ecology. Finnish pig-farm data examined antimicrobial use together with biosecurity, herd characteristics, and antimicrobial resistance, with several relationships remaining weak or inconsistent [22]. A substantially larger longitudinal German analysis showed that antibiotic consumption varied with farm and regional characteristics [23]. Accordingly, the architecture treats treatment as one response to disease already expressed or detected, while prevention of new exposure, amplification, and recurrence requires additional population-level controls.

Table 3 compares the evidentiary meaning of success at three nested scales, preventing improvement in treated animals from being misclassified as interruption of herd transmission.

Table 3. Treatment success interpreted at the animal, case-group, and herd levels

Control Question	Individual Animal	Affected Group or Eligible Cases	Herd or Population
Primary Objective	Achieve clinically appropriate benefit for the affected animal	Apply selective treatment to cases most likely to benefit	Reduce introduction, amplification, persistence, and recurrence
Evidence of Success	Clinical response and relevant diagnostic change	Defined cure, recurrence, production, or treatment outcomes in eligible cases	Declining or absent transmission-relevant signals across surveillance and exposure pathways
Inference That is Not Justified	Recovery does not prove the herd is controlled	Condition-specific equivalence does not validate all diseases or case definitions	Lower antimicrobial use alone does not prove better biological control
Complementary Control Requirement	Continue patient-specific reassessment	Audit eligibility and escalate when clinical status changes	Address movement, contact, environment, susceptibility, surveillance, and implementation as indicated

A proposed integrated herd-disease architecture

The central contribution of this article is a proposed integrated herd-disease architecture in which control decisions are organized around the current configuration of population risk rather than around the presence of a treated case. Introduction pressure, within-herd amplification, host susceptibility, movement and contact structure, environmental persistence, clinical signals, laboratory evidence, and intervention capacity remain analytically distinct because evidence in one domain cannot automatically cancel risk in another. Contact heterogeneity may alter exposure [5]; environmental signals require interpretation of viability and transmission potential [12]; laboratory results remain test- and sampling-dependent [14]; and layered biosecurity addresses different pathways [17]. Treatment therefore occupies one intervention domain rather than functioning as the architecture's endpoint [20].

The proposed decision logic is conditional and revisable. Surveillance evidence updates the inferred herd-risk state; the inferred state determines which combination of biosecurity, vaccination, treatment, environmental control, or intensified surveillance is proportionate; and subsequent evidence determines whether that decision should be maintained, modified, escalated, or withdrawn. Uncertainty remains explicit rather than being converted into an unsupported numerical score.

This multilevel logic is compressed visually because its key contribution lies in the relationships and boundaries among domains rather than in any single component.

