

ERVs on Trial: Do They Demonstrate Evolution, or Special Creation?

A Design-Based Model of Endogenous Retroviruses and a Response to the Viral-Fossil Narrative

D. Budinsky¹

ABSTRACT

Endogenous retroviruses (ERVs) are routinely presented as among the strongest evidence for universal common descent. On the standard account, shared ERV insertions are the fossilized scars of ancient germline infections, and their distribution in nested hierarchies across taxa is taken to be nearly decisive. In this paper I challenge that interpretation and advance a positive, design-based alternative. I distinguish two categories of ERV, fixed and unfixed, and argue that a functionally integrated subset of the fixed class comprises created genomic modules rather than degenerate viral relics, while the unfixed class reflects recent, polymorphic retroviral activity in a world subject to corruption. Drawing on the mainstream functional literature, I document that ERV-derived sequences supply enhancers and promoters, regulate the interferon response and innate immunity, help shape the p53 transcriptional network, mediate placentation through the syncytins, and confer antiviral superinfection resistance through receptor interference. Under a design framework the viral-like architecture of these elements is a functional requirement for defense and mimicry rather than a signature of viral ancestry. I develop an escape model in which exogenous retroviruses arise as corrupted derivatives of host retroelements, drawing on the documented mechanistic continuity between LTR retrotransposons, endogenous retroviruses, and infectious retroviruses while recognizing that the deep origin of retroviruses remains unresolved. I then meet the strongest form of the common-descent argument, the pattern of orthologous ERV insertions, on its own terms, and argue that it establishes shared genomic history rather than unguided common descent or any particular deep-time chronology. A contrasting PTERV1 case further demonstrates why orthology must be established locus by locus: improved mapping identified 99.8% of 540 elements in chimpanzee and gorilla genomes at nonorthologous positions despite their family-level sequence relationship. The koala retrovirus is examined as a particularly clear contemporary test case of germ-line invasion; complementary feline ERV-DC data show that endogenous copies can remain infectious, segregate polymorphically, reintegrate, and contribute sequence to recombinant viruses. I close with a set of questions the evolutionary account has not answered and situate the model within a biblical history of creation, corruption, and dispersal.

Keywords: *endogenous retrovirus; solo LTR; syncytin; superinfection resistance; interferon; created diversity; escape hypothesis; koala retrovirus; orthologous loci; intelligent design; young-earth creation*

Author's note on scope. *This paper is a focused, standalone treatment intended for peer review. It is not an exhaustive presentation of the full model, which I develop at greater length elsewhere, including a book-length treatment of fixed and unfixed ERVs, functional integration, viral mimicry, and the broader biblical-historical context. Here the aim is narrower: to state the design model of ERVs precisely, to ground its functional claims in the primary literature, to engage the strongest opposing argument honestly, and to respond to the most common form of the viral-fossil argument as advanced by its more prominent defenders.*

1. Introduction

Few lines of evidence are invoked as confidently in defense of common descent as endogenous retroviruses. Advocates describe them as the ancient remnants of exogenous retroviral infections that integrated into germline DNA and were then inherited across millions of years, and they point to shared insertions that fall into nested hierarchical patterns as being about as close to proof as biology offers. The claim is not marginal. It appears in textbooks, in public debate, and in the work of those who present themselves as specialists on the subject. I do not dispute much of the technical description of retroviruses and ERVs. Where I part company is at the level of interpretation. The central question is not whether ERVs resemble retroviruses; plainly they do. The real question is whether ERVs are, in the main, the fossilized scars of ancient viral infections, or whether a large and important subset of them are created units of genomic function that resemble viruses precisely because of what they were designed to do. Much of the popular case against biblical creation on this point succeeds only against a position I do not hold, namely that every ERV is strictly a viral remnant. That is a strawman. My model draws a principled distinction between two categories of element and makes different claims about each. Once

that distinction is in view, a number of the standard challenges dissolve, and several observations that are treated as awkward for design turn out to be predicted by it.

The paper proceeds as follows. I first describe the architecture of ERVs, which is the necessary background for everything that follows. I then set out the two-category model and the functional evidence for it, address the most common objections, develop a positive account of the origin of exogenous retroviruses, and confront the strongest common-descent argument, the pattern of orthologous ERV loci, at full strength. I examine the koala retrovirus as a test case, list the questions the evolutionary account has left unanswered, and close by situating the model within a biblical history of the genome.

2. Background: The Architecture of ERVs

A full-length ERV has the same basic organization as an integrated retroviral provirus (Figure 1). Internal coding regions, gag, pol, and env, are flanked by two long terminal repeats (LTRs). Each LTR is itself structured, carrying the enhancer and promoter elements that drive transcription along with signals for polyadenylation. This architecture is not incidental to the discussion, because it is the same architecture that makes these elements useful as regulatory devices.

Two facts about ERV structure are frequently underweighted in popular presentations. First, the overwhelming majority of ERV-derived sequence in the genome is not full-length provirus at all, but solo LTRs, single LTRs left behind when the two LTRs of a provirus recombine and excise the internal genes (Figure 1). Solo LTRs lack gag, pol, and env. On the viral-fossil reading this is simply decay. But a solo LTR retains exactly the parts that matter for gene regulation: promoters, enhancers, and transcription-factor binding sites, and this cis-regulatory reservoir is now well documented in the mainstream literature (Chen et al., 2024). Second, ERV-derived sequences are demonstrably active in the genome as regulatory elements, a point taken up in detail below. The structure that is read as the wreckage of a virus is, functionally, a compact regulatory cassette.

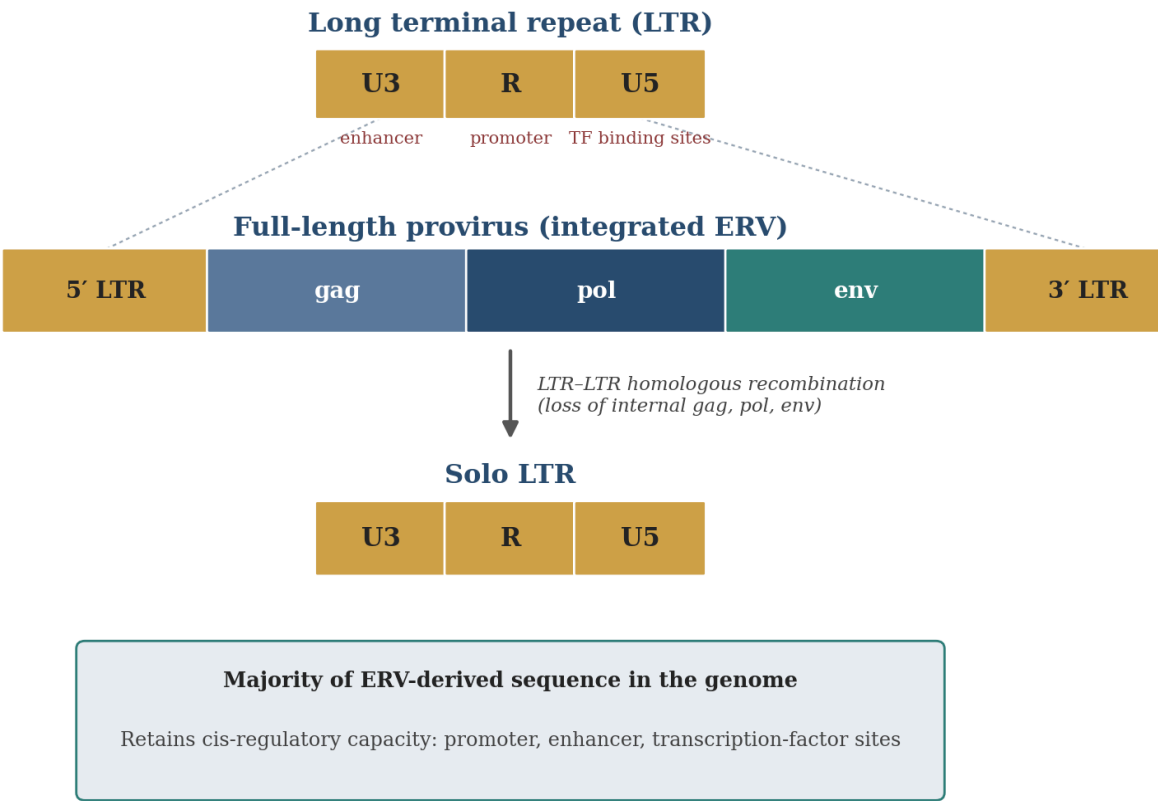


Figure 1. Architecture of a full-length ERV and the origin of solo LTRs. Internal genes (gag, pol, env) are flanked by long terminal repeats that carry enhancer, promoter, and polyadenylation signals. Homologous recombination between the two LTRs excises the

internal genes and leaves a solo LTR, which retains *cis*-regulatory capacity and constitutes the majority of ERV-derived sequence in the genome.

3. A Two-Category Model: Fixed vs. Unfixed ERVs

The single most important distinction in the model is between fixed and unfixed ERVs (Figure 2). It is a distinction the viral-fossil narrative tends to blur, and much of the confusion in the debate follows from that blurring.

