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Vaccination Strategy Should Adapt When Pathogen Ecology Changes: A Veterinary Decision Framework Integrating Antigenic Drift, Population Immunity, Exposure Heterogeneity, Host Structure, Disease Severity, Vaccine Performance, and Operational Feasibility

Nora Schneider[1]*, Tobias Frank[1], Andreas Müller[2], Christoph Meier[3]

[1]Department of Veterinary Vaccinology and Adaptive Strategy, Faculty of Veterinary Medicine, University of Freiburg, Freiburg, Germany.
[2]Department of Veterinary Population Immunity and Exposure Heterogeneity, Faculty of Veterinary Medicine, Heidelberg University, Heidelberg, Germany.
[3]Department of Veterinary Vaccine Performance and Operational Feasibility, Faculty of Veterinary Medicine, University of Hohenheim, Stuttgart, Germany.

***E-mail** ✉ nora.schneider@natfak.uni-freiburg.de

ABSTRACT

Veterinary vaccination strategies are often operationalized through predefined products, schedules, target populations, and coverage objectives, yet the biological and epidemiological conditions that originally justified those choices may change. Antigenic evolution can alter vaccine–field strain relationships; immunity can decline or be diluted by population turnover; age, maternal immunity, species, and production structure can modify vaccine responses; exposure may be spatially or demographically concentrated; and clinically meaningful protection may not parallel reduction in infection or shedding. This article develops an original non-empirical adaptive vaccination framework for interpreting these changes without assuming that every surveillance signal warrants immediate strategy modification. The framework separates pathogen change, population immunity, host structure, exposure heterogeneity, disease consequence, vaccine performance, duration of protection, vaccine–field strain match, and operational feasibility before these domains are jointly adjudicated. It further distinguishes booster, reformulation, and targeting decisions rather than treating adaptation as a single intervention. The proposed approach emphasizes noncompensatory constraints: strong performance in one domain should not automatically negate major deficiencies in another, and biological desirability does not establish operational feasibility. Surveillance is therefore positioned as a trigger for structured reassessment rather than as an automatic mandate for action. The framework is intended to improve conceptual consistency in veterinary vaccination decision-making, but it is not a prospectively validated prediction rule, universal schedule, or implementation standard. Its usefulness will depend on disease-specific measurements, locally relevant surveillance, endpoint-specific vaccine evaluation, and external validation across species, production systems, and epidemiological settings.

Keywords: Adaptive vaccination, Antigenic drift, Population immunity, Vaccine effectiveness, Exposure heterogeneity, Veterinary epidemiology

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Introduction

Vaccination is a central instrument of veterinary infectious-disease control, but its performance is not an intrinsic property of a product detached from pathogen, host, endpoint, and setting. A systematic review and meta-analysis

of highly pathogenic avian influenza vaccination in poultry found substantial contextual heterogeneity in reported vaccine efficacy, reinforcing the need to interpret performance against the conditions in which it is measured [1]. In cattle, commercial foot-and-mouth disease vaccines evaluated against East African virus strains showed differences in immunogenicity and persistence, illustrating that nominal vaccine availability does not ensure equivalent durability against locally relevant viruses [2]. Experimental H5 vaccination likewise produced host- and platform-dependent outcomes across chickens, broilers, and domestic ducks [3]. Multivalent H9N2 vaccination studies further demonstrate that humoral, mucosal, cellular, clinical, and shedding-related outcomes represent distinct dimensions of vaccine performance rather than interchangeable indicators [4].

This article therefore treats vaccination strategy as a revisable decision rather than a fixed schedule. It proposes that adaptation should depend on how pathogen evolution, population immunity, demographic replacement, heterogeneous exposure, disease consequence, vaccine effectiveness, duration, strain match, and feasibility interact. The framework does not assign universal thresholds or presume that change in one domain requires intervention; instead, it separates the evidence needed to justify reassessment from the evidence needed to justify a particular response.

Why vaccination strategy cannot remain static

Veterinary vaccination programmes commonly simplify decisions into product choice, interval, and coverage, but these variables operate within biological systems that change over time. Across avian influenza and foot-and-mouth disease studies, vaccine performance varies with vaccine formulation, field-virus relationship, host category, and temporal context [1–3]. A strategy that was defensible when initially adopted may therefore become progressively misaligned even if the vaccine itself remains unchanged.

