Eurasia Specialized Veterinary Publication

International Journal of Veterinary Research and Allied Science

ISSN:3062-357X

2026, Volume 6, Issue 1, Page No: 55-65

Available online at: www.esvpub.com/

Resilient Herd Health Requires Managing the Next Failure Before It Appears: A Dynamic Veterinary Architecture Integrating Biosecurity, Population Immunity, Pathogen Pressure, Production Stress, Detection Capacity, Response, and Recovery

Lukas Weber[1]*, Sophie Janssen[2], Jonas Richter[1], Elena Fischer[3]

[1]Department of Veterinary Herd Health and Resilience, Faculty of Veterinary Medicine, University of Stuttgart, Stuttgart, Germany.
[2]Department of Veterinary Biosecurity and Disease Prevention, Faculty of Veterinary Medicine, Eindhoven University of Technology, Eindhoven, Netherlands.
[3]Department of Veterinary Population Immunity and Recovery, Faculty of Veterinary Medicine, University of Basel, Basel, Switzerland.

***E-mail** ✉ lukas.weber@uni-stuttgart.de

ABSTRACT

Herd-health programs commonly organize prevention, surveillance, outbreak control, and recovery as separate activities. This fragmentation can leave a herd apparently secure while the capacity most likely to fail next is deteriorating. This article develops an original non-empirical architecture in which resilience is defined as the time-dependent ability of a herd system to limit pathogen entry, absorb exposure without disproportionate loss, detect consequential change, mobilize an appropriate response, and restore biological and operational reserve. The analysis separates external pathogen pressure from internal transmission pressure; population immunity from nominal vaccination coverage; production output from physiological reserve; test performance from detection capability; and pathogen control from recovery. It further proposes that outbreaks alter which constraint is binding. A biologically limited event may become a workforce, diagnostic, financial, welfare, or governance failure, while apparently successful control can conceal depleted reserve and greater vulnerability to recurrence. The resulting architecture treats herd condition as a trajectory across interacting pressure, reserve, capability, action, and recovery domains rather than as a static disease status. It supports adaptive intervention bundles selected for the current constraint and revised as that constraint migrates. The architecture is intended to guide structured reasoning, measurement development, and prospective evaluation, not to provide a validated score, universal threshold, or ready-to-deploy decision rule. Its applicability will depend on species, pathogen, production system, surveillance infrastructure, resource availability, welfare priorities, and the temporal resolution of available data.

Keywords: Herd resilience, Biosecurity capacity, Population immunity, Pathogen pressure, Veterinary surveillance, Outbreak recovery

Received: 20 December 2025
Revised: 12 March 2026
Accepted: 16 March 2026

How to Cite This Article: Weber L, Janssen S, Richter J, Fischer E. Resilient Herd Health Requires Managing the Next Failure Before It Appears: A Dynamic Veterinary Architecture Integrating Biosecurity, Population Immunity, Pathogen Pressure, Production Stress, Detection Capacity, Response, and Recovery. Int J Vet Res Allied Sci. 2026;6(1):55-65.
https://doi.org/10.51847/fCTkjttmkQ

Introduction

Herd protection is often described through visible controls: vaccination, movement restrictions, hygiene, testing, and written contingency plans. Their presence, however, does not establish that they will function together under changing exposure, labor, production, or diagnostic conditions. Biosecurity implementation is shaped by interacting behavioral and contextual drivers, so nominal adoption is not equivalent to dependable execution [1].

Surveillance is similarly uneven: many animal-health syndromic systems remain proofs of concept, and even operational systems may depend on fragile institutional support [2].
Resilience adds a temporal question: how does the herd respond when prevention is incomplete? Dairy production records demonstrate between-herd variation in disturbance and recovery patterns, but such indicators remain proxies rather than species-general measures of health resilience [3]. Recent H5N1 experience also shows that one shock can couple clinical disease, production loss, and economic consequences within the same enterprise [4]. This article therefore proposes a dynamic veterinary architecture that keeps exposure, transmission, immunity, stress, biosecurity, detection, response, and recovery analytically distinct while examining their interaction. It does not assert a universal mechanism or validated index; it identifies the next likely failure as the primary object of anticipatory herd-health management.

From disease prevention to herd resilience
Disease prevention asks whether infection or clinical disease was avoided. Herd resilience additionally asks how much reserve was consumed, how rapidly consequential change was recognized, whether response remained feasible, and what capacity remained afterward. This distinction prevents an uneventful period from being mistaken for robust protection. A biosecurity practice may be present yet behaviorally unreliable [1]; a surveillance stream may operate yet remain too immature or institutionally fragile for dependable action [2].
Production-derived perturbation measures illustrate the value and limitation of trajectory thinking. They can reveal variation in disturbance response between dairy herds, but they do not directly identify pathogen exposure, welfare state, immune competence, or operational readiness [3]. Resilience is therefore proposed here as a system property inferred from several time-linked domains, not a synonym for low incidence or stable output. Evidence of prevention remains important, but its interpretation depends on the pressures faced and the capacities actually tested. The practical shift is from asking whether the last control worked to asking which capacity is becoming least able to contain the next disturbance.