Fixed ERV-derived loci are shared across an entire species or kind. A subset of them, and this is the class the model rests on, have experimentally demonstrated regulatory, developmental, immune, or structural functions, some of them important or essential to particular biological processes. Fixation itself, however, is not taken here as proof of function. Fixation is a population-genetic status, the state of being present in every member of a group, and it must be argued to function locus by locus rather than assumed. Many fixed HERV loci are demonstrably defective or transcriptionally silenced, and I do not claim otherwise; the honest picture is a genome in which a functional core sits within a larger field of degraded and quiescent sequence. The functional signal also tracks structure. The clearest cases of organism-level function are intact or near-intact elements and co-opted coding genes, syncytin, the interferon-network enhancers, the p53 binding sites, whereas the abundant solo LTRs and short fragments retain *cis*-regulatory capacity, promoters, enhancers, and transcription-factor sites, without that capacity being exercised at every copy. That a fragment shows no measured activity in a given assay is not proof it is functionless, but neither is latent capacity a demonstration of function. The model therefore predicts a gradient, not a uniform field of purpose: greater functional density among the larger, more complete fixed elements, thinning toward the small and decayed. This is a narrower and more defensible claim than asserting that the fixed class as a whole is essential, and it is the claim the functional literature actually supports.

Unfixed ERVs are the opposite in nearly every respect. They vary across individuals and populations, are often the product of genuine exogenous retroviral activity, are sometimes functional in some organisms but are not essential, and are unevenly distributed exactly as recent polymorphic insertions should be. On my account the unfixed ERVs in humans are true ERVs in the classical sense. It is the functional core of the fixed class that I argue is designed.

This is not a rescue device bolted on after the fact. It is a segregated design framework that predicts the pattern observed: a core of shared, functional, deeply integrated elements plus a periphery of variable, dispensable, individually distributed ones. When a critic asks why some people carry a particular ERV and others do not, the answer is immediate: unfixed ERVs vary between individuals just like any other polymorphism. The question is only a puzzle if one assumes in advance that every ERV must be a fossilized infection.

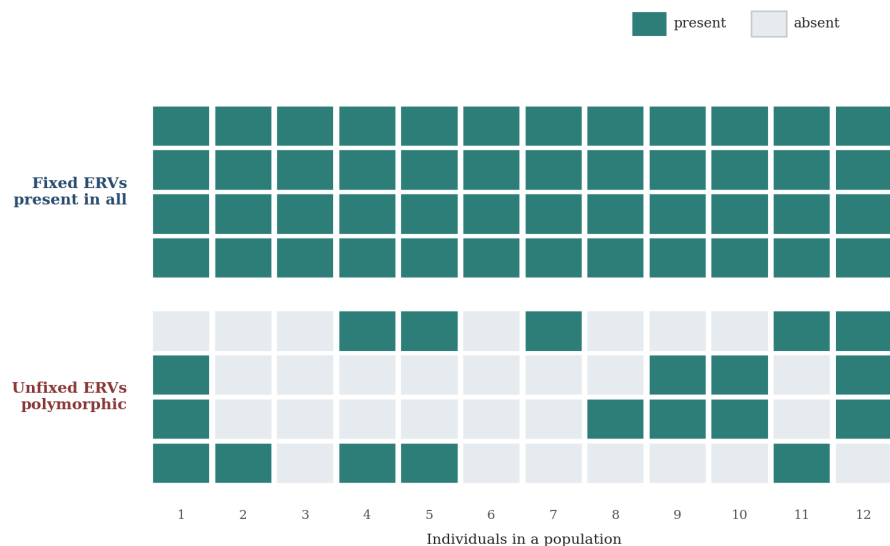


Figure 2. Schematic contrast between fixed and unfixed ERVs across individuals in a population. Fixed loci are present in every individual; unfixed loci are polymorphic, present in some individuals and absent in others. The figure is illustrative and not drawn from a specific dataset.

4. The Functional Reality of ERVs

The functional literature on ERVs has advanced dramatically, and it no longer supports the picture of ERVs as inert junk punctuated by rare exceptions. A functionally significant subset of ERV-derived sequences is now known to operate across a striking range of essential systems (Table 1).

They act as promoters and enhancers for host genes and as a natural reservoir of regulatory sequence (Feschotte, 2008; Rebollo et al., 2012). Specific ERV families seed the enhancers that coordinate the interferon response, a central branch of innate immunity, and deletion of a subset of these elements measurably impairs the induction of nearby immune genes (Chuong et al., 2016). Species-specific ERVs have helped shape the transcriptional network of the tumor suppressor p53 (Wang et al., 2007). The envelope gene of an ERV, syncytin, was captured, on the standard telling, to mediate the cell fusion that forms the placental syncytiotrophoblast, and independent syncytin genes have been identified across mammals (Mi et al., 2000; Blaise et al., 2003; Dupressoir et al., 2009). ERV elements are reactivated in a controlled way during early embryogenesis and in pluripotent cells (Grow et al., 2015). And, as discussed below, endogenous env proteins confer resistance to infection by related exogenous viruses (Nethe et al., 2005).

The point for present purposes is not that any of these authors endorses design; they do not, and they interpret their findings within an evolutionary frame of capture and co-option. The point is evidential: the elements at issue are not, as a class, broken relics. A meaningful subset is functional, and in many cases essential. Any adequate account of ERVs has to reckon with that fact, and a design model reckons with it directly, because in a design model, function is what these elements are for.

Table 1. Documented functional roles of ERV-derived sequences. The cited studies interpret these findings within an evolutionary frame; they are marshalled here for the empirical fact of function, not for their interpretation.

Functional role	Representative example	Reference
Enhancers and promoters for host genes	Diverse LTR-derived cis-regulatory elements	Feschotte 2008; Rebollo et al. 2012
Innate immunity and the interferon response	IFN-inducible enhancers from the MER41 ERV family (e.g., near AIM2, APOL)	Chuong et al. 2016
Tumor-suppressor network	p53 binding sites contributed by species-specific ERVs	Wang et al. 2007
Placentation	Syncytin envelope genes (syncytin-1, syncytin-2, syncytin-A)	Mi et al. 2000; Blaise et al. 2003; Dupressoir et al. 2009
Early embryogenesis and pluripotency	HERV-K reactivation in preimplantation embryos	Grow et al. 2015
Antiviral defense (superinfection resistance)	Env- and Gag-based restriction (Fv4, Fv1)	Best et al. 1996; Nethe et al. 2005

The observation that a functional subset of ERVs regulates development, immunity, and defense is exactly what one expects if the fixed core was built into the genome to do that work. The viral-fossil account, by contrast, has to treat this functionality as a long series of fortunate co-options of sequences that arrived by accident.

From promoter capacity to locus-level regulatory architecture

Genome-scale transcriptional surveys establish the breadth of ERV regulatory capacity, but breadth should not be confused with demonstrated biological function. Conley et al. (2008) mapped 51,197 ERV-derived transcription-start-site candidates, including 1,743 in gene-proximal promoter or 5'-UTR regions. More stringent paired-end transcript evidence connected 114 ERV-initiated start sites to 97 RefSeq gene loci. The authors also cautioned that some of the much larger candidate set could represent transcriptional noise. The proper inference is therefore tiered: the genome-wide count establishes extensive promoter potential, while organismal function must be demonstrated locus by locus.

The β 1,3-galactosyltransferase 5 (B3GALT5) locus supplies such a locus-level example. A solitary ERV-L LTR functions as an alternative, tissue-specific promoter and is the dominant promoter for B3GALT5 in the human colon; hepatocyte nuclear factor 1 binds within the LTR, and its binding correlates with promoter activation (Dunn et al., 2003). A follow-up comparison found that orthologous LTRs from four nonhuman primates were active in a human colorectal cell

line, that the baboon LTR was active in primary baboon colon tissue, and that the functional transcription-factor-binding sites were already represented in the inferred consensus sequence for that LTR class (Dunn et al., 2005). This does not establish that the locus underwent no subsequent change, but it does argue that its useful regulatory grammar did not have to be assembled *de novo* only after the insertion acquired a host role.

The NAIP locus presents an even more unusual pattern. Romanish et al. (2007) showed that unrelated LTRs serve as promoters for orthologous neuronal apoptosis inhibitory protein genes in humans, mice, and rats. In humans, an HERV-P LTR supplies a primarily testis-specific promoter; in rodents, an ancestral ORR1E LTR is the major constitutive promoter, while an MTC LTR provides secondary promoter activity for two mouse Naip copies. LTR sequence occupied 9.75% of the 12.5-kb upstream regions of the eight human inhibitor-of-apoptosis genes, placing that set in the 97th percentile of 1,000 random gene sets; the corresponding mouse value was 13.8%, exceeded by only three of 1,000 random sets. The authors considered several nonexclusive explanations, including integration preference, regional tolerance, selection, and a functional relationship between LTR activity and control of apoptosis.