The relevant question is not whether vaccination should be continually altered, but whether assumptions underlying the current strategy still hold. Declining immune responses, altered circulating strains, demographic replacement, uneven delivery, changing exposure, or different disease consequences can each weaken those assumptions. Conversely, a detected change may be biologically interesting without being decision-relevant. This article therefore proposes a distinction between change detection and strategy revision. Reassessment is warranted when credible evidence challenges a decision-relevant assumption; modification requires a second judgment about whether the change is sufficiently consequential, measurable, and actionable. The framework consequently rejects calendar time alone as a universal reason for revision and also rejects surveillance change alone as sufficient justification for a new vaccine strategy.

Antigenic change

Antigenic change is decision-relevant when pathogen evolution modifies recognition by vaccine-induced immunity, not merely when nucleotide or amino-acid differences accumulate. H9N2 avian influenza surveillance in eastern China identified an emergent clade with substantial antigenic drift, showing that phylogenetic change can coincide with altered antigenic phenotype [5]. Mechanistic evidence from foot-and-mouth disease virus further demonstrates that particular VP1 substitutions can alter neutralization through structural effects on an antigenically important loop [6]. These findings support functional evaluation of evolutionary change rather than assuming that all sequence differences are equivalent.

The clinical or population consequence of mismatch must nevertheless be measured separately. In a comparative H9N2 challenge experiment, a genotype-matched bivalent vaccine improved antibody and shedding-related outcomes against contemporary field strains, while important clinical protection was present in vaccinated comparison groups as well [7]. Thus, improved match may strengthen some outcomes without producing uniform superiority across every endpoint. In this framework, antigenic divergence is therefore a reassessment signal, whereas reformulation requires corroborating evidence that the divergence materially compromises the outcome the programme is intended to protect.

Pathogen evolution

Pathogen evolution supplies the process from which antigenic change may emerge, but evolutionary novelty should not itself be equated with vaccine escape. The H9N2 evidence shows that long-term genetic diversification can be mapped alongside antigenic relationships, allowing particular evolutionary branches to be investigated for functional divergence [5]. Foot-and-mouth disease work demonstrates the converse requirement: experimentally

defined substitutions can be linked to altered neutralization, establishing a functional consequence that sequence surveillance alone cannot provide [6].

This distinction matters because veterinary surveillance increasingly generates genomic evidence faster than vaccine effectiveness can be tested directly. The proposed framework therefore separates three questions: Has the pathogen changed? Has the change altered a vaccine-relevant phenotype? Has that phenotypic change altered clinically or epidemiologically important protection? Evidence can be strong for the first question while remaining weak for the latter two. Evolutionary surveillance is consequently treated as an upstream warning system, not a self-executing vaccine policy. The evidential requirement should rise as decisions become more disruptive, costly, or difficult to reverse, particularly when reformulation could displace a product that remains clinically useful.

Population immunity

Population immunity is not synonymous with the proportion of animals ever vaccinated. It is a changing population state influenced by achieved coverage, persistence of functional protection, demographic turnover, accessibility, and the distribution of susceptible animals. A decade-long canine rabies programme in Beijing illustrates how repeated vaccination, surveillance, and public engagement can operate together to sustain control rather than functioning as isolated interventions [8]. At smaller spatial scales, dog-vaccination data from Tanzania show that geographic barriers can generate markedly uneven access and achieved coverage [9].

Demographic turnover further destabilizes apparent coverage. System-dynamics modeling of African dog populations demonstrated that births, deaths, and replacement can reduce vaccination coverage between campaigns even when the initial campaign performs well [10]. These findings support treating population immunity as a trajectory rather than a static percentage. The framework therefore proposes that coverage estimates be interpreted with time since vaccination, population replacement, spatial distribution, and the biological endpoint being protected. An apparently satisfactory population average may coexist with local pockets of susceptibility, while repeated vaccination may have diminishing strategic value where susceptibility is instead concentrated in newly entering or poorly reached subgroups.

