What has the Presupposition of Evolutionary Science Done for Research on ERVs?

Evolutionists claim that creationists lack the ability to properly assess biological sciences due to their presupposition that God created life and that common descent is nonsense.

We submit that the opposite is true and that evolutionary-based presuppositions have resulted in a general lack of knowledge and a grievous hindrance in scientific research, discovery, and progress.

In this following quote, note that Francis Collins states, "It's a radical concept, one that a lot of scientists aren't very happy with …" Why in the world would any scientist NOT be excited by a new discovery? It's because many evolutionists are so zealous in their belief of evolution, that they are angered and threatened by any contradictory evidence that might challenge any of its doctrine. How very pathetic …:

- "'It's a radical concept, one that a lot of scientists aren't very happy with,' said Francis S. Collins, director of the National Human Genome Research Institute. 'But the scientific community is going to have to rethink what genes are, what they do and don't do, and how the genome's functional elements have evolved.'
- 'I think we're all pretty awed by what we're seeing,' Collins said. 'It amounts to a scientific revolution.'
- For half a century, the core concept in biology has been that every cell carries within its nucleus a full set of DNA, including genes. Each gene, in turn, holds coded instructions for assembling a particular protein, the stuff that keeps organisms chugging along.
- As a result, genes were assigned an almost divine role in biological 'dogma,' thought to govern not only such physical characteristics as eye color or hair texture, but even much more complicated characteristics, such as behavior or psychology. Genes were assigned blame for illness. Genes were credited for robust health. Genes were said to be the source of the mutations that underlay evolution.
- But the picture now emerging is more complicated, one in which illness, health, and evolutionary change appear to be the work of almost fantastical coordination between genes and swaths of DNA previously written off as junk.
- 'If the surprising amount of RNA transcribed from genomic 'junk' proves to be a powerful regulator of genes, understanding it will be critical in the fight against genetic disease, medical researchers predict. A big push is underway, for example, to develop so-called 'RNA interference' drugs, designed to turn off gene activity by mimicking the effects of RNA.'
- 'For medicine, it could be good news if disease is mainly caused by 'regulators' " in the genome, not mutations in genes themselves,' said Lander. 'It suggests that [cures] might be a matter of tweaking the controls – turning them up here, dialing them down there. Nothing about the gene is broken, but the dial may be powered up too high or turned low.'"
- http://www.boston.com/news/globe/health_science/articles/2007/09/
- 24/dna_unraveled/?page=4

Are Endogenous Retroviral Sequences (ERVs) Evidence for Evolution?

Don't be put off by the name. *Endogenous* means **something that comes from within the organism or cell.** This helps us understand that an **Endogenous RetroViral** sequence is a piece of DNA which was **put in** by a virus.

This is evolution's strongest case, so pay attention. It's easy to debunk when you know the theory.

Evolutionists think that a type of virus, called a 'retrovirus', once inserted DNA into our ape ancestor. Scientists have noticed that chimps and humans have these bits of viral ERVs at similar spots in our DNA. So they say: *our common ancestor acquired these ERVs, and since humans and chimps are closely related, we should have them in similar spots in our genomes.* We do, but this isn't a problem. If we and chimps didn't evolve from a common ancestor (which first acquired the ERVs), how is it possible then that we and chimps have ERVs in almost the same locations? The only plausible explanation, evolutionists say, is evolution. However, I can prove that ERVs were not put in by retroviruses, and that this is not evidence for the theory of evolution. If you are new to ERV's watch this https://www.youtube.com/watch?v=J6RKRyJlRSc

First, all viruses are complex parasites that cannot survive independently from a host cell. They act as delivery mechanisms that insert their viral genetic information into host cells, which is where they reside and reproduce:

- "Viruses are nanomachines built within the cell factory and designed to invade neighboring cells. Their building relies on timely and dynamically regulated assembly of individual components, including nucleic acids, proteins, and lipids, in specific cell locations. Their invading strategies rely on complex interactions with the cell plasma

membrane, involving several viruses and cellular proteins organized in dynamic complexes that are able to mediate viral penetration into the cell interior without rupturing the outside-inside plasma membrane frontier required for cell survival." American Society for Microbiology, "Virus Entry, Assembly, Budding, and Membrane Rafts (CONCLUDING REMARKS AND PROSPECTS)," June 2003. http://mmbr.asm.org/cgi/content/full/67/2/226

"We report the existence of 51,197 ERV-derived promoter sequences that initiate transcription within the human genome, including 1,743 cases where transcription is initiated from ERV sequences that are located in gene proximal promoter or 5' untranslated regions (UTRs)." https://www.ncbi.nlm.nih.gov/pubmed/18535086

"Now it appears that another level of evolution occurs that is not driven by point mutations. Instead, retroviruses insert DNA sequences and rearrange the genome, which leads to changes in gene regulation and expression. If such a change in gene regulation is beneficial, it is passed onto future generations." http://www.physorg.com/news114266805.html

The previous quote is very telling. There are tens of thousands of ERVs in human and chimp genomes. Yet they tell us they mostly are NOT beneficial, yet this article tells us the exact opposite and they will ONLY be passed on IF THEY ARE Beneficial!

"Our analysis revealed that retroviral sequences in the human genome encode tens-of-thousands of active promoters; transcribed ERV sequences **correspond to 1.16% of the human genome sequence** … and PET tags that capture transcripts initiated from ERVs cover 22.4% of the genome." https://academic.oup.com/bioinformatics/article/24/14/1563/182042

As we can see, it has been discovered that ERVs aid transcription in just **one fifth of the human genome.**

"The ancient retroviruses … helped a gene called p53 become an important "master gene regulator" ERVs jumped into new positions throughout the human genome and spread numerous copies of repetitive DNA sequences that allowed p53 to regulate many other genes, the team contends… Thus, p53 was crowned "guardian of the genome," as biologists now call it. Its job is to coordinate the surveillance system that monitors the well-being of cells. Indeed, p53 is so important that when it fails, cancer often results. About **half of all human tumors contain a mutated or defective p53 gene."** http://www.physorg.com/news114266805.html

"We report that human ERVs actively shape the p53 transcriptional network in a species-specific manner… At least one ERV insertion likely reshaped the transcriptional landscape of its surrounding genomic area and was instrumental in creating a new gene that became part of the human-specific p53 regulatory network…" http://www.pnas.org/cgi/content/full/104/47/18613

"Taken together, our findings suggest that HERVs behave like normal cellular genes and are a permanent component of the transcriptome of a cell." http://jvi.asm.org/cgi/content/full/79/1/341

One scientist said about junk DNA, of which ERVs are all part: "The failure to recognize the full implications of this – particularly the possibility that the intervening noncoding sequences may be transmitting parallel information … may well go down as one of the biggest mistakes in the history of molecular biology."
https://www.researchgate.net/publication/9045749_The_Unseen_Genome_Gems_among_the_Junk
ERVs are important in much of the genome.

Apoptosis

<Apoptosis *is a process of the body that kills dangerous cells. Foreign DNA injected into a cell by a virus would surely trigger the cell to commit suicide (apoptosis). So if tens of thousands of ERVs were really introduced by retroviruses, we should expect apoptosis to have gotten ridden of most of them long ago. The fact that we have so many ERVs indicates they could not have been injected by retroviruses – apoptosis should have ridden most of the cells.*

"Apoptosis, or programmed cell death, is a normal component of the development and health of multicellular organisms. Cells die in response to a variety of stimuli and during apoptosis they do so in a controlled, regulated fashion. The latter occurs when T-cells recognize damaged or virus infected cells and initiate apoptosis in order to prevent damaged cells from becoming neoplastic (cancerous) or virus-infected cells from spreading the infection."
http://www.sgul.ac.uk/depts/immunology/~dash/apoptosis/

Because *apoptosis* would kill most ERV-containing cells, why is it that we still have so many ERVs hundreds of thousands of years after supposedly being inserted by retroviruses?