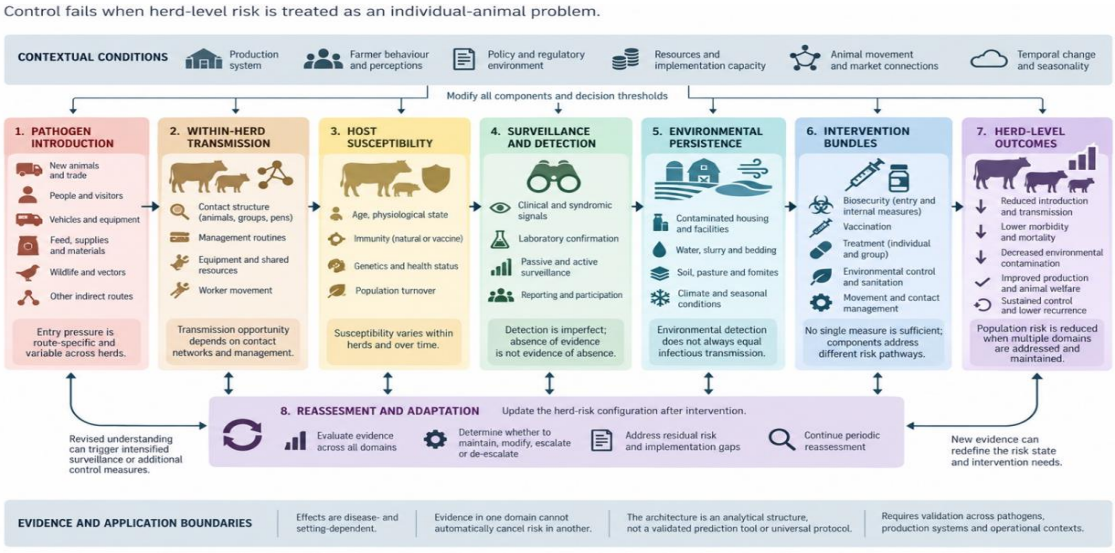

Figure 1. Herd risk is adjudicated across entry pressure, amplification, susceptibility, detection, environmental persistence, and intervention capacity

Intervention bundles and conditional escalation

An intervention bundle should not be interpreted as "more control" in the abstract; its value depends on whether its components address the risk configuration actually present. In village-chicken systems, animal-health interventions altered farmers' perceptions of disease susceptibility and preventive behaviour, showing that an intervention can change the behavioural environment in which subsequent control decisions occur [24]. In ASF-endemic smallholder settings, co-created community contracts supported changes in biosecurity, although complete implementation remained constrained [25]. Longitudinal *Mycoplasma bovis* control likewise demonstrates the relevance of repeated testing and combined herd measures [26]. At district scale, *Staphylococcus aureus* genotype B sanitation combined surveillance, farm measures, and targeted animal-level actions within one coordinated programme [27].

The architecture therefore proposes conditional escalation: add or intensify measures when unresolved introduction, transmission, susceptibility, environmental, detection, or implementation pathways justify doing so. Escalation is not necessarily maximal intervention. A narrowly targeted addition may be preferable when one residual pathway is identifiable, whereas broader bundles may be warranted when several domains remain unresolved. Component effects should not be inferred from uncontrolled bundle outcomes.

Reassessment after intervention

An intervention changes the conditions being managed, making the pre-intervention risk assessment progressively less reliable. Longitudinal evidence across animal-health interventions, herd-control programmes, and coordinated sanitation shows that biological status, behaviour, and implementation can change after action [24, 26, 27]. Reassessment is therefore part of control rather than an optional audit after completion.

The architecture proposes that reassessment compare the new herd-risk configuration with the configuration that justified intervention. Complete resolution is only one possible outcome. Other states include partial biological response with a residual exposure pathway, improved laboratory evidence with continuing syndromic concern, adequate biological control with incomplete implementation, or renewed risk following movement or population turnover. Participatory ASF evidence additionally shows that implementation can remain incomplete even when change has begun [25].

No universal reassessment interval is proposed. Timing should depend on pathogen dynamics, diagnostic windows, intervention mechanism, herd turnover, movement, environmental persistence, and the expected speed at which meaningful evidence can change.

Table 4 reads the post-intervention pattern across evidence streams, allowing residual exposure, incomplete delivery, and renewed vulnerability to remain distinguishable from biological resolution.

Table 4. Post-intervention herd patterns resolved across biological, epidemiological, and implementation evidence streams

Post-Intervention Pattern	Biological Response (Clinical and Laboratory)	Residual Exposure or Susceptibility	Delivery Fidelity	Control Interpretation
Apparent Resolution	Adverse signals decline and laboratory findings are concordant within the diagnostic window	No major current pathway is evident	Core controls are delivered	Maintain proportionate controls and consider cautious de-escalation; absence may still be incomplete
Partial Response With A Residual Pathway	Some outcomes improve while others or laboratory findings remain unresolved	Movement, environment, or susceptibility remains plausible	Delivery may be adequate	Target the unresolved pathway rather than automatically repeat the entire bundle
Evidence Discordance	The syndromic course and laboratory evidence do not align	Pathway evidence may support a competing account	Fidelity may be adequate or uncertain	Investigate timing, test performance, and alternative explanations before major reclassification