Waning immunity

Waning should be defined against a specified outcome. Declining antibody measurements, loss of challenge protection, increasing clinical susceptibility, and reduced control of infection or shedding are related but non-equivalent phenomena. In foot-and-mouth disease vaccine evaluations, immune responses varied over time and between assays, cautioning against treating a single serological measure as a universal representation of protection [2]. A vaccination programme may therefore face uncertainty even when longitudinal antibody data are available, because the relationship between measured immunity and the programme's intended endpoint may not be fully established.

Population-level decline can also occur without immunological decay within individual animals. In rapidly replacing populations, vaccinated animals may leave while susceptible animals enter, lowering effective coverage through demographic dilution [10]. The proposed framework therefore separates within-animal waning from population replacement. Both can create a rationale for reassessment, but they imply different responses: a booster may address inadequate persistence in previously protected animals, whereas altered timing, age targeting, or delivery frequency may better address newly susceptible entrants. Conflating these processes risks increasing dose frequency without correcting the actual source of population vulnerability.

Host demographic structure

Host structure modifies vaccination through biological maturity, reproductive state, maternal immunity, species, production stage, and replacement dynamics. Experimental classical swine fever vaccination shows that gestational timing and vaccine platform can shape maternally derived neutralizing immunity in offspring [11]. Such evidence argues against treating all animals within a vaccinated population as immunologically equivalent merely because they share a programme label.

Maternal immunity is especially important because it can protect early life while simultaneously modifying responses to subsequent vaccination. In piglets, maternally derived antibodies inhibited immune responses to an F4 enterotoxigenic *Escherichia coli* virus-like-particle vaccine despite robust responses in vaccinated sows [12]. This does not establish universal maternal-antibody interference, but it demonstrates a biologically plausible boundary on age-based scheduling. At the population level, rabies vaccination work in Tanzania further indicates

that continuous or decentralized approaches can respond to rapid dog-population turnover [13]. The framework consequently treats demographic structure as more than descriptive epidemiology: it determines who is susceptible, when susceptibility emerges, whether vaccination is immunologically well timed, and how rapidly protected cohorts are replaced.

Exposure heterogeneity

Equal vaccination coverage does not imply equal exposure or equal residual risk. Geographic access can create unevenly vaccinated populations, as demonstrated in Tanzanian dog-rabies campaigns where spatial barriers influenced achieved reach [9]. Population turnover can compound this heterogeneity when newly susceptible animals accumulate between campaign rounds, creating temporal as well as spatial pockets of vulnerability [13]. Exposure itself may also cluster through movement, contact, production organization, or localized pathogen circulation, although the specific structure and importance of those pathways must be established for the disease and setting under consideration.

The framework therefore keeps delivery heterogeneity separate from exposure heterogeneity. A poorly reached subgroup may be underprotected even if exposure is ordinary; a heavily exposed subgroup may remain disproportionately vulnerable despite acceptable vaccination uptake. A population average can obscure both states. **Table 1** presents how geographic access, population turnover, and contact intensity create hidden vaccination gaps.

Table 1. How geographic access, population turnover, and contact intensity create hidden vaccination gaps

Hidden subgroup	Why population coverage misleads	Turnover or exposure evidence	Targeting change	Separate map to refresh
Geographic protection gap	Coverage average masks poorly reached locations	**Conditions:** Distance, density, delivery access **Observed:** Persistent spatial coverage differences	Consider geographic retargeting	**Distinction:** Access does not prove higher exposure **Refresh:** Re-map reach and susceptibility **Basis:** Evidence-derived
Turnover-driven susceptibility pocket	New entrants dilute prior population protection	**Conditions:** Births, deaths, replacement rate **Observed:** Increasing unvaccinated young or new animals	Reconsider timing or delivery frequency	**Distinction:** Distinct from immune waning **Refresh:** Reassess between campaign rounds **Basis:** Evidence-derived
High-exposure subgroup	Similar coverage may yield unequal residual risk	**Conditions:** Contact intensity, production role, movement **Observed:** Clustered exposure or infection indicators	Consider targeted protection	**Distinction:** Requires disease-specific exposure evidence **Refresh:** Reassess subgroup risk **Basis:** Proposed synthesis
Delivery–exposure discordance	Lowest coverage coincides with greatest exposure	**Conditions:** Geography plus contact structure **Observed:** Overlap of access gaps and risk signals	Prioritize targeted intervention	**Distinction:** Joint pattern must be demonstrated **Refresh:** Reassess both reach and exposure **Basis:** Proposed synthesis