Now, evolutionists might ask why the positioning of human ERVs are so similar to the positioning of chimp ERVs if viruses didn't put them in the common ancestor? The answer seems to be in the overall similarity of the human and chimp genomes: since our DNA is fairly similar to chimps https://www.newscientist.com/article/dn2833-human-chimp-dna-difference-trebled/ we'd actually expect most ERVs to be in similar spots.

Similar ERVs in Unrelated Organisms

Another problem for the evolutionary explanation is that very similar ERVs exist in totally unrelated animals.
"We have sequenced and characterized an endogenous type D retrovirus, which we have named TvERV(D), from the genome of an Australian marsupial, the common brushtail possum (Trichosurus vulpecula). Intact TvERV(D) gag, pro, pol, and env open reading frames were detected in the possum genome. TvERV(D) was classified as a type D retrovirus, most closely related to those of Old World monkeys, New World monkeys, and mice, based on phylogenetic analyses and genetic organization." http://jvi.asm.org/cgi/content/full/75/5/249
"For instance gamma-retrovirus was isolated from trophoblastic cells of the baboon placenta. This virus was found to be very closely related antigenically and by sequence homology to the endogenous RD114 virus in cats (which is itself unrelated to endogenous FeLV). Benveniste and Todaro observed, like we did for jungle fowl, that only certain species of the cat genus, Felis, possessed this endogenous genome related to the baboon ERV. In contrast, all species of baboons carry this virus so it would appear to have been present in the germ line of primates much longer than in cats. Thus it seems evident that a horizontal, infectious event occurred to transfer the virus from baboons to cats, whereupon it became endogenous in the new species." http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1617120
How do the authors "explain" this?
"Since cats would be quite likely to scavenge and feed on baboon placentae, a possible exposure to the virus can be envisioned." Possible but keep in mind my research shows Baboons are actually predators to cats and eat them! http://www.pezulagolfestatehoa.com/board/Misconceptions%20about%20Baboons.pdf Besides, Mother **cats** will **eat** some of the **placentas** during birthing.

More Problems

It is interesting to note that ERVs are different from the retroviral genomes from which they are supposed to have originated from. Evolutionists usually explain this away by claiming that the ERV sequences have evolved to the point where they are quite different from their ancestral genomes. If this is so, then there is consequently very little to lead us to the conclusion that ERVs are derived from retroviruses.
If ERVs really are a product of retroviruses, how could they have been inserted into reproductive cells thousands of times without fatal damage to the host? Having healthy and strong reproductive cells is mandatory to produce a viable zygote, so why would viral-infected reproductive cells be considered more fit than ones without ERVs? Furthermore, how could they survive for hundreds of thousands of years in two different species?
Changes to the reproductive cells are rare and often harm the animal. So why should we believe that ERVs were inserted many thousands of times and no real problems arose back then but do now? It's not logical.
"In short, the notion that molecules of germ cells … are in states of perpetual change is not, in our present understanding of cell biology, tenable. This doesn't mean that "molecular change" does not occur; only that mechanisms provoking such change in germ cells are likely instantaneous and stochastic and probably often lethal (Maresca and Schwartz 2006) — which will preclude their persistence into future generations."http://www.mitpressjournals.org/doi/abs/10.1162/biot.2006.1.4.357
How could it be that unrelated ERVs in different species created essentially the same gene?
"ERVWE1/Syncytin-1 and ERVFRDE1/Syncytin-2 are specific to primates and thus do not exist in other placentae. However, this apparent endogenous retrovirus hijacking for placentation use is not restricted to the primates. Indeed two unique endogenous envelope genes of retroviral origin have been found in the mouse, i.e. Syncytin-A and –B … Altogether the data strongly argue for convergent evolution of endogenous retroviral envelopes to serve for placentation in mammals."[http://AtlasGeneticsOncology.org/Genes/ERVWE1ID40497ch7q21.html
One may argue that convergent evolution is the answer, as the author of the quote did, but this explanation has no real scientific basis. Convergent evolution is only ever used to explain similarities between organisms that are otherwise unrelated. But then why shouldn't we consign other 'proof-of-evolution' similarities to convergent evolution?

