# Closure in the Pair

## Avian Influenza Read Through the Vacuum.Net Theory

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## Abstract

Influenza resolves in most hosts without any prior specific immunity, and vaccination does not occur in nature. Protection at a first encounter therefore cannot rest on memory, and an account of avian influenza built on immunity as a stored quantity is incomplete at its foundation.

This essay reads the problem through the Vacuum.Net theory, in which closure is a property of the whole host-guest pair rather than of either member. Three findings follow. First, the pair reading is supported: the same clade runs in two bird species with opposite outcomes, and the difference is demonstrably on the host side. Second, an account proposed in the course of this work — that resolution follows from confinement of the path to a surface that discharges outward — is falsified by the evidence, and the falsification is reported here rather than removed. Third, what the evidence supports in its place is a timing relation rather than a geometry, and that step is formulated but not yet verified, for reasons stated in section 9.

The essay also separates two questions that current policy runs together: where a non-closing form is produced, and what is done once it appears. The measures in force act almost entirely on the second.

## 1. The question the standard frame does not hold

An ordinary influenza runs for about a week and is over, in a host that had no specific immunity to that strain beforehand. Nothing was stored in advance. Whatever brings the episode to an end is present at the first encounter, and it is present in a wild duck as much as in a person.

This is not a marginal observation. It removes the ground from the usual framing of the avian influenza problem, in which the question is how much immunity is present and how to raise it. Vaccination does not occur in nature, and the natural case is the one that resolves most reliably. An account that explains resolution by stored immunity explains the uncommon case and leaves the common one unexplained.

The question this essay takes up is therefore narrow and prior to policy: what brings an episode to an end when nothing was stored, and under what conditions does that fail.

Two facts frame the failure. In the Netherlands, three outbreaks in three days at the end of October 2026 led to a national housing order; the first, at a laying farm in Lutjegast on 26 October, involved about 45,000 birds. Globally, clade 2.3.4.4b now runs in wild birds, in poultry, and in a widening set of mammals, with 1,177 United States dairy herds in 20 states recorded by late September 2026. The episodes that do not end are the whole of the problem.

## 2. The vocabulary, applied

The Vacuum.Net theory is used here in its positional form: points in a position space, curves running between them, and connection points at which curves meet and a path can close. No physical claim is carried over. What is carried over is a single constraint on what may count as an explanation.

The constraint is that closure is a property of a whole path, not of a point on it. A point can be measured; closure cannot be read off any measurement at a point, however precise, because precision at a point says

nothing about whether the circuit returns. Applied to a host and an infection, this has an immediate consequence: whatever ends an episode is a property of the pair, and no property of the guest alone and no property of the host alone can carry it.

That constraint is also what forbids the vocabulary in which this subject is normally written. Phrasings in which a virus evades, hijacks or outwits, and in which a system defends or fights, place the explanation in an agent at a point. Nothing in what follows uses them, and the avoidance is not stylistic: a path has no intentions, and an account written in intentions cannot be checked.

The working definition of closure used throughout is the one developed earlier in this programme for tuberculosis immunity. Take a defined perturbation at time zero and measure two compartments. The transient compartment holds what rises on contact and is expected to subside. The registering compartment holds what remains changed afterwards. A path has closed when the transient, having been displaced, returns within a characteristic time to within a defined neighbourhood of its prior values, and the registering compartment carries a stable offset. A path has failed to close when the transient does not return.

Two quantities follow directly: the fraction of the displacement recovered, and the time over which recovery occurs. Both require at least two time points. Neither can be read from a single cross-section. That is the whole of the difference this reading claims over an account built on measured components.

## 3. The material

Five bodies of measurement carry the argument. Each is stated as measured, before any reading is placed on it.

**Replication in mallards.** In experimentally infected mallards, low-pathogenic influenza A replicates in the surface epithelium of the gastrointestinal tract, concentrated at the tips of the intestinal villi, with the colon and ileum carrying the most positive cells. Virus was present in cloacal swabs from all birds at one and two days after infection. No clinical signs occur, and the replication proceeds without gross or microscopic lesions (Vet Res 2018).