Disease severity and consequence

Vaccination strategy should reflect not only the probability of infection but also what infection means in a particular host and production context. In a PCV2d/PRRSV co-challenge model, homologous and heterologous PCV2 vaccination were evaluated under concurrent infection pressure, illustrating that observed vaccine performance is conditioned by the disease environment in which protection is tested [14]. H5 avian-influenza vaccine-trial synthesis similarly indicates that prevention of severe disease and reduction of viral shedding are distinct outcomes and should not be treated as interchangeable measures of programme success [15].

Consequences also extend beyond clinical signs and mortality. A peste des petits ruminants outbreak in northwest Ethiopia produced substantial production and economic losses for affected smallholders, demonstrating that vaccination decisions may legitimately incorporate livelihood and production consequences alongside biological severity [16]. These effects are context-specific and should not be transferred numerically between regions or diseases. The proposed framework therefore separates infection risk, clinical severity, transmission-related consequences, and production or economic consequence. Adaptation becomes more defensible when deterioration in vaccine performance affects an outcome that is consequential in the population being protected, rather than merely altering a laboratory marker of uncertain practical importance.

Vaccine effectiveness

Vaccine effectiveness must be defined against the outcome that matters for the programme rather than treated as a single scalar property. Experimental and review evidence already considered shows that prevention of clinical disease, reduction of infection, suppression of viraemia or shedding, and maintenance of population control can diverge. The practical implication is that a vaccine can remain valuable for one objective while becoming less adequate for another. For adaptive decision-making, effectiveness should therefore be specified by endpoint, host group, circulating pathogen context, and time since vaccination.

This distinction also protects against misclassifying partial protection as complete failure. Under coinfection or heterologous challenge, observed protection may differ from performance under homologous or simplified experimental conditions [14]. The proposed framework consequently asks whether the outcome currently deteriorating is the outcome the programme was designed to protect. A decline in a laboratory measure of uncertain relation to disease or transmission should prompt investigation, whereas deterioration in a validated, decision-relevant endpoint has greater actionability. No universal effectiveness threshold is proposed. This endpoint discipline is especially important when vaccination changes disease expression without eliminating infection, because programme success then depends on whether the protected outcome is individual health, production continuity, transmission reduction, or some combination of these objectives.

Duration of protection

Duration is not a fixed attribute transferable across vaccine products, serotypes, hosts, or endpoints. Evaluation of a multivalent foot-and-mouth disease vaccine in cattle demonstrated time-dependent variation in measured immunity, supporting explicit consideration of how long a given vaccination strategy sustains its intended biological effect [17]. Adenovirus-vectored foot-and-mouth disease vaccination has likewise been assessed with both humoral measurements and challenge protection over time, emphasizing that persistence of an immune marker and persistence of protection are not identical questions [18].

Regional field-oriented studies in Georgia, Azerbaijan, and Armenia further showed that neutralizing responses after foot-and-mouth disease vaccination can change materially across a six-month observation interval [19]. These data support longitudinal reassessment but do not establish a universal booster interval. In the proposed architecture, duration should therefore be indexed to a stated endpoint and setting. Booster decisions become more defensible when declining protection is demonstrated in the relevant host population, rather than inferred solely from elapsed calendar time. Where only surrogate measurements are available, uncertainty about their relation to protection should remain explicit rather than being converted into an assumed duration.

Vaccine–field strain match

Vaccine–field strain match should integrate genetic surveillance with functional evidence whenever feasible. H9N2 evolution, experimentally demonstrated foot-and-mouth disease antigenic substitutions, and challenge evidence with genotype-matched H9N2 vaccination collectively show that sequence change, antigenic phenotype, and protection-related outcomes are connected but not interchangeable [5–7]. Functional neutralization or challenge evidence therefore carries a different inferential role from sequence distance alone.

Match should also remain one domain within a broader decision. A better-matched vaccine may improve antibody or shedding outcomes without necessarily producing superior results for every clinical endpoint [7]. Conversely, imperfect match does not automatically imply that an existing vaccine has lost all utility. The proposed framework therefore treats match as a conditional constraint: demonstrated mismatch increases the need for reassessment, but reformulation should depend on whether mismatch affects the programme's intended outcome, whether alternative products improve that outcome, and whether changing formulation is operationally and epidemiologically justified.