About 85-90% of ERV elements are promoter and enhancer elements called solitary long terminal repeats (solo LTRs). The remaining 10-15% represent genetic sequences typically known to identify ERVs (gag, pol and env genes flanked by two LTRs). Because ERV sequences differ and have less genetic material than exogenous sequences, evolutionists claim that mutations, deletions, and homologous recombination altered ERV sequences so that they are now considered only remnants of previous exogenous infections: "Most (85%) of the LTR retroposon-derived 'fossils' consist only of an isolated LTR, with the internal sequence having been lost by homologous recombination between the flanking LTRs

…Finally, LTR retroposons appear to be teetering on the brink of extinction, if they have not already succumbed." http://www.nature.com/nature/journal/v409/n6822/full/409860a0.html

"ERVs become defective over time due to frameshift or nonsense mutations introduced during host DNA replication or via recombinational deletion of the internal region to leave a solo LTR structure. Solo LTRs are approximately tenfold more abundant than their undeleted, full-length counterparts." http://jvi.asm.org/cgi/content/full/79/19/12507

"Over time, replication-competent ERVs accumulate in-frame stop codons and frame-shift mutations as a result of host DNA replication and, within the human genome sequence, these processes have led to the inactivation of almost every element. Another mechanism by which ERVs are inactivated is via recombinational deletion between the two viral LTRs, which removes the internal region leaving a solo LTR structure. Solo LTRs are typically 10–100 times more numerous than their more intact, undeleted counterparts." http://www.pnas.org/cgi/content/full/101/14/4894

"They are thought to be remnants of ancient germ line infections by exogenous retroviruses …The majority of these LTR elements, however, lack sequence similarity to retroviral genes within their internal region or constitute solitary LTRs. About 40 families identified so far have at least some members that show discernible homology to coding regions of retroviruses, but most of them have not yet been analyzed in depth." http://jvi.asm.org/cgi/content/full/79/1/341

Because ERVs were originally cast off as 'junk' DNA, evolutionary-based research was primarily geared towards looking for how they **might relate to disease**. While no direct correlation was ever found, other research discovered that essential genetic material is directly attributed to ERVs. That's when the confusion began:

"The evolutionary origin of viruses has long fascinated evolutionary biologists. Are they remnants of an ancestral lifestyle, or more recent escapees from traditional genomes?" http://genetics.plosjournals.org/perlserv/?request=get-document&doi=10.1371%2Fjournal.pgen.0010044

- "Exogenous retroviruses may have originated from ERVs and ERV-Ls in particular may represent an intermediate between retrotransposons and exogenous viruses." http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=524511
- "Technologies to interrogate the relationship between transcription factors and repetitive sequences are currently much limited. Most genomewide analyses of gene regulation, transcription factor binding sites, and other functional genomics, including the ENCODE project designed to catalog all sequence elements in 1% of the human genome, tend to ignore the functional aspect of repetitive elements. The results reported here suggest that it may be valuable to take a deeper look at the role of these elements in the evolution of gene regulatory networks." http://www.pnas.org/cgi/content/full/104/47/18613
- "However, the activities (expression levels) of individual HERV sequences are mostly unknown …The data suggests that in many cases the retroviral sequence has been used as **a building block for something else** than retroviral proteins, for example human gene exons. However, for some HERVs there may be an alternative explanation: the retroviral transcripts may have RNA-mediated activities. We need to study the active elements more closely to discover the possible functions of retroviral transcripts." http://www.biomedcentral.com/1471-2105/8/s2/s11
- "These are endogenous viruses and some animal species have thousands of copies of these A-type viruses in their chromosomal DNA. Their function remains unknown." http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=mmed.section.3287
- "The expression of endogenous retroviruses and their control is not well understood." http://www3.niaid.nih.gov/labs/aboutlabs/lpvd/retroviralMolecularBiologySection/

FUNDAMENTAL PROBLEMS FOR ERVS BEING CONSIDERED GERMLINE INFECTIONS OF EXOGENOUS RETROVIRUSES INSTEAD OF INTRINSIC ESSENTIAL GENETIC MATERIAL

By Chance, How Did ERV Related Elements Insert Themselves into Germ Cells Thousands of Times Without Fatalistic Damage to the Host?