These results do not by themselves decide between evolutionary co-option and design. They do, however, sharpen the explanatory target. The evidence rises from widespread promoter potential, to experimentally grounded tissue-specific function, to repeated use of unrelated LTRs at the same orthologous regulatory problem. My model should rest on that evidential hierarchy, not on the claim that every LTR is functional, but on the finding that a defined subset displays native regulatory competence and recurrent, biologically coherent deployment.

5. The “Only Parts Are Functional” Objection

A common fallback is to concede that fragments of ERVs are functional while denying that any full element is. This objection does not do the work it is asked to do.

Consider the analogy. One would not say that a human being is not a functional organism on the grounds that only the heart, brain, and lungs are functional. The organism is functional through its components. In the same way, functional ERVs operate as integrated systems. Envelope proteins block viral entry; ERV expression can trigger antiviral immunity; viral mimicry, by its nature, requires viral-like structure. To perform these roles an element has to resemble a virus at the molecular level. That resemblance is a design requirement, not a confession of viral ancestry, and it is one straightforward reason these elements share sequence similarity with exogenous retroviruses.

The claim that only isolated fragments are functional is also increasingly out of step with the literature summarized above, in which intact regulatory and protein-coding contributions from ERVs are documented across immunity, development, and placentation.

6. Which Came First: The Virus or the Host?

A question the strongest form of the common-descent argument tends to leave underexamined is this: what came first, the retrovirus or the host genome it depends on? (Figure 3.) It is worth stating plainly at the outset that mainstream virology does not, in fact, hold a simple “virus first” position. It is worth stating plainly that mainstream virology does not require a simple “virus-first” account. One influential origin model proposes that retroviruses arose from LTR retrotransposons through acquisition of an envelope gene, although the deep origin of retroviruses remains unresolved and reverse transitions—from endogenous retroviruses to noninfectious LTR retrotransposons—also occur (Boeke & Stoye, 1997; Malik et al., 2000; Hayward, 2017). The empirical common ground is therefore narrower but still significant: retroviruses, endogenous retroviruses, and LTR retrotransposons occupy a mechanistically continuous and evolutionarily permeable system.

Retroviruses cannot replicate independently. They rely entirely on host machinery, borrowing the cell's enzymes, transcription systems, and codon usage. A virus that depends this completely on a host is a poor candidate for the original, autonomous entity. This asymmetry points toward the account that mainstream virology and the design model share: exogenous retroviruses are derived forms, degenerate descendants of originally functional host retroelements.

My disagreement with the standard account is therefore narrower and sharper than a reversed arrow. It is not about the direction of derivation, but about two things: whether the ancestral host retroelements were functional, purposefully

integrated components of the genome or undirected selfish sequence, and whether the escape to infectious form required deep time or occurred rapidly under corruption. This escape model sits within a well-recognized continuum of retroelements, from non-mobile sequences through LTR retrotransposons to full retroviruses, in which the acquisition or loss of a small number of features, envelope in particular, moves an element along the spectrum (Boeke & Stoye, 1997; Malik et al., 2000). Framed this way, the design and escape model is not a fringe reversal of virology's arrow; it accepts that arrow and contests what sat at its origin and how long the journey took.

An appeal to parsimony, which defenders of common descent sometimes make at this point, actually cuts toward the shared direction. The account that requires the fewest unexplained additions is the one in which the functional, host-based element comes first and the stripped-down, dependent virus comes second. What the design model adds is not a new direction but a claim about the starting material: a genome front-loaded with functional retroelements rather than colonized by accidents.

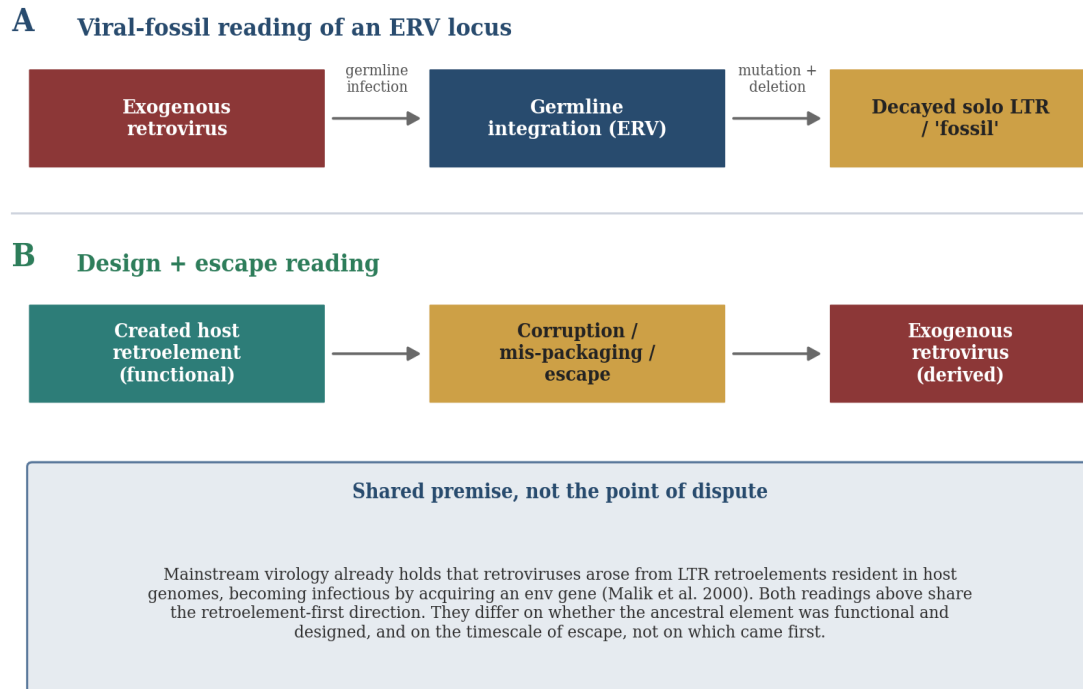


Figure 3. Two readings of an ERV locus. (A) The viral-fossil reading treats a given ERV as the decayed scar of an exogenous infection. (B) The design and escape reading treats the fixed, functional element as a created host module and the exogenous retrovirus as its corrupted, escaped derivative. Both readings operate within the documented mechanistic continuum between LTR retrotransposons, endogenous retroviruses, and infectious retroviruses; they differ on whether ancestral host elements were designed and functional and on the proposed history and timescale of transition.

7. Why Functional Elements Must Resemble Viruses

Critics often ask: if ERVs are designed, why do they look like viruses? The objection misunderstands how defensive and regulatory systems work. To block, regulate, or interfere with a virus, a system has to recognize, model, or mimic the relevant viral features. Resemblance is not surprising; it is required.

The mechanism is well documented. Some endogenous elements confer superinfection resistance: expression of an endogenous env protein blocks the receptor that a related exogenous virus needs for entry, so the cell cannot be infected (Nethe et al., 2005). In mice, the Fv4 locus encodes an Env-related protein whose receptor-binding domain resembles that of murine leukemia virus and blocks infection by it; the Fv1 locus, derived from a gag-like sequence of an endogenous element, restricts the same virus at a later step (Best et al., 1996; Nethe et al., 2005). Comparable env-based restriction is seen in other mammals. In each case a host element defends against a virus precisely because it resembles that virus at the molecular level relevant to the interaction.

Two analogies make the logic vivid. Antivirus software must model the signatures and behavior of malware in order to detect and neutralize it; it does not thereby become malware, but it must resemble it where the interaction happens. An undercover agent infiltrating a criminal organization does not arrive in uniform; he has to look the part and speak the language to do the job, and his resemblance to the criminals is evidence of his assignment, not of his origin among them. In the same way, an ERV that must interfere with viral replication, block viral receptors, and trigger innate defenses has to look viral. Its viral-like structure is expected under a design model, not evidence of descent from ancient infections.

Documented movement between endogenous and infectious forms

The feline ERV-DC/RD-114 system provides a concrete mechanistic bridge between inherited retroelements and circulating retroviruses. Phylogenetic comparison supported a recombinant origin for RD-114: its gag-pol region is related to the feline endogenous element FcEV, whereas its env derives from baboon endogenous virus (BaEV). The same analysis obtained cross-species infection by a BaEV-like virus followed by entry of the recombinant into the *Felis* germ line (van der Kuyl et al., 1999). This is direct evidence of historical transfer from a reconstructed sequence and distribution.

Anai et al. (2012) subsequently supplied functional and population-level evidence for mobility in the same feline system. ERV-DC10 was an infectious endogenous gammaretrovirus with broad cell tropism in vitro and was present at a homozygous or heterozygous locus in 92 of 244 domestic cats. A nearly identical copy at a second locus, ERV-DC18, was consistent with recent reintegration or translocation, while feline leukemia virus subgroup D was shown to have arisen through acquisition of an ERV-DC env sequence. Thus, endogenous copies can remain infectious, occupy insertionally polymorphic loci, move to new loci, and donate sequence to recombinant exogenous viruses.