**The duck-chicken comparison.** A transcriptome comparison of ducks and chickens infected with H5N1 and with H5N2 found the two species share almost nothing in their early response: two genes commonly raised in the ileum at one day, 68 in the lung at three days. Chickens lack RIG-I (DDX58) and its binding partner RNF135 in their genome and showed almost no IFITM response. Ducks raised IFITM1, IFITM2 and IFITM3 strongly against H5N1, with TLR and RIG-I pathway activity in both lung and ileum. In chickens, H5N1 produced an early ileal response at one day that did not last, and the birds died; H5N2 produced a sustained lung response at three days, dominated by T-cell and B-cell genes, and the birds survived (BMC Genomics 2015).

**The definitional position.** The WOAH Terrestrial Animal Health Code states that low-pathogenicity viruses, "especially H5 and H7 subtypes, have the potential to mutate into high pathogenicity avian influenza viruses", and makes a sudden and unexpected increase in virulence of low-pathogenicity viruses in poultry notifiable as an emerging disease. The high-pathogenicity form is thus defined, in the governing instrument, as something that appears in poultry (WOAH TAHC ch. 10.4).

**Wild bird mortality.** In the 2020–2023 panzootic, colonial breeding birds formed the main mass-mortality group. Northern gannets were among the worst affected: roughly 75% of North Atlantic colonies involved in summer 2022, a 70% reduction in colony size in Scotland, adult mortality above 58% in France, 11.5% population loss in eastern Canada. The review links coloniality to both mortality and persistent circulation (npj Biodiversity 2025).

**Current status.** Clade 2.3.4.4b is dominant worldwide, with 624 high-pathogenicity events in 25 countries reported in a single month to late September 2026, 1,177 United States dairy herds in 20 states, and infections recorded in goats, swine, sheep, seals, sea lions, dolphins, foxes and mustelids (FAO). Human cases stand at 1,003 with 479 deaths across 25 countries since 2003, and WHO holds that sustained human-to-human transmission is currently unlikely (WHO, 2 October 2026).

## 4. An account, and its falsification

The first account built on this material located closure in the geometry of the opening. It ran as follows. An infection confined to a surface that faces outward, discharges outward and renews itself comes to an end by construction: the material leaves, the surface is rebuilt, the path has nowhere further to run. The mallard data fit exactly — villus-tip epithelium, cloacal shedding, no lesions at all. Ordinary human influenza fits on a different surface. High-pathogenicity infection in gallinaceous birds runs through endothelium and inner organs, which face inward and do not discharge outward, and therefore has no exit; the path can only run, and ends with the host.

The account was attractive because it covered a second case from this programme. A tuberculosis granuloma is an opening with no outlet, and it stands unresolved for decades. One rule appeared to cover both: a path closes when its discharge has somewhere to go.

The account is false. In the duck-chicken comparison, H5N1 in ducks produced a strong response in both lung and ileum. The infection in the duck is therefore not confined to an outward-discharging surface, and the duck comes through it. A host that resolves an unconfined infection refutes the claim that confinement is what resolves it.

This is recorded here rather than quietly replaced, for two reasons. The step is instructive: the account was built from facts that were individually correct, and the error lay in which property of the pair was identified as decisive, not in the material. And a reading that is only ever reported after it survives cannot be judged for how often it fails.

## 5. What the evidence supports instead

The duck-chicken comparison puts the difference on the host side and names components: RIG-I and RNF135 absent from the chicken genome, and an IFITM response present in one species and nearly absent in the other. This is a component account, and the authors of that work state its limit themselves — the IFITM role is inferred from transcriptome and sequence data, not tested in vivo.

A component account is also insufficient on its own, and the same dataset shows why. The chicken lacks RIG-I in every infection, yet the chicken resolves H5N2 and dies of H5N1. Absent gene content cannot explain an outcome that varies while the gene content does not. The difference again sits in the pair.