Genetic alterations to germ cells are rare and have been mostly found to **harm overall genetic fitness**, not improve it. What would make retroviruses an exception?

- "In short, the notion that molecules of germ cells … are in states of perpetual change is not, in our present understanding of cell biology, tenable. This doesn't mean that "molecular change" does not occur; only that

mechanisms provoking such change in germ cells are likely instantaneous and stochastic and probably often lethal (Maresca and Schwartz 2006) – which will preclude their persistence into future generations."

- http://www.mitpressjournals.org/doi/abs/10.1162/biot.2006.1.4.357

Having healthy and strong germ cells is mandatory to produce a viable zygote, so why would harmful viral-infected egg/sperm cells be considered more fit (positive selection) versus ones without retroviral DNA? Apoptosis is an accepted biological phenomenon, so why wouldn't most germ cells with viral-infected DNA be eliminated?

"Apoptosis, or programmed cell death, is a normal component of the development and health of multicellular organisms. Cells die in response to a variety of stimuli and during apoptosis they do so in a controlled, regulated fashion … The latter occurs when T-cells recognise damaged or virus infected cells and initiate apoptosis in order to prevent damaged cells from becoming neoplastic (cancerous) or virus-infected cells from spreading the infection."

- http://www.sgul.ac.uk/depts/immunology/~dash/apoptosis/
- "Apoptosis occurs during the normal development of multicellular organisms and continues throughout adult life."
- http://www.sgul.ac.uk/depts/immunology/%7Edash/apoptosis/disease.htm

Evolutionists claim that retroviruses were in a dormant cycle (lysogenic) upon insertion and were intact before mutations, deletions, and recombination deleted and disabled them. What prohibited the thousands of retroviruses from changing into an infectious cycle (lytic), thus weakening or killing the embryo (especially in hosts with long gestation periods) and weakening or killing young and adult hosts?

Why would populations with harmful viral-infected germ lines be under positive selection and become prevalent and "fixed" over non-infected populations? (The supposed 'evolution' of ERV elements changing into beneficial genetic material must be assumed to have occurred after fixation.) Geneticists have problems with controlling retroviruses (vectors) for gene therapy, so how would ERV elements randomly invade healthy germ and somatic cells without damage, thus be eliminated by 'purifying selection'?

- "Scientists have tried to take advantage of the virus's biology and manipulate its genome to remove human disease-causing genes and insert therapeutic genes. However, viruses, while effective, introduce other problems to the body, such as toxicity, immune and inflammatory responses, and gene control and targeting issues."
- http://www.genome.gov/10004764

There are at least 98,000 different ERVs sequences and 158,000 ERV-like elements just in the human genome. Why is it that only ERVs have supposedly invaded germ cells hundreds of thousands of times, but no other virus has been discovered to do the same?

How is it that ERVS are Considered Copies of Disease Producing Exogenous Retroviruses but None Have Been Proven to Directly Cause Disease?