The significance for the escape model is real and backed by evidence. These studies demonstrate that the endogenous/exogenous boundary is mechanistically permeable and that retroviral genomes are modular: recombination, envelope exchange, reinfection, and germ-line entry can redistribute related sequences without following the host species tree. Consequently, a shared ERV family in divergent hosts is not equivalent to a shared orthologous insertion. Vertical inheritance, horizontal infection, and recombination must be distinguished with locus boundaries, flanking host DNA, target-site duplications, and population data.

8. The Koala Retrovirus as a Test Case

The koala retrovirus (KoRV) is often cited as decisive confirmation of the viral-fossil account, and it deserves direct treatment because it is the single most instructive live case. KoRV remains one of the clearest contemporary examples of a retrovirus occupying both exogenous and endogenous states. KoRV provirus is essentially universal and high-copy in the northern populations examined, whereas southern populations exhibit lower and more heterogeneous prevalence and copy number, including virus-negative individuals; this geographic gradient is a major reason KoRV is interpreted as an ongoing germ-line invasion (Tarlinton et al., 2006; Simmons et al., 2012).

I accept that KoRV documents a virus entering a germline, that is, endogenization in the classical direction. What is often missed is how favorable this case is to my framework on two counts.

First, timescale. KoRV shows that a retrovirus can move from exogenous infection to inherited, and toward fixation, within a population over a span measured in generations rather than in millions of years. A process that fast is entirely compatible with a young timescale in which a substantial number of unfixed ERVs were acquired after a population bottleneck and are still segregating. The real-time koala data undercut the assumption that the mere presence of an inherited retroviral element requires deep time.

Second, defense. Once endogenized, retroviral elements can protect their host against related exogenous viruses through the receptor-interference mechanism described above. An endogenous element that blocks a harmful virus behaves, functionally, like resident antivirus software, and it can do so only because it resembles the threat. This is forward-looking function, and it is exactly the kind of role the model assigns to the fixed, defensive class. The koala

case, read carefully, illustrates both that endogenization is fast and that endogenous elements can be protective, which are two of the load-bearing claims of the model, not objections to it.

9. The Strongest Common-Descent Argument: Orthologous ERV Loci

A model earns the right to its conclusion only by meeting the strongest form of the opposing case, not the weakest. For ERVs that strongest case is the pattern of orthologous loci, and I take it here at full strength. The result of that engagement is not surrender but clarity: orthology establishes a shared genomic history, and it does not, by itself, establish either unguided common descent or the deep-time chronology usually attached to it.

The relevant observation is not merely that humans and nonhuman primates contain similar retroviral-like sequences. Closely related species can carry an ERV-derived element at the corresponding chromosomal position, in the same orientation, between homologous host-DNA flanks. In the strongest cases, the element has matching insertion boundaries; the occupied allele is bordered by a short target-site duplication (TSD) generated during integration; an outgroup or insertionally polymorphic individual retains the corresponding empty site; and the internal ERV sequence contains derived substitutions, deletions, or recombination products whose distribution can be compared across species. This multilayered evidence is substantially stronger than sequence similarity alone, and it should be conceded plainly.

Why orthologous loci are considered powerful evidence

The standard common-descent interpretation is straightforward. A retrovirus infected a germ-line cell in an ancestral population, its provirus became fixed or remained polymorphic, and descendants inherited the occupied locus as ordinary chromosomal DNA. Johnson and Coffin (1999) emphasized three complementary phylogenetic signals: the presence or absence of the insertion among taxa, divergence among orthologous proviral sequences, and divergence between the 5' and 3' long terminal repeats of a provirus. Because the two LTRs are formed as identical copies during reverse transcription, their later differences record changes accumulated after integration. A congruent result across these signals is stronger than any one observation by itself. The integration-associated TSD adds a further mechanistic feature: retroviral integrase makes staggered cuts in host DNA, and repair duplicates of a short segment, commonly about 4 to 6 base pairs, on the two sides of the provirus. The TSD is not a long, unique barcode, but the complete arrangement is informative: one copy of the target sequence at an unoccupied allele, two copies flanking the occupied allele, matching boundaries, matching orientation, and homologous host sequence on both sides. Here the honest position must be stated without flinching. Under an ordinary, unguided infection model, a single ancestral insertion followed by inheritance is generally more economical than repeated independent insertions at exactly corresponding sites. Integration bias alone cannot presently explain hundreds of matching loci with matching boundaries and homologous flanks as well as vertical inheritance can. This is the real force of the argument, and I do not evade it.

What the 211-locus HERV-W study actually found

The evidence is, however, more qualified than common summaries imply, and the most cited dataset shows why. The HERV-W comparative study by Grandi et al. (2018) is frequently summarized too loosely. The preceding human-genome catalog contained 213 HERV-W-related loci, but these were not 213 equivalent, full-length proviruses. Grandi et al. (2016) classified the catalog as 65 proviruses with two LTRs, 135 LINE-1-mediated processed pseudogenes with characteristically truncated LTR structures, and 13 elements that could not be classified because both LTRs were absent. Most members of this dataset were therefore not created by 135 additional exogenous retroviruses independently infecting 135 germ cells; they were copies of HERV-W transcripts mobilized intracellularly by LINE-1 machinery. That distinction does not eliminate their value as inheritance markers, but it changes what biological event each locus represents. For the comparative analysis, two loci on the human Y chromosome were excluded, leaving 211 human loci. Of these, 123 were unambiguously present in every analyzed Catarrhine genome from human through rhesus. The authors raised that number to 131 when they included loci present in human and rhesus but apparently missing in one or more intervening taxa, interpreting the gaps as possible deletion, rearrangement, assembly, or alignment effects. Four qualifications are essential. First, “211 loci analyzed” does not mean all 211 were shared by all six Catarrhine species; the universally shared subset was 123, or 131 under the authors’ gap-tolerant interpretation. Second, “HERV-W locus” does not mean “intact provirus”; the catalog combined true proviruses, LINE-1-generated processed pseudogenes, and unclassifiable fragments. Third, the reported counts are not a perfectly monotonic series: gorilla had 207

human-orthologous loci, whereas chimpanzee and orangutan each had 205, so the pattern is not a flawless, exceptionless ladder produced by presence and absence alone. Fourth, the study was a comparative-genomics survey, not 211 independent experimental demonstrations of every insertion criterion in every species. The same study documented biological processes that complicate a simple presence and absence tree: nineteen nonhuman ERV-W proviruses corresponded to a solitary LTR at the human orthologous position, consistent with LTR-LTR recombination; and of 88 nonhuman loci with corresponding empty sites in humans, 81 were interpreted as species-specific insertions. The authors explicitly cautioned that some apparent differences could reflect deletion, rearrangement, assembly error, or comparative-alignment error (Grandi et al., 2018). The real dataset therefore contains shared loci, lineage-specific loci, solo-LTR conversion, apparent losses, uncertain elements, and assembly-dependent gaps. That is a richer and more complicated result than the slogan “211 shared viral scars.”

PTERV1 as an empirical control: 299 mapped sites and the collapse of apparent orthology

An unusually revealing counterpoint to the HERV-W dataset is supplied by the PTERV1 study of Yohn et al. (2005). PTERV1, or *Pan troglodytes* endogenous retrovirus 1, is important because it allows the criteria for orthology to be tested against a retroviral family distributed across several primates but overwhelmingly absent from corresponding genomic positions. The study demonstrates that three observations commonly blended together in popular ERV arguments must be kept separate: the presence of related retroviral sequences in two species, placement within the same broad chromosomal neighborhood, and insertion at the exact orthologous position. PTERV1 satisfies the first criterion across several primates and occasionally appeared to satisfy the second, but almost never satisfied the third.

Yohn et al. (2005) identified PTERV1 through a comparison of chimpanzee and human genomic sequences. The investigators found a chimpanzee segment containing recognizable *gag*, *pol*, *env*, and long terminal repeat sequences that was absent from the corresponding human sequence. Full-length PTERV1 elements ranged from approximately 5 to 8.8 kb. Some contained large deletions within the reverse-transcriptase region, and all initially examined full-length copies contained multiple stop codons, indicating that the sampled elements were no longer autonomously replication competent.

Southern hybridization nevertheless detected more than 100 PTERV1-related copies in African apes and Old World monkeys. Strong signals were observed in chimpanzees, bonobos, gorillas, colobus monkeys, baboons, and macaques. By contrast, no PTERV1 signal was detected in a panel of human DNA or in the Asian apes examined, including orangutan, siamang, and white-handed gibbon. The survey included multiple individuals from each ape lineage, 16 humans, and nine baboons. The resulting distribution was striking: a large related retroviral family occurred in African apes and Old World monkeys but was undetectable in humans and Asian apes, interrupting the nested pattern expected if all copies represented one set of vertically inherited ancestral insertions.

For positional analysis, the investigators screened large-insert BAC libraries from common chimpanzee, gorilla, olive baboon, and rhesus macaque. They end-sequenced 1,467 BAC clones containing substantially full-length PTERV1 elements and mapped their positions against the human genome reference assembly.