External pathogen pressure
External pathogen pressure is the changing force of introduction generated by pathogen prevalence, host range, infectious material, movement networks, wildlife interfaces, vectors, markets, and neighboring premises. It is not a farm attribute alone. The emergence of H5N1 in dairy cattle demonstrates how host-range expansion can abruptly change the relevant source population and invalidate established assumptions about exposure [5]. Detection of infection in cattle and farm-associated cats further shows that a farm can sit within a connected multi-host interface [6].
Pressure also depends on topology. Movement-informed modelling of African swine fever at wildlife–livestock interfaces indicates that connectivity can alter plausible spread patterns, although model predictions remain conditional on regional ecology and assumptions [7]. The proposed architecture therefore treats external pressure as time-varying and route-specific. A herd with unchanged internal controls may become less protected because surrounding prevalence, movement, host ecology, or environmental carriage has changed. Conversely, pathogen presence outside the herd does not quantify incursion probability without route, contact, and control information. External pressure should trigger reassessment, not automatic attribution of infection.

Internal tansmission pressure
After incursion, external pressure becomes internal transmission pressure only through effective contacts among susceptible animals, people, equipment, biological carriers, shared air or water, and contaminated environments. The distinction matters because measures that reduce entry may not contain amplification. H5N1 evidence from affected dairy settings is consistent with a transition from cross-species introduction to cattle-associated spread [5], while concurrent infection in cattle and cats identifies additional within-farm interfaces without proving a single dominant route [6].
Internal pressure is therefore proposed as the product of infectious burden, contact opportunity, shedding duration, compartmental separation, and removal or isolation speed. Movement structure is relevant within as well as between populations, but transmission models remain conditional rather than direct measurements of every contact pathway [7]. Herd density, grouping, milking order, shared maternity areas, replacement flows, and sick-pen organization may change amplification even when perimeter biosecurity is stable. Interpretation must also allow hard cases: clustered detection may reflect shared exposure rather than animal-to-animal spread, and

negative tests may reflect mistimed sampling. Control should target plausible amplification routes while preserving uncertainty about unmeasured pathways.

Population immunity

Population immunity is not synonymous with vaccine coverage. It includes the distribution of immune protection across animals, pathogens or strains, age groups, prior exposures, time since vaccination, and physiological states. A randomized field trial of foot-and-mouth disease vaccination supports protective effectiveness under its Ethiopian conditions, but effectiveness remains linked to vaccine, circulating strains, delivery, and exposure context [8].

Serological evidence from German sheep and goats exposed to Schmallenberg virus demonstrates temporal and between-population heterogeneity, while also showing why seropositivity should not be treated as a fixed or complete measure of protection [9]. Controlled bovine respiratory syncytial virus work further indicates that maternal antibodies and vaccination route can modify protection in calves [10]. The architecture therefore separates four questions: who received an intervention, who developed a measurable response, who remains functionally protected under current exposure, and where susceptible clusters persist. Herd-average coverage can conceal vulnerable cohorts. Immunity assessment should be pathogen-, time-, and subgroup-specific, and should inform contact management and surveillance rather than operate as a standalone assurance.

Production and physiological stress

High production can coexist with declining reserve. Milk yield, growth, reproduction, feed intake, behavior, and routine activity may remain acceptable during compensation, then change abruptly when thermal, nutritional, immune, social, or management demands exceed adaptive capacity. Production perturbation indicators can identify altered trajectories, but their biological meaning is not self-evident [3]. Likewise, outbreak-associated production loss demonstrates consequence, not the physiological pathway that produced it [4].

The architecture therefore distinguishes production demand, physiological load, compensation, and decompensation. Immunity evidence also cautions that age and immune context modify the response to intervention [10], while field vaccine effectiveness remains exposure-dependent [8]. Proposed “reserve erosion” should not be inferred from one low output value or one apparently normal value. It requires convergent trends, relevant modifiers, and a defined time window. A falling feed-intake trajectory during heat load carries different meaning from chronically low output in a stable system. **Table 1** presents production trajectories that distinguish compensation, absorbed disturbance, decompensation, and residual vulnerability.

Table 1. Production trajectories that distinguish compensation, absorbed disturbance, decompensation, and residual vulnerability

Trajectory question	Stable output under rising load	Brief deviation, rapid return	Persistent decline with discordance	Output recovered; domains lag	Subgroup-specific decline
Physiological reading	Possible compensation	Disturbance absorbed	Decompensation or disease possible	Residual vulnerability proposed	Localized susceptibility proposed
Load or subgroup conditions	Heat, gestation, regrouping	Stage, routine, measurement cadence	Exposure, ration, welfare, immunity	Recent outbreak, weight loss, depleted labor	Age, parity, pen, immune timing
Concurrent production and animal evidence	Intake, activity, temperature, or behavior diverges	Bounded perturbation	Coupled output and clinical change	Fertility, health, or resources lag	Herd mean masks cohort change
Management response	Reduce load; intensify observation	Document trigger and recovery	Escalate diagnostics	Continue recovery management	Target sampling
Alternative explanation	Normal output can conceal strain	Error remains possible	Nonspecific pattern	Asynchronous normalization	Composition may distort trends
Confirmation window	Verify against baseline	Check recurrence	Test competing causes	Delay “resolved” status	Recalculate by subgroup

Note: Proposed distinctions; not thresholds or an additive resilience score.