- "Whether other examples of pathogenic endogenous retroviruses will be found, particularly in humans, is unknown. Numerous reports of retroviral particles in human tumor specimens have not been verified. For now, it appears that few, if any, replication-competent human endogenous retroviruses capable of causing disease exist."
- http://www.ncbi.nlm.nih.gov/books/bv.fcgi?rid=cmed.section.4250
- "HERVs have frequently been proposed as etiological cofactors in chronic diseases such as cancer, autoimmunity and neurological disease. Unfortunately, despite intense effort from many groups, there remains little direct evidence to support these claims, and moreover some studies have served only to muddy the waters for others."
- http://genomebiology.com/2001/2/6/reviews/1017
- "Retroviral activity might cause disease … although a causal role of HERVs in these conditions is highly uncertain."
- http://www.biomedcentral.com/1471-2105/8/s2/s11
- "The causative or disease-promoting association of HERV protein expression with germ cell tumors, mammary carcinomas, various autoimmune diseases, and neurological disorders, such as schizophrenia, has yet to be conclusively demonstrated."
- http://www.pnas.org/cgi/content/full/101/suppl_2/14572#REF65
- "Although it is generally accepted that the integration of retroelements can cause significant harm by disrupting or dysregulating essential genes, the role of HERV expression in the etiology of malignancies and autoimmune and neurologic diseases remains controversial."
- http://www.pnas.org/cgi/content/full/101/suppl_2/14572

- "Overall, while a number of articles highlight some steps in determining the role of HERVs, it will be crucial in the next few years to establish firmly clear associations of HERVs that may contribute to the pathology of disease states and/or aetiology "
- http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1809191
- "Tumor development is a multistep process in which both genetic and epigenetic events cooperate for the emergence of a malignant clone. The possibility that endogenous retroviruses promote the expansion of a neoplastic clone by subverting immune surveillance has been proposed, but remained elusive … Future work should now be aimed at the identification of a possible role of such genes in human tumorigenesis."
- http://cancerres.aacrjournals.org/cgi/content/full/65/7/2588
- "This illustrates that HERV expression is **not automatically higher in malignant tissues**."
- http://www.liebertonline.com/doi/abs/10.1089/aid.2006.22.551?
- cookieSet=1&journalCode=aid

By Chance, What Made ERVS Evolve from Being the Cause of Exogenous Infection into Elements that Resist Exogenous Infection?

- "'But these sorts of ancient interactions may have influenced how humans are able to combat these retroviruses today. These proteins help protect us against current retroviruses.' Indeed, HERV-K may well have helped to shape the modern APOBEC3G defense."
- Rockefeller University, "New Evidence Of Battle Between Humans And Ancient Virus," July 23, 2008, ScienceDaily.
- http://www.sciencedaily.com/releases/2008/07/080721112616.htm
- "We propose a model in which Iris has "switched sides," having been recruited by host genomes to combate baculoviruses and retroviruses, which employ homologous envelope genes to mediate infection."
- http://genetics.plosjournals.org/perlserv/?request=get-document&doi=
- 10.1371%2Fjournal.pgen.0010044
- "These results suggest that the loss of ERV3 mRNA expression is associated with susceptibility to choriocarcinoma." http://www3.interscience.wiley.com/cgi-bin/abstract/112716625/ABSTRACT
- "They also block exogenous retrovirus replication by receptor interference or antisense mRNA."
- http://lib.bioinfo.pl/pmid%3A17199106
- "The HERV-W env gene product has also been shown to block infection by an exogenous retrovirus, suggesting that the expressed HERV-W env gene could have a beneficial function to the host (Ponferrada et al., 2003Down)." http://vir.sgmjournals.org/cgi/content/full/85/5/1203

"Thus, HERV-W Env confers host cell resistance to infection by SNV. This is the first report of a human endogenous retrovirus gene product blocking infection by any exogenous retrovirus."http://www.ncbi.nlm.nih.gov/pubmed/12664292

"A possible biological role hypothesized for ERVs is to help the host resist infections of pathogenic exogenous retroviruses, affording a selective advantage to the host bearing them. For instance, some avian and murine ERVs can block infection of related exogenous retroviruses at entry by receptor interference; mouse Fv-1 blocks infection at a preintegration step, also can be viewed as an ERV." http://www.pnas.org/cgi/content/full/101/30/11117

"Of note in this respect is the observation that endogenous Jaagsiekte sheep retrovirus (JSRV) blocks the entry of the corresponding exogenous virus." http://www.pnas.org/cgi/content/full/101/suppl_2/14572#REF66

"However, "in the case of both Fv4 and Rmcf, the mode of defense is by the domesticated env gene blocking the receptor required for retrovirus entry." Study

By Chance, What Made ERV Elements Change From Viral Activities to Cellular Activities and Create New Essential Genes?