PTERV1 locus calls and unambiguously nonorthologous locations

Species	Mapped locus calls	Unambiguously nonorthologous
Common chimpanzee	91	86
Gorilla	81	78
Olive baboon	81	73
Rhesus macaque	46	38
Total	299	275

Table 2. PTERV1 locus calls and unambiguously nonorthologous locations reported by Yohn et al. (2005). The 24 unresolved species-level calls could be paired into a maximum of 12 potentially corresponding intervals, producing 287 distinct loci for the authors' calculation of 95.8% nonorthology.

The arithmetic requires careful explanation. Yohn et al. reported 299 species-level locus calls. Of these, 275 mapped unambiguously to different genomic locations. The remaining 24 calls occurred in pairs within similar regions of the human reference genome. If every pair represented one genuinely shared insertion, those 24 calls would collapse into a maximum of 12 potentially shared loci. The denominator for the authors' calculation was therefore 275 nonorthologous loci plus 12 potentially shared loci, or 287:

$$275 \div 287 = 95.8\% \text{ nonorthologous.}$$

The authors explicitly described 95.8% as a lower-bound estimate. The 24 ambiguous calls had not been demonstrated to occupy matching nucleotide positions. They merely mapped somewhere within the same BAC-scale interval, generally encompassing approximately 160 kb. Two insertions separated by tens or hundreds of thousands of base pairs could therefore appear to overlap under the limited resolution of the method. The 24 calls must not be described as 24 confirmed orthologous sites. At most, they represented 12 candidate pairs requiring higher-resolution testing.

The phylogenetically discordant distribution of the candidate intervals

The pairwise distribution of the 12 ambiguous intervals was itself irregular. Five joined baboon with macaque, three joined chimpanzee with macaque, two joined gorilla with baboon, one joined chimpanzee with baboon, one joined chimpanzee with gorilla, and none joined gorilla with macaque. Thus, the closest ape pair represented in the mapping analysis, chimpanzee and gorilla, possessed only one potentially corresponding interval, whereas chimpanzee and the more distantly related macaque appeared to share three. Gorilla and baboon appeared to share twice as many candidate intervals as gorilla and chimpanzee. Yohn et al. (2005) concluded that this distribution was inconsistent with the accepted Catarrhine species tree if the intervals were interpreted as vertically inherited orthologous insertions.

The paper then exposed the deletion burden created by treating all candidate intervals as ancestral. Because humans and orangutans lacked detectable PTERV1 sequence, accepting the ambiguous ape and monkey intervals as genuinely orthologous would require at least six ancestral PTERV1 loci to have been deleted independently from the human lineage. The same six positions would also have to have been deleted independently from the orangutan lineage. Yohn et al. regarded such parallel losses at the same genomic positions as unlikely.

This argument is conditional. The study did not demonstrate that six deletions occurred in humans and another six in orangutans. The authors used that improbable requirement as evidence that the broad overlaps probably were not ancestral orthologs. Their preferred explanation was independent, lineage-specific integration following exposure to related viruses. The result does not establish that the accepted primate tree is false. It demonstrates that the distribution of a retroviral family cannot automatically be treated as a vertical species tree.

Base-pair resolution overturned apparent correspondence

The investigators attempted to resolve the three intervals that appeared to be shared between chimpanzee and macaque. One could not be refined because it was dominated by repetitive sequences. The other two were resolved to single-base-pair precision, and neither was orthologous.

At the exact chimpanzee insertion sites, the corresponding macaque sequence remained contiguous and lacked the chimpanzee insertion. The macaque PTERV1 elements responsible for the original apparent overlaps were situated elsewhere within the same approximately 160 kb neighborhoods. What appeared at low resolution to be the same locus was actually two independent insertions in the same broad region. These two resolved pairs converted four of the 24 ambiguous species-level calls into confirmed independent events. Precise mapping also revealed 4 bp and 5 bp AT-rich target-site duplications flanking the chimpanzee elements. These duplications had the length and composition expected from retroviral integration. The result simultaneously supported authentic retroviral insertion and disproved the proposed cross-species orthology. Authenticity of integration and orthology of position are therefore separate questions.

Yohn et al. proposed two possible reasons why independent integrations might cluster within similar genomic neighborhoods. PTERV1 could possess integration preferences that make certain regions more likely targets, or purifying selection could remove harmful integrations from functionally intolerant regions, leaving surviving insertions concentrated within the portions of the genome where they are better tolerated. Either process would produce more regional convergence than a model in which every nucleotide is treated as equally probable and equally survivable.

The viral sequence tree did not follow the host tree

The investigators also sequenced approximately 823 bp from the *gag* and *env* regions of 101 PTERV1 elements: 42 from chimpanzee, 25 from gorilla, 20 from olive baboon, and 14 from rhesus macaque. The resulting viral tree did not reproduce the accepted primate species tree.

Chimpanzee and gorilla PTERV1 elements formed a relatively close group with approximately 3% to 4% divergence. Baboon and macaque elements showed approximately 9% to 11% divergence and formed at least three viral groups with comparatively little separation according to host species. One Old World monkey group appeared related to the chimpanzee and gorilla group, but the overall pattern was more compatible with multiple viral lineages and cross-species transmission than with uninterrupted vertical inheritance of one fixed ancestral insertion set.

Separate analyses of *gag*, *env*, and LTR sequence produced broadly similar topologies. In the LTR comparison, macaque and baboon interproviral divergence ranged from approximately 14% to 24%, whereas chimpanzee and gorilla divergence was approximately 3% to 4%. These values greatly exceeded the neutral host-sequence divergences used by the authors for comparison.

One candidate chimpanzee–gorilla overlap pair was also represented in the phylogenetic tree. If the two elements were orthologous descendants of one ancestral insertion, they should have grouped together as close relatives. They did not cluster, providing sequence-level evidence that the apparent overlap was unlikely to represent a true ortholog.

The study's inference was not that PTERV1 sequences lacked a relationship. The family plainly possessed a shared viral origin. The inference was that related PTERV1 sequences entered or expanded within the different host genomes independently. Viral relatedness does not require positional orthology, and neither viral relatedness nor broad regional overlap proves that a particular insertion was inherited from the common ancestor of the host species.

LTR divergence, activity, and surviving polymorphism

Yohn et al. compared the left and right LTRs of 101 substantially full-length elements. Because the two LTRs are produced as identical copies during integration, their subsequent divergence was used as a model-dependent measure of elapsed time. The average numbers of mismatches across the examined region were 3.4 ± 2.2 for chimpanzee, 2.9 ± 2.3 for gorilla, 1.6 ± 1.4 for baboon, and 1.6 ± 1.5 for macaque.

Using their selected substitution calibration, the authors interpreted these values as evidence of infections in the chimpanzee and gorilla lineages approximately 3 to 4 million years ago and younger activity in baboons and macaques approximately 1.5 million years ago. These ages are not direct measurements. They depend on the calibration and assumptions underlying LTR dating. The observed data consist of the LTR mismatches and their distributions.

The mismatch distributions were consistent with a comparatively concentrated burst of activity within each lineage, although the authors acknowledged that multiple integrations distributed across a shorter period could generate a similar pattern. Approximately 5% to 10% of PTERV1 sites also appeared polymorphic within chimpanzee and baboon populations, possibly reflecting deletion or more recent germ-line reinfection. The family therefore contained both widely inherited and still-variable insertions rather than one uniform category of ancient, universally fixed loci.

Strong bias away from genes

PTERV1 also demonstrates that observable ERV distributions are filtered by biological effects after integration. Among 107 full-length chimpanzee insertions, only 14, approximately 13%, occurred within annotated introns. Seventy-five were located more than 50 kb from a transcription start site. Under the authors' simulation, approximately 29.2% of randomly positioned insertions would have been expected inside introns. None of 10,000 simulated datasets produced a genic distribution as depleted as the observed 14 of 107, yielding $p < .001$.

Yohn et al. interpreted this depletion as evidence of strong purifying selection against germ-line insertions in or near genes. The final distribution of surviving ERVs is therefore shaped by at least three filters: where the virus can integrate, which integrations permit survival and reproduction, and which insertions subsequently reach substantial population frequency. Most sampled PTERV1 elements were also extensively methylated, indicating that the host genome had placed much of the family under transcriptional repression.

Of the 14 genes containing intronic PTERV1 insertions, ten were represented in the authors' human–chimpanzee expression dataset. Six showed significant expression differences, and five of those six were expressed at lower levels in chimpanzee tissues. The enrichment was only weakly significant, $p = .0489$, and the authors cautioned that the sample was too small for a definitive conclusion. The result is suggestive evidence that some insertions influenced nearby transcription, not proof that PTERV1 caused every observed expression difference.

Confirmation and refinement using improved ape assemblies

The principal positional finding survived much more complete genome sequencing. Kronenberg et al. (2018) constructed a high-quality map of 540 PtERV1 elements, including full-length and solo-LTR copies, in chimpanzee and gorilla. They reported that 99.8% of the integration events were nonorthologous. They also confirmed that the distribution was biased against genes and toward antisense orientation, patterns consistent with purifying selection.