Environmental exposure

Environmental exposure includes physical conditions that alter host reserve and environmental routes that carry infectious material. Controlled heat exposure in dairy calves was associated with segment-specific changes in intestinal barrier markers, inflammatory signals, and microbial communities, supporting biological effects beyond reduced feed intake while not establishing later disease causation [11]. Dairy welfare evidence likewise shows that thermal load affects behavioral and physiological domains that production measures alone may miss [12].

The environment can also bridge perimeter and internal transmission. Detection of highly pathogenic avian influenza material in field-collected blowflies supports vector potential near poultry farms, but does not demonstrate that flies caused farm infection or quantify their transmission contribution [13]. The architecture consequently separates environmental load on the host from environmental carriage of a pathogen. They may interact: heat, ventilation, housing, manure handling, water systems, insects, wildlife access, and shared surfaces can modify susceptibility or contact opportunity through different pathways. Environmental signals should therefore prompt route-specific investigation, not a generic "environmental risk" label or unverified causal conclusion.

Biosecurity capacity

Biosecurity capacity is the ability to execute appropriate separation, hygiene, movement, sourcing, isolation, and containment practices reliably under ordinary and outbreak conditions. A checklist records reported or observed measures; it does not by itself establish correct execution, continuity, pathogen specificity, or resistance to workload shocks. Behavioral evidence indicates that implementation depends on motivation, social and organizational context, perceived feasibility, and available resources [1].

Capacity must therefore be assessed as a functioning control system. Relevant questions include whether responsibility is assigned, supplies are available, dirty–clean boundaries are workable, new or returning animals can be segregated, deviations are detected, and practices persist during staff shortage or production pressure. External and internal pathogen pressures determine which barrier matters most at a given time [5]. Environmental carriers may also reveal routes that routine perimeter logic overlooks [13]. The proposed distinction is between documented biosecurity, practiced biosecurity, and stress-tested biosecurity. None can be reduced to a universally comparable score without validated measurement, and an apparent gap should be linked to a plausible exposure or amplification pathway before intervention is prioritized.

Detection capability

Detection capability is the probability that consequential change will be recognized, reported, sampled, tested, interpreted, and communicated early enough to alter action. A prospective livestock-abortion surveillance program in Tanzania demonstrates how coordinated notification, field investigation, and laboratory testing can reveal diverse livestock and zoonotic causes [14]. Its performance, however, depends on a resource-intensive chain rather than on a test alone.

Modelling of surveillance in porcine reproductive and respiratory syndrome-free regions shows that early-detection sensitivity can vary with the mix and timing of surveillance components [15]. Broader methodological work similarly frames surveillance as socio-technical: participation, technologies, data flows, governance, and fit-for-purpose design shape what becomes observable [16]. Capability should therefore be described as a state, not a binary asset. **Table 2** presents where recognition, reporting, sampling, testing, and actionability fail in the herd detection chain.

Table 2. Where recognition, reporting, sampling, testing, and actionability fail in the herd detection chain

Failure point	Signal transformation lost	Conditions producing the loss	Repair point	Residual risk and audit clock
Sparse signal generation	**Meaning:** Change poorly observable **Observed bottleneck:** Nonspecific observations	Visits, sensors, recognition	Increase targeted observation	**Residual risk:** Added data can add noise **Audit:** Recheck baseline and coverage
Reporting interrupted	**Meaning:** Recognition fails to escalate **Observed bottleneck:** Concern without notification	Trust, incentives, staffing	Repair reporting route	**Residual risk:** Silence cannot quantify underreporting **Audit:** Audit escalation time

Sampling delayed	**Meaning:** Diagnostic clock advances **Observed bottleneck:** Recognition-to-collection gap	Distance, handling, authority	Pre-position kits and transport	**Residual risk:** Early sampling may miss shedding **Audit:** Review collection timing
Result not actionable	**Meaning:** Output lacks timely context **Observed bottleneck:** Late or indeterminate result	Turnaround, metadata, test limits	Link tests to decision triggers	**Residual risk:** Detection is not diagnosis **Audit:** Audit decision time
Integrated warning proposed	**Meaning:** Signals converge operationally **Observed bottleneck:** Time-stamped signal-to-action record	Staffing, governance, feedback	Act proportionately; retain confirmation	**Residual risk:** Benefit unvalidated **Audit:** Evaluate prospectively

Note: Proposed failure locations, not performance grades or clinical-utility claims.

Diagnostic delay

Diagnostic delay is not merely laboratory turnaround time. It accumulates through recognition, reporting, case definition, sampling, transport, testing, interpretation, and authorization. Syndromic surveillance may move recognition earlier, yet many systems remain non-operational or institutionally fragile [2]. Field surveillance shows that diagnostic yield depends on coordinated case capture and investigation [14], whereas PRRS modelling indicates that cadence and component coverage alter expected early-detection sensitivity [15].

Delay allows continued mixing, exposes susceptible cohorts, and may narrow response options. Yet faster is not automatically better when noisy signals trigger disproportionate action. Participatory and digital approaches can widen observation but require governance, verification, and a defined intervention path [16]. The architecture treats delay as a distributed, auditable interval. Programs should time-stamp each transition, identify the dominant bottleneck, and distinguish an alert from a confirmed outbreak. No universal acceptable delay is proposed; the boundary depends on pathogen kinetics, consequences, test characteristics, feasible precaution, and response reversibility.