What varies between those two chicken cases is not whether a response occurs but whether it carries through. Against H5N1 the response rose at one day in the ileum and did not last. Against H5N2 it developed over three days and held. In the terms of section 2, the first is a transient that was displaced and did not return; the second is a transient that ran its course. Non-closure here is a response begun and not completed.

That suggests the decisive property is a relation between two rates: how fast the infection reaches a state from which no response can retrieve it, against how long a response takes to develop. On this reading the host components matter because they shorten the second, and the high-pathogenicity form matters because it shortens the first. Neither is decisive alone; their ratio is.

The four pairs in the data arrange consistently on that reading.

| Pair | Time to unretrievable spread | Time to a developed response | Outcome |
|---|---|---|---|
| Duck, low-pathogenicity | Long | Barely called on | Closes |
| Duck, high-pathogenicity | Short | Short (RIG-I, IFITM present) | Closes |
| Chicken, low-pathogenicity | Long | About three days | Closes |
| Chicken, high-pathogenicity | Short | About three days | Fails |

The reading also accounts for the cases that would otherwise be anomalies. Ducks are not universally spared by clade 2.3.4.4b; waterfowl mortality has been recorded since 2021. On a ratio account that is expected wherever dose, route, age or concurrent load moves either rate far enough to invert the inequality, and it requires no special pleading.

**Status of this step.** It is formulated, consistent with every case in the material, and not verified. Verification requires the experimental literature on replication efficiency and the early course of innate responses in chickens, which could not be retrieved in the course of this work (section 9). Until that is read, the ratio account stands as the best available reading of the data assembled here and not as an established result.

## 6. Where the non-closing form is produced

A second question sits behind the first and is usually merged with it. Granted that some pairs do not close, where does the member that cannot be closed come from?

The governing instrument answers plainly. Low-pathogenicity H5 and H7 viruses have the potential to become high-pathogenicity ones, and the event of a sudden increase in virulence is defined and made notifiable as something occurring in poultry. The form that does not close is not what arrives from the wild. It is what a particular configuration makes of what arrives.

Three features of that configuration bear on it, and they can be stated as quantities rather than as descriptions.

1. **Passage length.** The number of successive transmissions within a population before the chain ends. A configuration that supports long passage gives many opportunities for the form to change; one that ends chains quickly gives few.
2. **Route diversity.** The number of distinct paths to the same connection point. A commercial flock is one genome in tens of thousands of copies, and is one route.
3. **Carry.** Whether a registering offset persists in the population. A bird slaughtered at about six weeks carries nothing forward; a laying flock can.

Age synchrony compounds all three: where a flock is of one age, a perturbation meets every animal at the same point of its course.

The reading is testable outside the stall, and the test is already run. If the configuration rather than the taxon is decisive, wild populations should show the stall pattern exactly where their structure resembles a stall. Colonial breeders are the main mass-mortality group in the panzootic, and the same review links coloniality to persistent circulation. Dispersed waterfowl do not show that pattern. A breeding colony is a natural stall, and behaves like one.

The same reading covers the mammalian cases without extension. Dairy herds combine density with a shared milking process that shortens the interval between exposures. Fur farms combine density, uniformity and naivety, and are repeatedly where signals of mammalian adaptation appear. On a configuration account that is not coincidence but the expected location.

## 7. The measures in force, read through this

The Dutch package of 28 October 2026 comprises a national housing order for commercially kept birds, a screening requirement for species that cannot be housed, a national exhibition ban, a tightened visitor restriction, and culling of affected flocks. A plan to make vaccination of laying hens compulsory is due with the Tweede Kamer at the end of the year.

**Culling** acts on none of the three quantities. It removes the host and leaves the configuration, which is the deepest available discharge with carry set to zero. Recurrence is then not a risk but a consequence, and the annual pattern of Dutch outbreaks is what that consequence looks like.

**Housing orders** act on the rate of introduction at the boundary, and only while in force. They are a rate measure of real value and no structural effect; they move the date of the next episode rather than its likelihood.