The improved assemblies did identify one genuine chimpanzee–gorilla ortholog that had been missed or left unresolved by earlier draft genomes. This 379 bp PtERV1-related element was nested inside an older LTR within a repeat-rich region corresponding to human chromosome 19. Kronenberg et al. called it the “source PtERV1” and proposed that it was present in the ancestral African ape population before contributing to independent expansions at hundreds of nonorthologous positions in chimpanzees and gorillas. They were forced to explain its absence from modern humans through incomplete lineage sorting rather than repeated precise deletions.

Among 540 elements mapped with improved assemblies, only one was identified as a chimpanzee–gorilla ortholog, while approximately 99.8% occupied noncorresponding locations. One inherited source locus and hundreds of nonorthologous descendants represent different stages of the proposed history.

What the PTERV1 result establishes

Yohn et al. interpreted the discordant distribution as evidence of horizontal viral transmission followed by probable separate germ-line invasions and lineage-specific amplification. Kronenberg et al. later proposed that those were not the best explanations but rather that one ancestral source element contributed to those independent expansions. No agreed upon scenario has been reached and other concepts are being added.

What the dataset directly refutes is the claim that related ERV sequences in related organisms must represent the same ancestral insertions. Related PTERV1 sequences occur by the hundreds across several primate genomes, yet approximately 96% of loci in the original analysis and 99.8% in the later high-resolution study were **nonorthologous**. Even placement within the same approximately 160 kb interval failed to establish positional identity.

PTERV1 consequently functions as an empirical control for the orthology argument. It demonstrates that family-level similarity is insufficient, approximate synteny is insufficient, and broad interval overlap is insufficient. A strong orthology claim requires exact insertion boundaries, matching orientation, homologous flanking sequence, appropriate target-site architecture, an empty preintegration site where available, and a congruent history of derived sequence changes. These criteria cannot be replaced by an aggregate count of ERV-related sequences.

Within the design and escape framework developed here, PTERV1 represents bona fide lineage-specific retroviral activity, regardless of whether some copies subsequently became fixed within their host populations. Present-day fixation describes population frequency, not original function or mode of origin. PTERV1 displays multiple signatures of acquired retroviral activity: lineage-specific positions, target-site duplications, a viral phylogeny discordant with the host tree, surviving population polymorphism, and extensive epigenetic repression.

The decisive lesson is methodological. ERV orthology must be demonstrated locus by locus, not inferred from family identity, species distribution, or chromosomal proximity. PTERV1 shows that independent integrations can accumulate in related organisms and cluster within the same broad genomic neighborhoods while remaining positionally distinct. This leads directly to the next question: why independent retroviral insertions are not distributed uniformly throughout the genome.

Nonrandom integration: a necessary correction

Popular presentations sometimes calculate the probability of shared ERVs by treating every base pair in a three-billion-base genome as an equally likely and independent target. That null model is biologically unrealistic. Withers-Ward et al. (1994) found that some avian retroviral sites were used up to 280 times more frequently than expected under a random model, and Mitchell et al. (2004), in a genome-wide study of 3,127 sites, found distinct preferences among HIV, murine leukemia virus, and avian sarcoma-leukosis virus. These findings defeat the simplistic claim that every nucleotide is equally probable. They do not, however, show that HERV-W or its LINE-1-mobilized derivatives repeatedly target hundreds of exact orthologous nucleotide positions in separately originating lineages. Regional preference for active genes or promoters is not equivalent to deterministic selection of the same base, orientation, boundaries, subtype, and flanking architecture. Published “one chance in the genome raised to the number of loci” calculations are unjustified without an empirical integration model, yet known integration biases have not, on their own, replaced ancestral inheritance as the best-developed natural explanation for the full orthologous-locus pattern.

What orthology establishes, and what it does not

The inference becomes less automatic when the competing hypothesis is purposeful common design rather than independent random infection. A probability calculation based on two viruses choosing sites independently does not test

a design hypothesis, because deliberate placement is, by definition, correlated rather than independent. If an ERV-derived sequence performs a conserved regulatory, developmental, immune, or structural role at a particular genomic address, a design model can predict that related organisms will possess the same module at the same address, just as the improbability of two independently assembled machines containing the same circuit at the same position does not argue against a common engineering plan.

This response is logically valid but scientifically incomplete unless the design model specifies why particular elements were shared and makes testable predictions. Common design cannot be an unrestricted explanation for every matching nucleotide, including every disabled reading frame, neutral substitution, and insertional footprint. A successful design explanation should identify independently demonstrable functions or architectural constraints and distinguish designed sequence from later mutational damage. That distinction is exactly why the functional discoveries documented earlier matter: HERV-W locus ERVWE1 at 7q21.2 encodes Syncytin-1 (Mi et al., 2000), and other ERV-derived elements have been experimentally implicated in pluripotency and innate-immune regulation (Chuong et al., 2016; Lu et al., 2014). Viral resemblance is not equivalent to biological uselessness.

The evidential hierarchy for an orthologous ERV should therefore be stated explicitly, from weakest to strongest: (1) family-level similarity, related ERV-like sequence in two genomes, the weakest level; (2) syntenic correspondence, the element within homologous host-DNA neighborhoods; (3) boundary-level orthology, matching orientation and insertion boundaries within long homologous flanks, which strongly favors a single historical origin; (4) mechanistic insertion evidence, matching TSD architecture with a verified empty site in an outgroup or unoccupied allele; and (5) shared derived history, congruent derived substitutions, indels, disabling mutations, solo-LTR states, or 5'/3' LTR phylogenies, the strongest level because it tests not merely common placement but a common sequence history after placement. The HERV-W survey powerfully documents levels 2 and 3 across many loci and supplies examples relevant to levels 4 and 5, but it should not be cited as though every one of the 211 loci was independently verified at all five levels.

Orthology is not an independent proof of deep time

Even if an orthologous ERV is accepted as inherited from a shared ancestral population, its presence does not by itself establish a particular numerical age. Presence and absence brackets the event relative to whatever species history is assumed. Numerical estimates from 5'/3' LTR divergence additionally require a mutation-rate calibration, a distance model, intact and correctly aligned LTRs, and an assessment of gene conversion, selection, CpG effects, and regional rate variation. Van der Kuyl (2025) reviewed these problems and found that choices of mutation rate and analytical method can produce incompatible insertion-time estimates. Orthology, common inheritance, phylogenetic topology, and multimillion-year dating are related but distinct claims. Rejecting a particular molecular-clock estimate does not erase positional orthology; accepting positional orthology does not require accepting every proposed integration date.

This is the decisive point for the debate, and it is where the confident creation reading is strengthened rather than weakened by the concession. Creationists already accept common ancestry within a created kind. For an ERV shared across all living humans, the creation model and the evolutionary model agree that it was inherited from a common human ancestral population, and a single ancestral insertion reaching fixation is most easily explained in a small founding population, which the creation model supplies natively at the origin of each kind. The parsimony-of-a-single-insertion argument therefore poses no difficulty for within-kind sharing at all. It becomes an argument for universal common descent only when it is stretched across the boundaries between kinds, and there the design reading has an answer the descent reading does not require: the same functional module at the same address is precisely what common design predicts, while the truly discriminating test, a long series of shared, apparently neutral disabling lesions with no functional role, must be established locus by locus rather than assumed from an aggregate count. Until that locus-by-locus test is performed on modern telomere-to-telomere assemblies, the orthologous-locus pattern is fully consistent with a designed genome inherited within kinds and does not compel the deep-time, cross-kind descent it is usually said to prove.

10. Challenges the Evolutionary Account Has Not Resolved

The viral-fossil narrative is strongest when it is asked only to interpret similarity. It is weakest when it is asked to supply mechanism and history. I close the positive argument with a set of questions that remain unanswered, and I present the

contrasting expectations of the two models side by side (Table 2). First, the stepwise origin of LTR regulatory content. An LTR contains enhancers, promoters, clustered transcription-factor binding sites, chromatin signals, integration motifs, and polyadenylation control. What is the mutation-by-mutation pathway by which such an element is built? Asserting co-option does not answer the question; it presupposes that the regulatory content already exists. Second, the origin of pre-loaded regulatory function: if ERVs already carry regulatory elements when they insert, their insertion cannot itself demonstrate the evolution of new regulatory function. Third, the borrowing problem: defenders routinely say that vertebrates borrowed regulatory functionality from viruses, yet do not explain how the viruses evolved it first; the explanatory debt is moved, not paid. Fourth, coordination and timing: how does random viral insertion produce coordinated developmental timing, such as ERV activation precisely at implantation? Fifth, the fixed-versus-unfixed pattern: why are essential ERVs fixed across a species while non-essential ERVs are polymorphic? This follows naturally from design plus corruption but is an additional thing to be explained under random infection plus co-option. Sixth, the missing contemporary co-options: if co-option accounts for the essential roles of fixed ERVs, why are unfixed ERVs not observed being drafted into essential roles today? Seventh, direct evidence of stepwise evolution: can even one study be cited that directly demonstrates the stepwise evolution of ERV regulatory complexity over time, as opposed to inferring it from the end state?