Response capacity

Response capacity converts a credible signal into coordinated action while preserving animal care and essential farm functions. It includes authority, trained personnel, isolation space, diagnostic access, supplies, communication, and endurance. Surveillance modelling shows why response cannot be inferred from detection: earlier recognition has limited value if no feasible action follows [15]. Participatory and digital surveillance likewise requires governance and an intervention pathway [16].

Capacity is pathogen- and phase-specific. A herd may isolate a few animals yet be unable to reorganize milking, maintain welfare during restrictions, or replace absent staff. Practices can degrade as workload and feasibility change [1]. The architecture distinguishes nominal plans, mobilizable resources, and sustained performance. It proposes no universal threshold. Plans should instead identify decisions that become irreversible with delay and resources whose failure would force a less effective or less welfare-compatible response.

Recovery capacity

Pathogen control does not define recovery. Recovery capacity concerns restoration across health, reproduction, production, welfare, workforce, liquidity, market access, and preparedness. Dutch cattle data associated bluetongue virus serotype 3 with mortality, abortion, and premature births, illustrating burdens beyond recognized acute cases [17].

African swine fever modelling in Haiti indicates that control and recovery trade-offs can fall unevenly across vulnerable livelihoods [18]. Foot-and-mouth disease evidence from Thailand similarly shows differing impacts among dairy, beef, and pig systems [19]. These studies do not establish a common trajectory; they support multidimensional, context-specific recovery. The architecture asks whether resumed output accompanies restored biological reserve, reproductive continuity, workforce function, financial flexibility, and future response capacity. Any impaired domain can become the next binding constraint.

Recurrent exposure and loss of resilience

Repeated exposure need not produce identical outcomes. Effects depend on the interval, remaining immunity, susceptible replacement, incomplete recovery, accumulated stress, and operational losses. Temporal variation in Schmallenberg virus seropositivity demonstrates changing immune landscapes [9]. Bluetongue-associated reproductive and mortality burdens can extend beyond immediate detection [17], while foot-and-mouth disease losses differ across production systems [19].

Loss of resilience is proposed here as a narrowing ability to absorb another disturbance without crossing an escalation boundary. It cannot be diagnosed from recurrence alone: repeated outbreaks may reflect rising external pressure, poor fit of controls, pathogen evolution, or surveillance improvement. Nor does stable production prove preserved reserve. **Table 3** presents alternative explanations for recurrent herd disturbance and the observations that separate them.

Table 3. Alternative explanations for recurrent herd disturbance and the observations that separate them

Observed recurrence	Internal reserve claim	External or system explanation	Evidence separating accounts	Management priority	Comparison event
New incursion	Not established	**Conditions:** Source, movement, host range **Competing account:** May not be internal failure	Distinct introduction	Reassess perimeter	Update pressure map
Persistent circulation	Containment inadequate possible	**Conditions:** Contacts, shedding, isolation **Competing account:** Shared exposure may mimic spread	Cases without clearance	Target amplification	Reconstruct timing
Re-exposure before recovery	Vulnerability proposed	**Conditions:** Immunity, weight, reproduction **Competing account:** Recovery is domain-specific	Event during impairment	Reduce load; protect	Track reserve
Output restored; capacity depleted	Hidden loss proposed	**Conditions:** Labor, liquidity, surveillance **Competing account:** Output is insufficient	Resources remain reduced	Rebuild reserve	Audit capacities
Disturbances intensify	Resilience loss possible	**Conditions:** Pathogen, measurement, turnover **Competing account:** Exposure may have increased	Deeper deviations	Investigate	Compare equivalent events

Note: Proposed interpretations; no recurrence threshold or causal diagnosis is established.

A proposed herd-resilience architecture

The architecture organizes herd state through pressure, reserve, capability, action, and recovery. Pressure combines introduction and amplification; reserve includes immunity and physiological capacity; capability includes biosecurity, detection, and response. Action is what is mobilized, not what is recorded. Recovery covers biological and operational restoration. Biosecurity reviews show variation in tool scope, scoring, and validation, cautioning against interchangeable capacity scores [20].

Dairy biosecurity measures cluster rather than occur independently, although co-occurrence does not demonstrate synergy [21]. Kenya's surveillance evolution illustrates that farm capability is nested within laboratory, workforce, data, and governance systems [22]. The architecture does not add domain scores. It asks which constraint is closest to failure, how it changes other observations, and whether action reduces pressure or relocates vulnerability. **Figure 1** compresses these states, dependencies, and proposed escalation boundaries.