Post-Intervention Pattern	Biological Response (Clinical and Laboratory)	Residual Exposure or Susceptibility	Delivery Fidelity	Control Interpretation
Implementation-Limited Response	Improvement is absent or smaller than expected, and efficacy cannot be established under incomplete delivery	Targeted pathways may remain open	Uptake or fidelity is demonstrably incomplete	Adapt implementation and reassess both delivery and biological outcome
Renewed Vulnerability	New herd signals or changing laboratory evidence emerge after earlier improvement	New animals, movements, waning protection, or exposure reopens risk	Earlier implementation may have been successful	Re-enter the current risk configuration rather than assume previous success persists

Note: The post-intervention patterns are proposed analytical configurations, not validated thresholds or prognostic classes.

The recursive decision logic is also shown graphically because reassessment can redirect rather than simply repeat the original intervention.

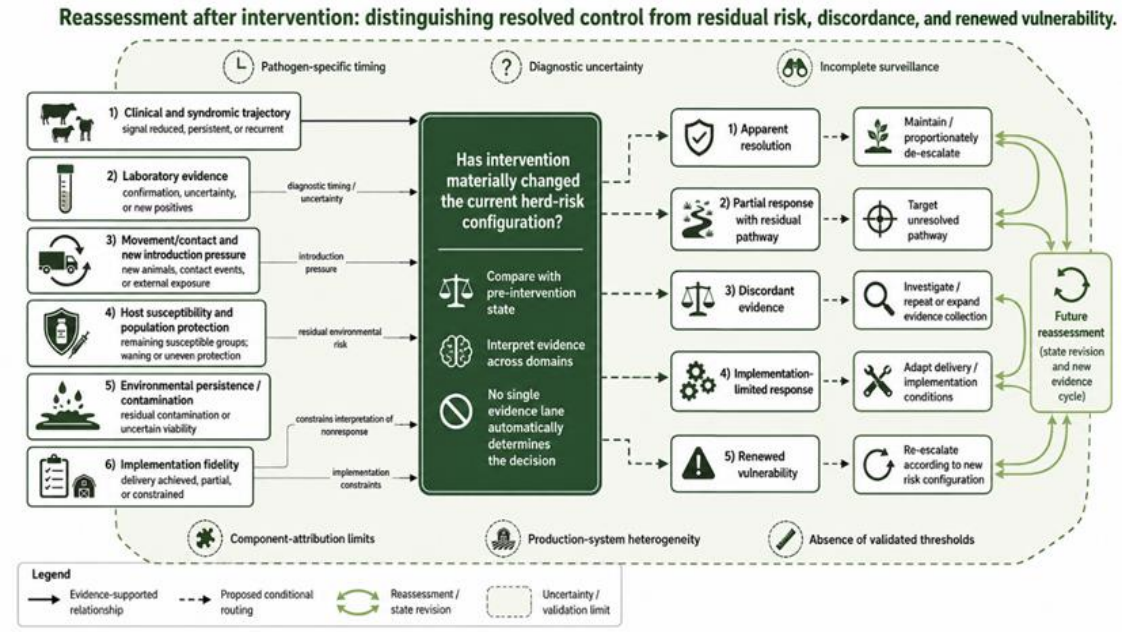

Figure 2. Reassessment separates resolved control from residual exposure, incomplete implementation, discordant evidence, and re-emergent herd risk

Post-intervention evidence is re-entered into a proposed reassessment field rather than interpreted as a binary success/failure outcome. Parallel evidence lanes preserve biological response, surveillance findings, residual exposure, population susceptibility, environmental conditions, and implementation fidelity. Conditional branches distinguish apparent resolution, partial response, discordance, implementation-limited response, and renewed vulnerability. Dashed connectors represent proposed routing; return connectors indicate repeated state revision.