Observation	Viral-fossil (common descent)	Design + escape model
Pervasive essential function of a subset of ERVs	Explained by repeated fortunate co-option of accidental insertions	Expected: the fixed core was built to function
Fixed vs. unfixed distribution	An additional fact requiring a separate explanation	Predicted: created core (fixed) plus post-Fall activity (unfixed)
Similarity of ERVs to exogenous viruses	Evidence of viral ancestry	A functional requirement for defense and mimicry
Origin of the ancestral retroelement	Unguided; retrovirus derived from a retroelement over deep time	Created and functional; escape to exogenous form under corruption, rapidly
Real-time endogenization (koala)	Consistent with a slow fossilization process	Shows endogenization is rapid; compatible with a young timescale
Matching boundaries, orientation, flanks, TSD/empty-site architecture	Strongly expected after one ancestral insertion	Within a kind, expected from inheritance; across kinds, reuse of a functional module or demonstrated recurrent targeting
Shared apparently-neutral disabling mutations and derived indels	Expected if acquired before lineage separation	The genuine test; must be established locus by locus, not assumed
A numerical age from LTR divergence	Requires a calibrated mutation model and assumptions about conversion and selection	Not determined by orthology; the same calibration questions remain

Table 3. *Contrasting expectations of the viral-fossil account and the design and escape model for the same observations.*

11. A Biblical-Historical Framework for ERV Origins

The technical argument above stands on its own, but it is part of a larger and, I would argue, more coherent history of the genome (Figure 4). I sketch it here not as an add-on but because a model is stronger when it can place its data within a single narrative rather than a series of accidents.

At creation the original kinds were made with genuine genetic diversity and with functional retroelements, including ERV-like sequences, already in place. These elements were neither junk nor pathogens; they carried regulatory and developmental roles from the start. Because the original creation was very good, harmful viruses did not yet exist, but the genome was equipped with latent systems that would matter later, much as safety features in a vehicle go unnoticed until conditions turn dangerous. With the entrance of corruption at the Fall, decay and mutation began to accumulate, and functions that had been redundant or latent became critical. ERV-like sequences already embedded in developmental and

regulatory networks now also served in defense, immunity, and viral mimicry; their similarity to retroviruses was not accidental, because to block viruses effectively they had to resemble them.

The global Flood was a genomic turning point. Severe bottlenecks, environmental stress, and accelerated decay created the conditions under which some retroelements escaped their original boundaries, recombining or mis-packaging into exogenous retroviruses. This is where the escape model fits most naturally, and it is worth noting that the direction it requires, host retroelement to infectious virus, is the same direction mainstream virology independently infers for the origin of retroviruses (Malik et al., 2000). Following the Flood, and especially after the dispersal at Babel, rapid population fragmentation produced the pattern now observed: fixed ERVs shared across all humans or entire kinds, a functional and deeply integrated core clearly part of the original design; and unfixed ERVs, variable across individuals, often tied to post-Flood viral activity, and by turns beneficial, neutral, or harmful. This mixed pattern is not an embarrassment for the framework. It is what the framework predicts for a designed genome now operating under corruption.

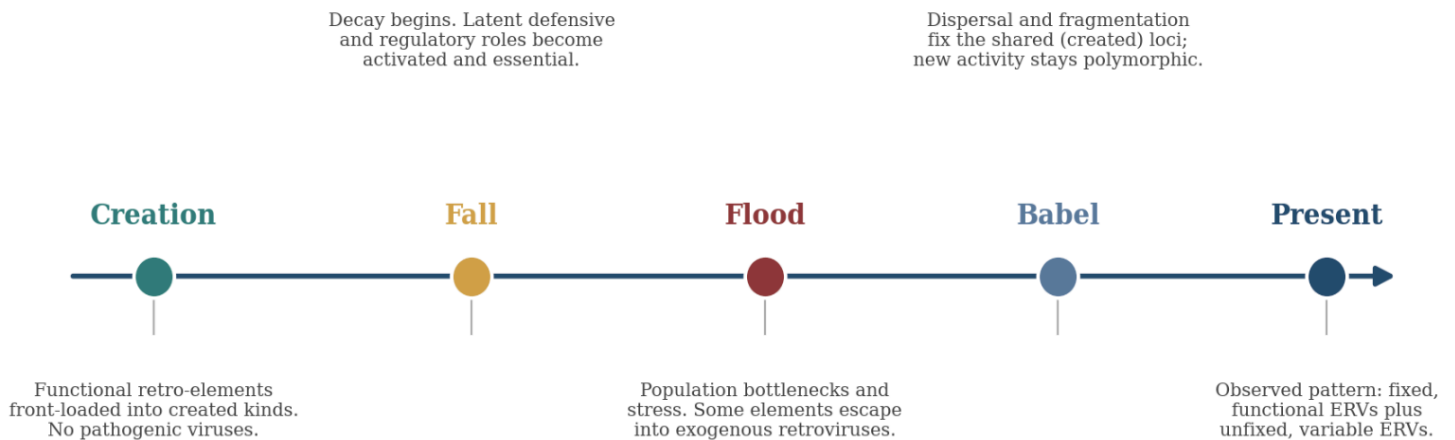


Figure 4. A biblical-historical framework for the state of the genome, from creation through the Fall, the Flood, and the dispersal at Babel to the present. Fixed and unfixed ERVs are predicted by this history rather than accommodated after the fact.

12. Conclusion

The real issue in the ERV debate is origin and interpretation, and on both the design and escape model is the stronger account. It does not flinch from the data. It grants that a functional subset of ERVs is real and essential, that most ERV sequences are degraded, that integration is nonrandom, and that orthologous loci record a genuine shared genomic history. Having granted all of that, it still explains the full pattern more economically than the viral-fossil narrative does.

Consider what each account must carry. The viral-fossil reading must treat the pervasive, sometimes essential functionality of ERVs as a long chain of fortunate accidents; must move rather than pay the explanatory debt for where LTR regulatory content came from; must accept, along with mainstream virology, that retroviruses descend from host retroelements, which already concedes the design model's direction; and must attach to the orthologous-locus data a deep-time chronology that the data themselves do not supply. The design and escape model carries none of these burdens. The functional core was built to function. The viral-like architecture is a requirement of defense and mimicry, not a confession of ancestry. In this model, exogenous retroviruses in circulation are corrupted derivatives of host retroelements. Feline ERV-DCs do not prove that universal origin claim, but they demonstrate the required mechanistic permeability: an endogenous copy can remain infectious, reintegrate, and donate sequence to a recombinant virus. And the shared orthologous loci are exactly what a designed genome, inherited within kinds and later fragmented at Babel, would display.

The strongest argument the other side possesses, orthology, does not survive contact as a proof of unguided common descent. It establishes shared history, and shared history within a kind is common ground, not a difficulty. The PTERV1 comparison demonstrates the necessary distinction empirically. Related retroviral sequences, and even apparent overlaps at BAC-scale resolution, did not establish identical insertion sites; improved assemblies placed 99.8% of 540 PTERV1 elements at nonorthologous positions and confirmed only one chimpanzee–gorilla ortholog (Kronenberg et al., 2018;

Yohn et al., 2005). The result shows why shared insertion must be demonstrated from exact boundaries and homologous flanking sequence rather than inferred from ERV family identity or regional proximity. Stretched across the boundaries between kinds, the same positional data are predicted by common design of functional modules, and the one pattern that could truly discriminate, a long series of shared neutral lesions with no function, has never been established locus by locus on modern assemblies. Until it is, the orthologous-locus pattern is fully consistent with design and the predictions made based on the model, and the deep-time chronology usually bolted onto it remains the assumption, not a result. Evolution has already had to retrofit the failed predictions regarding ERVs as JUNK DNA long ago, while it was creationists who made the correct predictions regarding them (Nailor M., 2026).

Endogenous retroviruses, then, are not the ancient dead fossil remnants of deep evolutionary descent. They are among the clearest genomic evidences of a genome that was designed to function, that carries the viral-like architecture its defensive work requires, and that now operates under corruption. Read honestly and at full strength, the evidence does not merely permit the design interpretation; it fits it better than the account it is usually said to refute. Until the evolutionary account can supply the mechanism and the history it currently assumes, that is where the weight of the evidence lies.