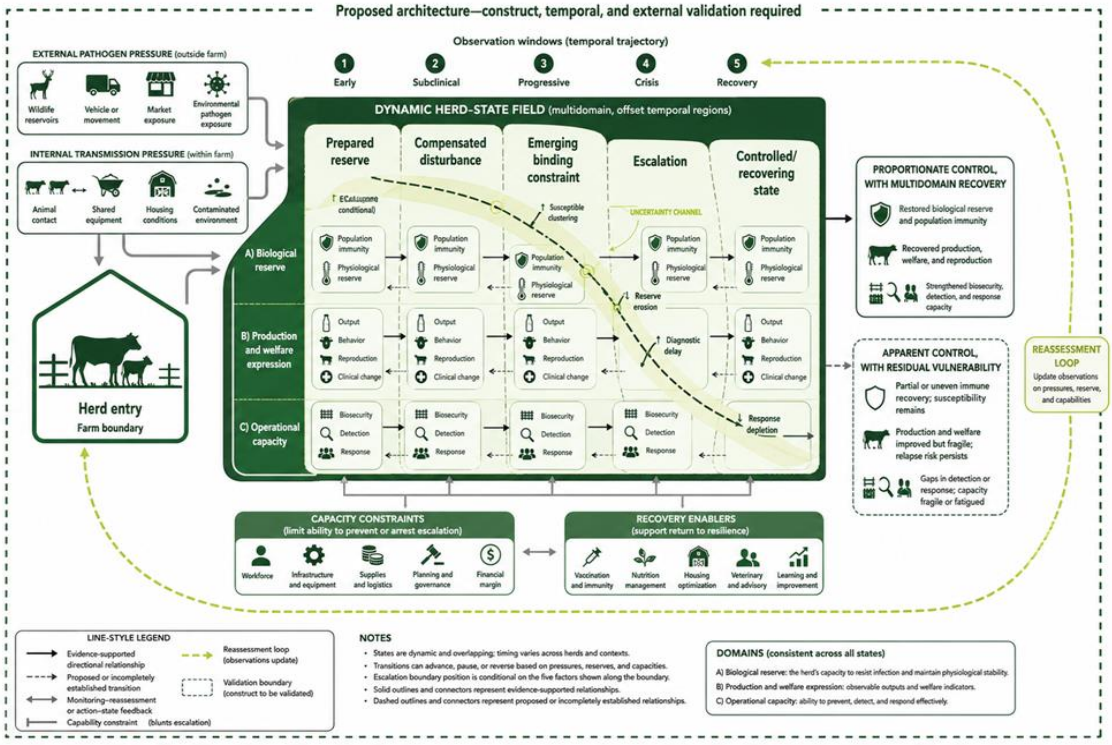

Figure 1. Herd-state trajectories and migrating escalation boundaries under interacting pathogen pressure and capacity loss

Constraint migration during outbreaks

An outbreak changes the system controlling it. Cases increase care needs; isolation changes workflow; diagnostics consume supplies; movement controls disrupt production; and income loss restricts choices. H5N1-associated health, production, and economic consequences illustrate simultaneous loading of several domains [4]. Bluetongue reproductive losses can extend beyond acute detection [17], while African swine fever modelling shows control–livelihood interactions [18].

Constraint migration is the proposed shift in the domain limiting feasible control. A pathogen-limited event may become laboratory-, workforce-, welfare-, or liquidity-limited. Constraints do not follow a proven sequence; order depends on species, scale, regulation, markets, and redundancy. Reassessment should follow major actions because a measure can reduce transmission while creating another bottleneck. Capacity consumption therefore warrants monitoring alongside case counts.

Failure propagation across herd systems

Failures propagate through more than infection chains. Modelling suggests poultry market structure can shape conditions favoring virulent pathogens, but does not establish a universal market effect [23]. African swine fever evidence shows interaction among hosts, persistence, movements, and control limitations [24]. Stress physiology provides another route: immune and metabolic demands compete for resources, linking challenge to production [25].

The architecture distinguishes contagious propagation, shared-resource propagation, informational propagation, and policy-mediated propagation. These pathways may reinforce one another, but identical arrows would misrepresent their evidence. Delayed information can prolong exposure; workforce depletion can weaken barriers; production pressure can reduce feasible isolation; and poorly aligned incentives can undermine reporting. **Table 4** presents distinct routes by which infectious, resource, informational, production, and policy failures propagate.

Table 4. Distinct routes by which infectious, resource, informational, production, and policy failures propagate

Propagation route	What propagates	Infrastructure carrying it	Signature in the herd	Interruption point	Rival explanation and activation trigger
Contagious	Internal amplification likely	Contacts, shedding, segregation	Linked group cases	Interrupt contacts	**Rival account:** Common source

Propagation route	What propagates	Infrastructure carrying it	Signature in the herd	Interruption point	Rival explanation and activation trigger
					Trigger: Contact map changes
Shared-resource	Demand weakens capacity	Staff, supplies, isolation	Care and barriers deteriorate	Protect limiting resource	**Rival account:** Coincident disruption **Trigger:** Use exceeds replacement
Informational	Delay spreads error	Reporting, metadata, authority	Repeated late decisions	Repair handoffs	**Rival account:** Test uncertainty **Trigger:** Action interval worsens
Production-mediated	Control becomes infeasible	Milking, housing, welfare	Routines conflict with containment	Redesign workflow	**Rival account:** Unrelated nonadherence **Trigger:** Feasibility changes
Policy-mediated	Incentives redirect behavior	Compensation, access, trust	Cooperation changes	Align safeguards	**Rival account:** Stated versus actual behavior **Trigger:** Participation changes

Note: Proposed pathways; no causal ordering or effectiveness is established.

Adaptive intervention bundles

Single measures rarely address every constraint. Adaptive bundles combine entry control, contact reduction, immunity management, environmental modification, surveillance, care, welfare safeguards, communication, and resource protection according to state. Feasibility and context affect whether biosecurity persists [1]. Dairy measures also co-occur, but association does not establish bundle synergy [21].