**Vaccination** requires a correction to an earlier version of this reading. Vaccination installs a registering offset in advance, and on a memory account that is the one measure addressing the deficit directly. On the account reached in section 5, it is not. Closure at a first encounter does not rest on a stored offset, so installing one does not supply what is missing. What vaccination does supply is a shortening of the time to a developed response, which on the ratio account is a genuine effect — but it does so against the challenge it was built for, through one route in a population that has one route.

Two consequences follow, and they point in opposite directions. Vaccination is better founded than a purely memory-based rationale would make it, because its effect falls on the rate that matters rather than on stored quantity. And it is more limited than that rationale suggests, because reduced clinical signs in a flock whose passage continues means the configuration keeps operating while it is harder to observe.

The measure that acts on the cause rather than the episode is passage length. Everything that shortens it — flock size, detection interval, separation of waterfowl from gallinaceous poultry, and the total number of birds held at passage-supporting density — addresses where the non-closing form is produced. Nothing currently in force does.

## 8. What would refute this

Three tests are available, and each can return an answer that ends the reading.

**The timing measurement.** Apply a standardised perturbation and record the transient compartment at a minimum of two time points, in both species and against both virus forms. Recover the two quantities of section 2: fraction of displacement recovered, and time to recovery. The ratio account requires that the failing pair be the one in which spread outruns response development. If the failing pair recovers on the same timescale as the surviving ones, the account is wrong. The measurement needs no new instrumentation.

**The sequence record.** The configuration account requires that high-pathogenicity forms appear first where passage is sustained. Take each episode in which such a form appeared and locate the earliest detection: poultry or dispersed wild birds. The record as it stands points to poultry, and the lineage itself began there. An episode that begins cleanly in a dispersed wild population is a refutation. This test is run against existing archives and was not performed here.

**The population structure prediction.** Within wild birds, mass mortality and persistent circulation should track colony structure and not taxonomy. This test has effectively been run and the reading survives it, but it will continue to be run by every subsequent season, and a season in which dispersed species carry the mortality would count against it.

A fourth claim, from an earlier stage of this work, should be recorded as withdrawn rather than tested. It proposed a specific inequality between re-exposure interval and a return time in dense flocks. The return time has no measured value, so the inequality has no content as it stands, and it is superseded by the ratio formulation of section 5.

## 9. Limits, and what is not verified here

The reading gives a direction and not a set of values. Every quantity named in sections 5 and 6 — passage length, route diversity, carry, the two rates — is in principle measurable and none is measured here.

Four gaps are specific and should not be read past.

The experimental work on replication efficiency against the early course of innate responses in chickens, which is where the ratio account of section 5 would be confirmed or broken, could not be retrieved. The same applies to the pathogenesis work on clade 2.3.4.4b in mallards, which bears on how far ducks remain a closing pair. Both are named in the references and marked as not read.

The panzootic review was available only as a truncated excerpt, so its figures are reported as given and its full argument is not represented. The global situation page was read in part, about two thirds of its length.

The detection of high-pathogenicity influenza in captive mink in Utah is reported here through trade press citing the United States Department of Agriculture, not from the department directly. Claims circulating in late October 2026 about a specific mutation recovered from that farm could not be confirmed in any official source and are excluded from this essay entirely.

Finally, the economic question is untouched. Density, uniformity and short turnover are the three features this essay identifies as the site of the problem, and they are also the basis on which the sector operates. An account that names them without addressing that is a diagnosis, not a plan.

## Annotated references

Read and used, with what each carries and what weight it bears.