References

- Anai, Y., Ochi, H., Watanabe, S., Nakagawa, S., Kawamura, M., Gojobori, T., & Nishigaki, K. (2012). Infectious endogenous retroviruses in cats and emergence of recombinant viruses. *Journal of Virology*, 86(16), 8634–8644. <https://doi.org/10.1128/JVI.00280-12>
- Best, S., Le Tissier, P., Towers, G., & Stoye, J. P. (1996). Positional cloning of the mouse retrovirus restriction gene Fv1. *Nature*, 382, 826–829.
- Blaise, S., de Parseval, N., Bénit, L., & Heidmann, T. (2003). Genomewide screening for fusogenic human endogenous retrovirus envelopes identifies syncytin 2, a gene conserved on primate evolution. *Proceedings of the National Academy of Sciences*, 100, 13013–13018.
- Boeke, J. D., & Stoye, J. P. (1997). Retrotransposons, endogenous retroviruses, and the evolution of retroelements. In J. M. Coffin, S. H. Hughes, & H. E. Varmus (Eds.), *Retroviruses* (pp. 343–435). Cold Spring Harbor Laboratory Press.
- Chen, M., Huang, X., Wang, C., Wang, S., Jia, L., & Li, L. (2024). Endogenous retroviral solo-LTRs in human genome. *Frontiers in Genetics*, 15, 1358078. <https://doi.org/10.3389/fgene.2024.1358078>
- Chuong, E. B., Elde, N. C., & Feschotte, C. (2016). Regulatory evolution of innate immunity through co-option of endogenous retroviruses. *Science*, 351(6277), 1083–1087. <https://doi.org/10.1126/science.aad5497>
- Chuong, E. B., Elde, N. C., & Feschotte, C. (2017). Regulatory activities of transposable elements: From conflicts to benefits. *Nature Reviews Genetics*, 18, 71–86.
- Conley, A. B., Piriyaongsa, J., & Jordan, I. K. (2008). Retroviral promoters in the human genome. *Bioinformatics*, 24(14), 1563–1567. <https://doi.org/10.1093/bioinformatics/btn243>
- Dunn, C. A., Medstrand, P., & Mager, D. L. (2003). An endogenous retroviral long terminal repeat is the dominant promoter for human β 1,3-galactosyltransferase 5 in the colon. *Proceedings of the National Academy of Sciences*, 100(22), 12841–12846. <https://doi.org/10.1073/pnas.2134464100>
- Dunn, C. A., van de Lagemaat, L. N., Baillie, G. J., & Mager, D. L. (2005). Endogenous retrovirus long terminal repeats as ready-to-use mobile promoters: The case of primate β 3GAL-T5. *Gene*, 364, 2–12. <https://doi.org/10.1016/j.gene.2005.05.045>
- Dupressoir, A., Vernochet, C., Bawa, O., Harper, F., Pierron, G., Opolon, P., & Heidmann, T. (2009). Syncytin-A knockout mice demonstrate the critical role in placentation of a fusogenic, endogenous retrovirus-derived envelope gene. *Proceedings of the National Academy of Sciences*, 106, 12127–12132.
- Feschotte, C. (2008). Transposable elements and the evolution of regulatory networks. *Nature Reviews Genetics*, 9, 397–405.
- Grandi, N., Cadeddu, M., Blomberg, J., & Tramontano, E. (2016). Contribution of type W human endogenous retroviruses to the human genome: Characterization of HERV-W proviral insertions and processed pseudogenes. *Retrovirology*, 13, 67. <https://doi.org/10.1186/s12977-016-0301-x>
- Grandi, N., Cadeddu, M., Blomberg, J., Mayer, J., & Tramontano, E. (2018). HERV-W group evolutionary history in non-human primates: Characterization of ERV-W orthologs in Catarrhini and related ERV groups in Platyrrhini. *BMC Evolutionary Biology*, 18, 6. <https://doi.org/10.1186/s12862-018-1125-1>
- Grow, E. J., Flynn, R. A., Chavez, S. L., Bayless, N. L., Wossidlo, M., Wesche, D. J., Martin, L., et al. (2015). Intrinsic retroviral reactivation in human preimplantation embryos and pluripotent cells. *Nature*, 522, 221–225.
- Hayward, A. (2017). Origin of the retroviruses: When, where, and how? *Current Opinion in Virology*, 25, 23–27. <https://doi.org/10.1016/j.coviro.2017.06.006>
- Hughes, J. F., & Coffin, J. M. (2004). Human endogenous retrovirus K solo-LTR formation and insertional polymorphisms: Implications for human and viral evolution. *Proceedings of the National Academy of Sciences*, 101(6), 1668–1672. <https://doi.org/10.1073/pnas.0307885100>
- Johnson, W. E., & Coffin, J. M. (1999). Constructing primate phylogenies from ancient retrovirus sequences. *Proceedings of the National Academy of Sciences*, 96(18), 10254–10260. <https://doi.org/10.1073/pnas.96.18.10254>
- Kronenberg, Z. N., Fiddes, I. T., Gordon, D., Murali, S., Cantsilieris, S., Meyerson, O. S., Underwood, J. G., Nelson, B. J., Chaisson, M. J. P., Dougherty, M. L., Munson, K. M., Hastie, A. R., Diekhans, M., Hormozdiari, F., Lorusso, N., Hoekzema, K., Qiu, R., Clark, K., Raja, A., . . . Eichler, E. E. (2018). High-resolution comparative analysis of great ape genomes. *Science*, 360(6393), eaar6343. <https://doi.org/10.1126/science.aar6343>

- Lu, X., Sachs, F., Ramsay, L., Jacques, P.-É., Göke, J., Bourque, G., & Ng, H.-H. (2014). The retrovirus HERVH is a long noncoding RNA required for human embryonic stem cell identity. *Nature Structural & Molecular Biology*, 21(4), 423–425. <https://doi.org/10.1038/nsmb.2799>
- Malik, H. S., Henikoff, S., & Eickbush, T. H. (2000). Poised for contagion: Evolutionary origins of the infectious abilities of invertebrate retroviruses. *Genome Research*, 10(9), 1307–1318. <https://doi.org/10.1101/gr.145000>
- Mi, S., Lee, X., Li, X., Veldman, G. M., Finnerty, H., Racie, L., et al. (2000). Syncytin is a captive retroviral envelope protein involved in human placental morphogenesis. *Nature*, 403(6771), 785–789. <https://doi.org/10.1038/35001608>
- Mitchell, R. S., Beitzel, B. F., Schroder, A. R. W., Shinn, P., Chen, H., Berry, C. C., Ecker, J. R., & Bushman, F. D. (2004). Retroviral DNA integration: ASLV, HIV, and MLV show distinct target site preferences. *PLOS Biology*, 2(8), e234. <https://doi.org/10.1371/journal.pbio.0020234>
- Nailor, M., & Budinsky, D. (2026). Predictions on trial: Comparing creation and evolutionary predictions across biology and development. *Truth in Research*, 1(1). <https://doi.org/10.67690/p13n5z85>
- Nethe, M., Berkhout, B., & van der Kuyl, A. C. (2005). Retroviral superinfection resistance. *Retrovirology*, 2, 52.
- Rebollo, R., Romanish, M. T., & Mager, D. L. (2012). Transposable elements: An abundant and natural source of regulatory sequences for host genes. *Annual Review of Genetics*, 46, 21–42.
- Romanish, M. T., Lock, W. M., van de Lagemaat, L. N., Dunn, C. A., & Mager, D. L. (2007). Repeated recruitment of LTR retrotransposons as promoters by the anti-apoptotic locus NAIP during mammalian evolution. *PLOS Genetics*, 3(1), e10. <https://doi.org/10.1371/journal.pgen.0030010>
- Stoye, J. P. (2006). Koala retrovirus: A genome invasion in real time. *Genome Biology*, 7, 241.
- Simmons, G. S., Young, P. R., Hanger, J. J., Jones, K., Clarke, D., McKee, J. J., & Meers, J. (2012). Prevalence of koala retrovirus in geographically diverse populations in Australia. *Australian Veterinary Journal*, 90(10), 404–409. <https://doi.org/10.1111/j.1751-0813.2012.00964.x>
- Tarlinton, R. E., Meers, J., & Young, P. R. (2006). Retroviral invasion of the koala genome. *Nature*, 442, 79–81.
- van der Kuyl, A. C., Dekker, J. T., & Goudsmit, J. (1999). Discovery of a new endogenous type C retrovirus (FcEV) in cats: Evidence for RD-114 being an FcEV(Gag-Pol)/baboon endogenous virus BaEV(Env) recombinant. *Journal of Virology*, 73(10), 7994–8002. <https://doi.org/10.1128/JVI.73.10.7994-8002.1999>
- van der Kuyl, A. C. (2025). Mutation rate variation and other challenges in 2-LTR dating of primate endogenous retrovirus integrations. *Journal of Molecular Evolution*, 93(1), 62–82. <https://doi.org/10.1007/s00239-024-10225-5>
- Wang, T., Zeng, J., Lowe, C. B., Sellers, R. G., Salama, S. R., Yang, M., Burgess, S. M., Brachmann, R. K., & Haussler, D. (2007). Species-specific endogenous retroviruses shape the transcriptional network of the human tumor suppressor protein p53. *Proceedings of the National Academy of Sciences*, 104, 18613–18618.
- Withers-Ward, E. S., Kitamura, Y., Barnes, J. P., & Coffin, J. M. (1994). Distribution of targets for avian retrovirus DNA integration in vivo. *Genes & Development*, 8(12), 1473–1487. <https://doi.org/10.1101/gad.8.12.1473>
- Yohn, C. T., Jiang, Z., McGrath, S. D., Hayden, K. E., Khaitovich, P., Johnson, M. E., Eichler, M. Y., McPherson, J. D., Zhao, S., Pääbo, S., & Eichler, E. E. (2005). Lineage-specific expansions of retroviral insertions within the genomes of African great apes but not humans and orangutans. *PLOS Biology*, 3(4), e110. <https://doi.org/10.1371/journal.pbio.0030110>