Three rules are proposed: target the binding constraint; protect labor, isolation, diagnostic throughput, and trust consumed by the bundle; and define triggers for intensifying, replacing, or withdrawing components. Pathogen-specific control remains necessary because African swine fever hosts, persistence, and feasibility cannot be generalized [24]. Physiological stress should be managed where it narrows reserve, without assuming that stress reduction prevents infection [25]. Bundle effectiveness requires comparative evaluation.

Recovery and reassessment

Recovery should be tested against the next disturbance, not declared when case counts or output improve. A multiagency simulation of transboundary disease elimination in wildlife exposed coordination and operational gaps before a real event, supporting exercises as learning tools without proving field effectiveness [26]. Producer research in the United States shows heterogeneous attitudes toward biosecurity and indemnification policy, cautioning that technical plans may meet different behavioral responses [27].

Life-course evidence in dairy cattle further suggests that earlier conditions can shape later resilience, although pathways are heterogeneous and often associative [28]. Reassessment should therefore compare pre-event and post-event biological reserve, immunity distribution, production and welfare trajectories, barrier reliability, detection intervals, response resources, and actor feasibility. **Figure 2** separates apparent recovery from restored reserve and shows why recurrence may reopen an incompletely recovered trajectory rather than start a new one.

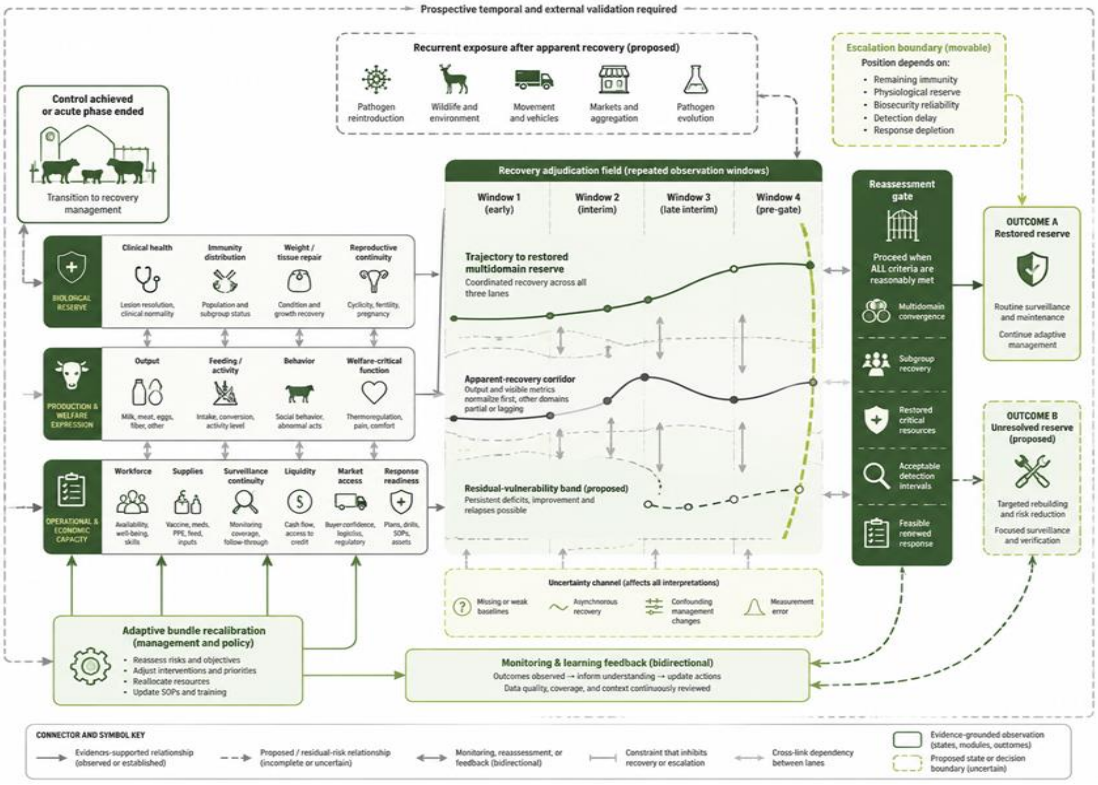

Figure 2. Why restored output can coexist with residual herd vulnerability after outbreak control

Implementation in herd-health programs

Implementation need not treat the architecture as validated. Recurring review can document external pressure, internal amplification routes, immunity heterogeneity, production and physiological trajectories, functioning barriers, detection intervals, response resources, and incomplete recovery. Biosecurity tools differ, so local measurements are not automatically comparable [20]. Surveillance also depends on laboratory, workforce, data, and governance capacity beyond the farm [22].

Each review should identify a binding constraint, plausible next failure, supporting evidence, and revision trigger. Exercises can expose operational gaps [26], while plans should anticipate heterogeneous incentives [27]. This is a structured conversation, not a resilience score. Records should preserve uncertainty, welfare implications, responsibility, and time-stamped decisions. Adoption requires caution when measurement increases workload, consequences discourage reporting, or controls are infeasible.

Validation priorities

Validation must address components and usefulness. Sensor-derived step-count perturbations have potential as dairy resilience indicators, but meaning, reliability, and transportability remain uncertain [29]. Metritis-prediction research shows that preprocessing and modelling choices alter performance, requiring separation of discrimination from clinical utility [30]. Veterinary diagnostic studies also exhibit recurring bias and applicability limitations [31].