4. **Alternate routes of influenza A virus infection in Mallard (Anas platyrhynchos)**, Veterinary Research, 2018. Experimental infection of mallards; replication localised to gastrointestinal surface epithelium at villus tips, cloacal shedding at one and two days, no clinical signs and no lesions. *Carries section 3 and the premise of section 4. Primary experimental work; the tissue localisation is directly observed. Its own tissue analysis did not cover the respiratory tract, so it establishes where replication was found and not where it was absent.*

5. **A comparative analysis of host responses to avian influenza infection in ducks and chickens highlights a role for the interferon-induced transmembrane proteins in viral resistance**, BMC Genomics, 2015. Transcriptome comparison across two species and two virus forms; absence of RIG-I and RNF135 from the chicken genome; IFITM1/2/3 response in ducks and near-absence in chickens; the chicken response to H5N1 raised at one day and not sustained, the response to H5N2 developed by three days with survival. *The single most load-bearing source here. It falsifies section 4 and supplies the within-species contrast that drives section 5. The*

*authors state that the IFITM role is inferred from transcriptomic and sequence evidence rather than tested in vivo, and that caveat is carried into this essay.*

6. **Infection with avian influenza viruses, Terrestrial Animal Health Code, chapter 10.4**, WOAH. Definitional position; low-pathogenicity H5 and H7 viruses have the potential to become high-pathogenicity; a sudden and unexpected increase in virulence in poultry is notifiable as an emerging disease. *Carries section 6. This is a regulatory instrument rather than a research finding: it records the settled position of the veterinary authorities and is strong evidence of consensus, not independent evidence of mechanism.*

7. **The role of wild birds in the global highly pathogenic avian influenza H5 panzootic, 2020–2023**, npj Biodiversity, 2025. Colonial breeders as the main mass-mortality group; gannet figures by region; coloniality linked to mortality and to persistent circulation. *Carries the wild-population test in section 6. Read as a truncated excerpt only — figures are reported as given and the full argument is not represented here.*

8. **Global AIV with Zoonotic Potential**, FAO, update of 24 September 2026. Circulating clades; 624 high-pathogenicity events in 25 countries in one reporting month; 1,177 United States dairy herds in 20 states; the mammalian host list. *Status reporting for section 3. Read in part, roughly two thirds of its length.*

9. **Avian Influenza Weekly Update**, WHO, 2 October 2026. 1,003 human cases and 479 deaths in 25 countries since 2003, through 6 September 2026; pandemic risk unchanged; sustained human-to-human transmission currently unlikely. *Human figures in section 3. Note that the case fatality implied by these totals reflects surveillance weighted toward severe presentation and should not be read as a per-infection rate.*

10. **Landelijke ophok- en afschermplicht ingesteld na vogelgriepuitbraken** and **Vogelgriep vastgesteld in Lutjegast**, Rijksoverheid, 26 and 28 October 2026. The three outbreaks, the 45,000-bird laying farm, the composition of the national measures, and the compulsory-vaccination plan due at year end. *Carries section 7. Primary announcements from the responsible ministry.*

11. **Association of RIG-I with innate immunity of ducks to influenza**, PNAS, 2010. The original identification of RIG-I in ducks in relation to influenza. *Cited for provenance of the component in reference 2. Abstract-level only; the full text was not retrieved.*

12. **Differential innate responses of chickens and ducks to low-pathogenic avian influenza**, Avian Pathology, 2012. Companion comparative work on the two species against the low-pathogenicity form. *Listed as corroborating context for reference 2. Not read in full.*

**Named and not read.** Two sources would decide section 5 and could not be retrieved in this work. They are listed so the gap is addressable rather than invisible: *Association of Increased Pathogenicity of Asian H5N1 Highly Pathogenic Avian Influenza Viruses in Chickens with Highly Efficient Viral Replication Accompanied by Early Destruction of Innate Immune Responses*, Journal of Virology, 2009 — which by its title addresses precisely the relation between replication rate and the collapse of an initiated response; and *The pathogenesis of a 2022 North American highly pathogenic clade 2.3.4.4b H5N1 avian influenza virus in mallards*, Avian Pathology, 2023 — which bears on how far the duck remains a closing pair under the current clade.

**Prior work in this programme**, referenced in sections 2 and 4: *Protection Without a Component* (closure defined operationally for tuberculosis immunity, October 2026); *Closure, Carry and the Ladder*; *Where Illness Comes From*, eighth edition; and *Curves in the Vacuum.Net*, which states the positional vocabulary

used throughout. The foundational statement of the theory is the *Vacuum.Net Theory Foundational Paper* and its Ninth Edition.