Operational feasibility

A biologically preferable vaccination strategy may still fail to achieve meaningful population coverage if its delivery requirements do not fit the production or community setting. Rural–urban rabies vaccination experience in Zambia shows that accessibility, settlement pattern, and campaign design can alter programme reach [20]. Comparative pilot work in Uganda similarly indicates that different delivery strategies can achieve different operational profiles and coverage patterns [21]. Qualitative evidence from rural Uganda further identifies

awareness, dog handling, workforce, resource, and communication constraints that can limit mass vaccination [22].

Operational feasibility should therefore be treated as a decision domain rather than a downstream implementation detail. The relevant question is not whether a strategy is theoretically deliverable, but whether its required timing, workforce, cold chain, animal handling, owner participation, surveillance support, and geographic reach can be sustained in the intended setting. **Table 2** presents delivery bottlenecks that separate biological vaccine failure from implementation failure.

Table 2. Delivery bottlenecks that separate biological vaccine failure from implementation failure

Delivery bottleneck	Implementation resource that fails	Observed delivery failure	Strategy redesign	Infrastructure and evaluation need
Reach-limited delivery	**Meaning:** Eligible animals remain inaccessible **Conditions:** Distance, density, ownership, handling	Persistent missed subgroups	Redesign delivery channel	**Limit:** Access may vary by campaign **Recheck:** Reassess geographic reach **Basis:** Evidence-derived
Capacity-limited delivery	**Meaning:** Strategy exceeds available personnel or logistics **Conditions:** Workforce, cold chain, timing	Delays, incomplete rounds	Simplify or phase delivery	**Limit:** Capacity can change rapidly **Recheck:** Reassess before each cycle **Basis:** Proposed synthesis
Participation-limited delivery	**Meaning:** Uptake is constrained despite service availability **Conditions:** Awareness, trust, handling	Repeated refusal or nonattendance	Strengthen communication and access	**Limit:** Motives are context-specific **Recheck:** Reassess community response **Basis:** Evidence-derived
Surveillance-limited adaptation	**Meaning:** Revision cannot be evaluated reliably **Conditions:** Diagnostics, reporting, follow-up	Incomplete post-vaccination evidence	Delay or narrow adaptive claims	**Limit:** Absence of signal may reflect weak detection **Recheck:** Reassess evidence infrastructure **Basis:** Proposed synthesis

A proposed adaptive vaccination architecture

The article proposes an adaptive architecture in which vaccination strategy is reconsidered through several analytically separate domains rather than by a single trigger. Pathogen change, population immunity, host structure, exposure heterogeneity, disease consequence, vaccine effectiveness, protection duration, field-strain match, and operational feasibility are first evaluated independently. They are then brought into a central adjudication step that asks whether the current strategy remains aligned with its intended outcome.

The architecture is deliberately noncompensatory. Strong performance in one domain should not automatically cancel a critical weakness in another. High nominal coverage, for example, cannot resolve a severe geographic protection gap, and good clinical protection cannot be assumed to eliminate transmission-related risk. Operational infeasibility can also constrain an otherwise biologically attractive strategy [20]. The resulting decision remains revisable rather than final: surveillance can strengthen, weaken, or redirect the evidence supporting continued use, boosting, reformulation, targeting, or combinations of these responses.

Figure 1 compresses this logic by separating evidence-supported inputs from the proposed adjudication and feedback relationships, while preserving explicit uncertainty and validation boundaries.