Failure of single-measure control strategies

The central criticism of single-measure control is not that every isolated intervention is ineffective, but that no intervention can be assumed to address every mechanism sustaining herd risk. Interviews with German pig farmers showed that perceived threat, implementation uncertainty, and practical conditions could limit ASF biosecurity decisions despite available information [28]. In Italian smallholder pig systems, economic, social, cultural, ecological, and regulatory conditions further constrained implementation [29]. Biological effectiveness is equally conditional: a randomized Australian field trial found that the tested commercial pinkeye vaccine did not reduce disease incidence under the evaluated conditions [30]. Surveillance itself can also be misallocated when

geographically or network-defined risk is heterogeneous; combining spatial statistics and social-network analysis has been used to identify differentiated disease-circulation areas for risk-based surveillance [31].

These examples support a proposed taxonomy of single-measure failure: pathway mismatch, incomplete implementation, context-dependent biological performance, residual susceptibility, unresolved environmental risk, and surveillance mis-targeting. They do not establish that single measures always fail. A focused intervention may be sufficient when the relevant mechanism is narrow, correctly identified, and adequately controlled.

Implementation across production systems

Implementation determines whether a biologically plausible intervention becomes an operational control. Dairy evidence indicates that veterinarian–farmer communication can shape biosecurity adoption [18], while poultry evidence shows that producers' perceptions and practical circumstances influence application of recommended measures [19]. Community-based ASF work demonstrates that locally co-created approaches can support change under constrained smallholder conditions [25]. German pig-farmer interviews emphasize uncertainty, regulation, perceived threat, and trusted guidance [28], whereas Italian smallholder analysis highlights broader social, cultural, ecological, and economic constraints [29].

The architecture therefore treats implementation capacity as a boundary condition rather than as a final step after intervention selection. Intensive systems may have greater infrastructure but complex movement networks; smallholder systems may face capital, labour, market, or cultural constraints that alter feasible control. These differences should not be converted into assumptions about compliance or effectiveness. Instead, the relevant question is whether the selected control can be delivered with adequate fidelity in the production system concerned. Where delivery is limited, adaptation should be distinguished from abandonment of the underlying biological objective.

Validation priorities

The proposed architecture requires validation at several levels before it could support predictive or prescriptive use. A 2025 analysis of animal-health syndromic surveillance systems found that many identified systems remained proof-of-concept rather than fully operational, illustrating the gap between technical detection and sustained deployment [32]. Stakeholder research further indicates that participation can depend on standardization, trust, privacy, data integration, and perceived value [33]. Operational validation must therefore include participation and workflow, not merely signal-detection performance.

Model validity also depends on the evidence used to characterize transmission. PARAMETRA documents substantial variation in available livestock transmission parameters across pathogens and contexts, cautioning against uncritical transfer of parameter values between settings [34]. Formal risk tools similarly require explicit development and validation; a scoring system for PRRS introduction vulnerability in swine breeding herds provides a disease-specific methodological example [35].

Future testing should therefore examine construct validity, prospective classification, calibration, external transportability, comparative decision utility, surveillance timeliness, intervention fidelity, reassessment value, and robustness to missing or discordant evidence. The architecture should be considered refutable: empirical work may require domains to be merged, separated, reweighted, or abandoned.

Conclusion

Herd disease control is not equivalent to successful management of diseased animals. Population risk emerges from interacting introduction pathways, contact structure, susceptibility, environmental conditions, surveillance performance, intervention effects, and implementation capacity. The architecture proposed here preserves those domains as distinct but connected and places treatment, vaccination, biosecurity, environmental control, and surveillance within a revisable population-health decision process. Its central requirement is reassessment: intervention changes the system being managed, so the evidence supporting the original decision cannot be assumed to remain current. The framework does not establish universal thresholds, causal sufficiency, predictive performance, or implementation readiness. Its value is presently conceptual—providing a disciplined structure for testing whether veterinary disease-control decisions adequately address the herd-level conditions that allow infection to enter, circulate, persist, escape detection, or recur.

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