Studies should define constructs, time-stamp pressure, state, action, and recovery, compare proposed states with prespecified outcomes, and test calibration across species, pathogens, herds, climates, and services. External validation should precede thresholds. Bundle comparisons should measure welfare, production, epidemiological, economic, and implementation consequences. Missingness, surveillance intensity, management change, and regression to the mean require analysis. The architecture should be revised or rejected if distinctions are unreliable, add no decision value, or worsen inequity and reporting.

Conclusion

Resilient herd health requires managing capacity before its failure becomes visible as uncontrolled disease, production collapse, welfare loss, or delayed recovery. The proposed architecture separates pathogen pressure,

internal amplification, population immunity, physiological reserve, biosecurity, detection, response, and recovery, then reconnects them through time. Its central proposition is that the binding constraint can migrate during an outbreak and that apparent recovery can conceal residual vulnerability. This supports anticipatory reassessment and adaptive intervention bundles, but not a universal score or prescriptive threshold. Species, pathogen, production system, resource, welfare, and governance differences remain decisive. Prospective, externally validated studies must determine whether the architecture improves prediction or decisions and whether its benefits justify measurement and implementation burdens.

Acknowledgments: None

Conflict of Interest: None

Financial Support: None

Ethics Statement: None

References

1. Dogusan MM, De Carvalho Ferreira HC, Moya Durán SJ, Moskalenko L, Brennan ML, De Meneghi D, et al. Behavioural drivers of on-farm biosecurity implementation in livestock: A literature overview with insights for research. Prev Vet Med. 2026;248:106768. doi:10.1016/j.prevetmed.2025.106768
2. Shapiro JT, Cazeau G, DiBiagio R, Dupuy C, Morignat E, Dórea F, et al. Current practice and future directions in syndromic surveillance for animal health: A scoping review and analysis. Prev Vet Med. 2025;241:106532. doi:10.1016/j.prevetmed.2025.106532
3. Poppe M, Mulder HA, Kamphuis C, Veerkamp RF. Between-herd variation in resilience and relations to herd performance. J Dairy Sci. 2021;104(1):616-27. doi:10.3168/jds.2020-18525
4. Rodriguez Z, O'Connor A, Bradford BJ, Picasso-Risso C. Characterization and health, productivity, and economic effects of highly pathogenic avian influenza hemagglutinin type 5 and neuraminidase type 1 outbreak in dairy cattle. J Dairy Sci. 2025;108(6):6349-58. doi:10.3168/jds.2025-26377
5. Caserta LC, Frye EA, Butt SL, Laverack M, Nooruzzaman M, Covaleda LM, et al. Spillover of highly pathogenic avian influenza H5N1 virus to dairy cattle. Nature. 2024;634(8034):669-76. doi:10.1038/s41586-024-07849-4
6. Burrough ER, Magstadt DR, Petersen B, Timmermans SJ, Gauger PC, Zhang J, et al. Highly pathogenic avian influenza A(H5N1) clade 2.3.4.4b virus infection in domestic dairy cattle and cats, United States, 2024. Emerg Infect Dis. 2024;30(7):1335-43. doi:10.3201/eid3007.240508
7. Herraiz C, Triguero-Ocaña R, Laguna E, Jiménez-Ruiz S, Peralbo-Moreno A, Martínez-López B, et al. Movement-driven modelling reveals new patterns in disease transmission networks. J Anim Ecol. 2024;93(9):1275-87. doi:10.1111/1365-2656.14142
8. Jemberu WT, Molla W, Fentie T. A randomized controlled field trial assessing foot and mouth disease vaccine effectiveness in Gondar Zuria district, Northwest Ethiopia. Prev Vet Med. 2020;183:105136. doi:10.1016/j.prevetmed.2020.105136
9. Kiene F, Ganter M, Bauer BU. Exposure of small ruminants to the Schmallenberg arbovirus in Germany from 2017 to 2018—animal-specific and flock-management-related risk factors. Prev Vet Med. 2024;230:106274. doi:10.1016/j.prevetmed.2024.106274
10. Kolb EA, Buterbaugh RE, Rinehart CL, Ensley D, Perry GA, Abdelsalam KW, et al. Protection against bovine respiratory syncytial virus in calves vaccinated with adjuvanted modified live vaccine administered in the face of maternal antibody. Vaccine. 2020;38(2):298-308. doi:10.1016/j.vaccine.2019.10.015
11. Yu Z, Cantet JM, Paz HA, Kaufman JD, Orellano MS, Ipharraguerre IR, et al. Heat stress-associated changes in the intestinal barrier, inflammatory signals, and microbiome communities in dairy calves. J Dairy Sci. 2024;107(2):1175-96. doi:10.3168/jds.2023-23873
12. Polsky L, von Keyserlingk MAG. Invited review: Effects of heat stress on dairy cattle welfare. J Dairy Sci. 2017;100(11):8645-57. doi:10.3168/jds.2017-12651