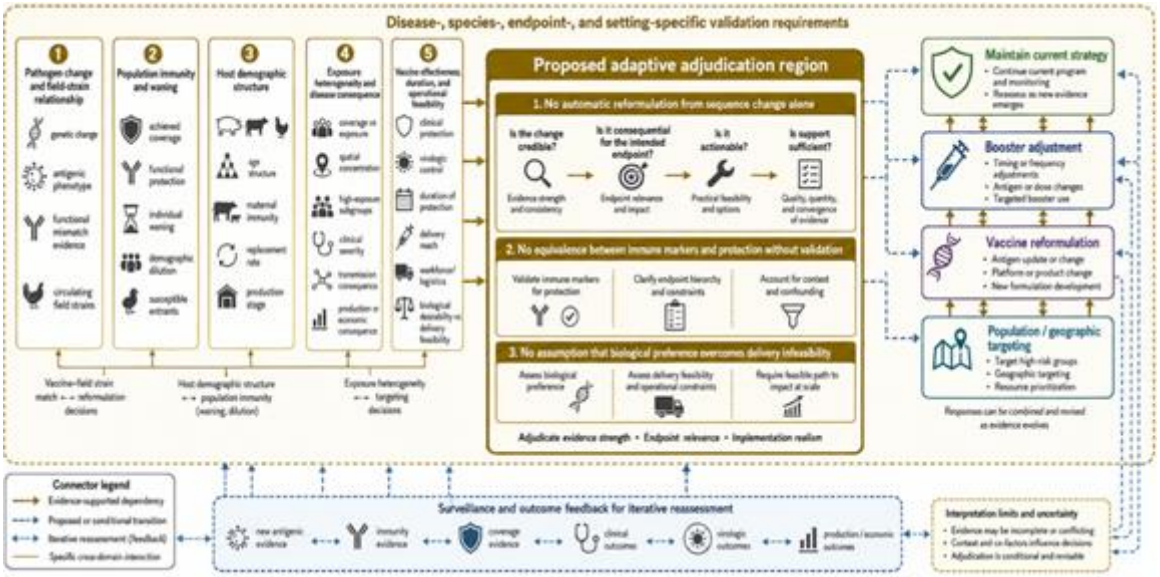

Figure 1. Conditional adjudication of changing vaccine fit across pathogen, host, population, and delivery states

Surveillance-triggered strategy revision

Surveillance should trigger structured reassessment when it detects a credible change in an assumption underlying the current vaccination strategy. Spatiotemporal modeling of classical swine fever in Japan demonstrates how surveillance-informed risk estimation can help delimit where vaccination may be most relevant geographically [23]. For H9N2 influenza, antigenic-landscape modeling has been used to identify candidate vaccine-strain recommendations, showing how surveillance can prioritize hypotheses for vaccine updating [24]. In vaccinated poultry populations, surveillance must also be capable of detecting infection and potential vaccine escape because vaccination changes what can be inferred from clinical observation alone [25].

These examples support surveillance as an information-generating component, not an automatic decision rule. The proposed architecture therefore requires escalation from signal detection to confirmation of consequence and actionability. A genomic or antigenic signal may justify targeted phenotyping; a spatial risk signal may justify retargeting; and evidence of infection under vaccination may justify investigation of match, duration, or delivery. The strength of action should remain proportional to the strength and relevance of the evidence. Repeated or concordant signals across antigenic, epidemiological, immunological, and field-performance data should carry greater interpretive weight than an isolated anomaly, while discordant signals should open an uncertainty pathway rather than being forced into a predetermined action.

Booster, reformulation, and targeting decisions

Booster, reformulation, and targeting decisions address different failure mechanisms and should not be treated as interchangeable responses. Booster adjustment is most coherent when protection in previously vaccinated animals declines over the relevant interval; regional foot-and-mouth disease studies illustrate why persistence must be examined over time rather than assumed [19]. Reformulation is more coherent when surveillance suggests antigenic divergence that is subsequently supported by functional evidence; predictive antigenic models can prioritize candidate updates but do not themselves demonstrate field benefit [24]. Targeting is more coherent when susceptibility or exposure is unevenly distributed and delivery strategies can be redirected toward the affected subgroup [21].

The proposed framework therefore matches the response to the mechanism. A booster is unlikely to correct geographic nonaccess, reformulation does not solve low uptake, and broad population revaccination may be inefficient when risk is spatially concentrated. Combined responses may sometimes be warranted, but only when the relevant mechanisms coexist. No universal hierarchy among the three options is proposed. The same surveillance event can therefore justify different responses in different populations because the decisive mechanism may be waning in one setting, mismatch in another, and concentrated susceptibility or access failure in a third.