13. Fujita R, Tachi T, Hino M, Nagata K, Saiki M, Inumaru M, et al. Blowflies are potential vector for avian influenza virus at enzootic area in Japan. Sci Rep. 2024;14(1):10285. doi:10.1038/s41598-024-61026-1
14. Lankester F, Kibona TJ, Allan KJ, de Glanville WA, Buza JJ, Katzer F, et al. Livestock abortion surveillance in Tanzania reveals disease priorities and importance of timely collection of vaginal swab samples for attribution. Elife. 2024;13:RP95296. doi:10.7554/eLife.95296
15. Willkan M, Chapot L, Cameron A, Boldsen SK, Fertner M. Quantification of early detection surveillance in PRRS-free regions. Prev Vet Med. 2025;238:106461. doi:10.1016/j.prevetmed.2025.106461
16. Hutchison J, Mackenzie C, Madin B, Happold J, Leslie E, Zalcman E, et al. New approaches to aquatic and terrestrial animal surveillance: The potential for people and technology to transform epidemiology. Prev Vet Med. 2019;167:169-73. doi:10.1016/j.prevetmed.2018.10.009
17. van den Brink KMJA, Brouwer-Middelesch H, van Schaik G, Lam TJGM, Stegeman JA, van den Brom R, et al. The impact of bluetongue serotype 3 on cattle mortality, abortions and premature births in the Netherlands in the first year of the epidemic. Prev Vet Med. 2025;239:106493. doi:10.1016/j.prevetmed.2025.106493
18. Jean-Pierre RP, Hagerman AD, Rich KM. An analysis of African swine fever consequences on rural economies and smallholder swine producers in Haiti. Front Vet Sci. 2022;9:960344. doi:10.3389/fvets.2022.960344
19. Chanchaidechachai T, Saatkamp H, Inchaisri C, Hogeveen H. Analysis of epidemiological and economic impact of foot-and-mouth disease outbreaks in four district areas in Thailand. Front Vet Sci. 2022;9:904630. doi:10.3389/fvets.2022.904630
20. Duarte F, Tamminen LM, Kjosevski M, Ciaravino G, Delpont M, Correia-Gomes C, et al. Methods to assess on-farm biosecurity in Europe and beyond. Prev Vet Med. 2025;239:106486. doi:10.1016/j.prevetmed.2025.106486
21. Farison F, Lima-Campêlo VR, Paradis ME, Buczinski S, Fecteau G, Roy JP, et al. Farmers who implemented this, also implemented that: Use of association-rule-learning to improve biosecurity on dairies. Prev Vet Med. 2025;239:106516. doi:10.1016/j.prevetmed.2025.106516
22. Kahariri S, Thumbi SM, Bett B, Mureithi MW, Nyaga N, Ogendo A, et al. The evolution of Kenya's animal health surveillance system and its potential for efficient detection of zoonoses. Front Vet Sci. 2024;11:1379907. doi:10.3389/fvets.2024.1379907
23. Sheen JK, Rasambainarivo F, Saad-Roy CM, Grenfell BT, Metcalf CJE. Markets as drivers of selection for highly virulent poultry pathogens. Nat Commun. 2024;15(1):605. doi:10.1038/s41467-024-44777-3
24. Dixon LK, Stahl K, Jori F, Vial L, Pfeiffer DU. African swine fever epidemiology and control. Annu Rev Anim Biosci. 2020;8:221-46. doi:10.1146/annurev-animal-021419-083741
25. Horst EA, Mayorga EJ, Baumgard LH. International Symposium on Ruminant Physiology: Integrating our understanding of stress physiology. J Dairy Sci. 2025;108(7):7675-95. doi:10.3168/jds.2024-25794
26. Chalkowski K, Pepin KM, Lavelle MJ, Miller RS, Fischer JW, Brown VR, et al. Operational lessons learned from simulating an elimination response to a transboundary animal disease in wild animals. Prev Vet Med. 2025;234:106365. doi:10.1016/j.prevetmed.2024.106365
27. Baye RS, Zia A, Merrill SC, Clark EM, Koliba C, Smith JM. Biosecurity indemnification and attitudes of United States swine producers towards the prevention of an African swine fever outbreak. Prev Vet Med. 2024;227:106193. doi:10.1016/j.prevetmed.2024.106193
28. Lewis K, Carter LS, Bradley A, Dewhurst R, Forde N, Hyde R, et al. Quantification of the effect of in-utero events on lifetime resilience in dairy cows. J Dairy Sci. 2024;107(7):4616-33. doi:10.3168/jds.2023-24215
29. Poppe M, Mulder HA, van Pelt ML, Mullaart E, Hogeveen H, Veerkamp RF. Development of resilience indicator traits based on daily step count data for dairy cattle breeding. Genet Sel Evol. 2022;54(1):21. doi:10.1186/s12711-022-00713-x
30. Vidal G, Sharpnack J, Pinedo P, Tsai IC, Lee AR, Martinez-Lopez B. Impact of sensor data pre-processing strategies and selection of machine learning algorithm on the prediction of metritis events in dairy cattle. Prev Vet Med. 2023;215:105903. doi:10.1016/j.prevetmed.2023.105903
31. Downs SH, More SJ, Goodchild AV, Whelan AO, Abernethy DA, Broughan JM, et al. Evaluation of the methodological quality of studies of the performance of diagnostic tests for bovine tuberculosis using QUADAS. Prev Vet Med. 2018;153:108-16. doi:10.1016/j.prevetmed.2017.03.006