Vaccine failure and immune escape

Vaccine failure is not a single state. It may refer to failure to prevent clinical disease, infection, viraemia, shedding, transmission, or population-level persistence, and these endpoints can diverge. Experimental redesign of a PRRSV-1 vaccine has produced broader heterologous immune responses and enhanced cross-protection, indicating that formulation can influence breadth of protection [26]. Comparative challenge of three commercial PRRS modified-live vaccines also demonstrated differences in viraemia and nasal shedding after heterologous challenge [27].

Cross-genotype PRRS vaccination provides an especially important boundary: clinical outcomes may improve while control of viral load or shedding remains incomplete [28]. Such discordance should not be mislabeled as either complete success or complete failure. In the proposed architecture, "immune escape" is reserved for evidence that pathogen variation compromises relevant vaccine-induced immunity, whereas "vaccine failure" is indexed to the specific endpoint that is not adequately protected. This prevents clinical benefit from being mistaken for transmission blocking and prevents residual shedding from erasing meaningful disease protection. Apparent failure should also prompt examination of administration, timing, coverage, host immunity, and exposure intensity before pathogen escape is assigned as the explanation.

Implementation priorities

Implementation should begin with the minimum infrastructure required to know whether adaptation is succeeding. Delivery systems must be capable of reaching the intended animals, and field experience shows that rurality, accessibility, density, and campaign design can materially affect that reach [20]. Community-level barriers such as awareness, communication, handling difficulty, personnel, and resource constraints must likewise be addressed because they can limit vaccination even when biological rationale is strong [22]. Adaptive strategies additionally require surveillance capable of interpreting infection in vaccinated populations and detecting relevant change [25]. The practical priority is therefore not maximal complexity but minimum viable adaptiveness: reliable coverage measurement, clearly defined outcome endpoints, suitable diagnostic or surveillance capacity, responsibility for reassessment, and feasible mechanisms for changing schedule, product, or targeting when evidence warrants it. These requirements remain context-dependent. The framework does not establish that every veterinary programme needs identical surveillance intensity or governance arrangements, nor does it demonstrate that adaptive management will necessarily outperform a well-designed fixed strategy in every setting. Implementation should therefore preserve a feasible fallback strategy when evidence is incomplete, rather than making programme continuity dependent on data streams that cannot be maintained.

Validation requirements

Validation must occur at more than one level. Candidate biological measurements require evidence that they predict the outcome they are intended to represent. In foot-and-mouth disease, an IgG1 avidity ELISA predicted heterologous challenge protection within a defined dataset, illustrating the potential value of a candidate immune correlate while also requiring external validation before universal use [29]. PRRS studies similarly show that immune correlates can vary with heterologous strain and outcome [30]. Genetic relatedness also cannot be assumed to establish functional vaccine matching without serological or phenotypic confirmation [31].

The broader architecture requires an additional level of testing. Immunogenicity may be demonstrable while clinical effectiveness remains unresolved, as illustrated by an autogenous *Streptococcus suis* maternal-vaccination study in which clinical-event scarcity limited inference about protection [32]. Candidate correlates, genetic match measures, and predictive assays therefore should not be presumed universal surrogates [29–31]. Prospective multi-setting studies should test whether the proposed architecture improves decision consistency or outcomes compared with existing practice, while allowing the framework to be revised or rejected when its assumptions fail. Validation should include temporal recalibration, species and production-system external validation, comparison of alternative decision rules, predefined clinical and virologic outcomes, and explicit tests of whether proposed noncompensatory boundaries improve decisions or merely add complexity.

Conclusion

Veterinary vaccination strategy should be treated as conditionally revisable because pathogen ecology, immunity, host structure, exposure, disease consequence, vaccine performance, and delivery conditions can change

independently and at different rates. The framework developed here separates those domains before combining them in a proposed adjudication process, thereby avoiding automatic responses to isolated genomic, serological, clinical, or operational signals. It also distinguishes booster, reformulation, and targeting as responses to different mechanisms of reduced vaccine fit. The central contribution is therefore a structured way to ask when a previously defensible vaccination strategy may no longer align with its intended endpoint. The framework remains non-empirical and disease-dependent; it requires prospective testing, endpoint-specific validation, locally relevant surveillance, and explicit attention to feasibility before it could support operational decision rules. Its value is therefore presently conceptual and testable rather than established.

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