- *"Apart from detecting ERVs, the thesis demonstrates their expression in bovine tissue for the first time. It involves, moreover, a controlled expression and, therefore, valuable for the protection of the host. As shown in the thesis, this expression occurs mainly in the endocrine glands and in embryos, and so could be linked to the protection of the embryo."*
- Basque Research, "PhD Thesis Describes 35 Hitherto Unknown Families Of Endogenous Retroviruses, After Analysing Cattle And Horses," December 1, 2010, Medical News Today.
- http://www.medicalnewstoday.com/articles/209622.php
- "In some cases, transcription factor binding sites that interact with cell type-specific nuclear factors could be identified, demonstrating that the expression of HERVs is regulated in a complex and diverse manner comparable to cellular genes … Our data reveal that the activity of endogenous retroviruses is regulated differentially and is cell type specific, similar to normal gene regulation … Taken together, our findings suggest that HERVs behave like normal cellular genes and are a permanent component of the transcriptome of a cell."
- http://jvi.asm.org/cgi/content/full/79/1/341
- "It appears that each of the functioning genes was originally formed from mobile elements and that in some process of molecular evolution a coding sequence was derived that could be translated into a protein that is of some importance to human biology. In one case (AD7C), the coding sequence is 99% made up of a cluster of Alu sequences. In another example, the gene BNIP3 coding sequence is 97% made up of sequences from an apparent human endogenous retrovirus. The Syncytin gene coding sequence appears to be made from an endogenous retrovirus envelope gene … These observations contribute an additional bit to the growing mass of evidence that indicates that mobile elements/repeats are not always junk and have made important contributions to the "host."
- http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=534736
- Note that syncytin-1 is regulated differently in different tissues and at different times:
- "Syncytin-1 is a captive envelope glycoprotein encoded by one of human endogenous retroviruses W. It is expressed exclusively in the placental trophoblast where it participates in cell-to-cell fusion during differentiation of syncytiotrophobast. In other tissues, however, syncytin-1 expression must be kept in check because inadvertent cell fusion might be dangerous for tissue organization and integrity."
- http://www.ncbi.nlm.nih.gov/sites/entrez?cmd=Retrieve&db=
- PubMed&list_uids=16427621&dopt=Citation

<u>How Could ERVS Create a Specie-Specific Regulatory Network that Controls the Expression of Cells in a Collective Manner?</u>

"The ancient retroviruses–distant relatives of the human immunodeficiency virus (HIV)–helped a gene called p53 become an important "master gene regulator" in primates, according to a study published this week in the online early edition of Proceedings of the National Academy of Sciences … Scientists have long wondered how a master regulator such as p53 gained the ability to turn on and off a broad range of other genes related to cell division, DNA repair, and programmed cell death. How did p53 build its complex and powerful empire, so to speak.

Using the tools of computational genomics, the UCSC team gathered compelling evidence that retroviruses helped out. ERVs jumped into new positions throughout the human genome and spread numerous copies of repetitive DNA sequences that allowed p53 to regulate many other genes, the team contends.

Thus, p53 was crowned "guardian of the genome," as biologists now call it. Its job is to coordinate the surveillance system that monitors the well-being of cells. Indeed, p53 is so important that when it fails, cancer often results. About half of all human tumors contain a mutated or defective p53 gene." http://www.physorg.com/news114266805.html

"We report that human ERVs **actively shape the p53 transcriptional network in a species-specific manner** … At least one ERV insertion likely reshaped the transcriptional landscape of its surrounding genomic area and was instrumental in creating a new gene that became part of the human-specific p53 regulatory network … We discovered a unique distribution pattern of p53 sites within repetitive sequences of the human genome, and several ERV families emerged as being substantially enriched for p53 sites in their LTRs." http://www.pnas.org/cgi/content/full/104/47/18613

- "The reasons why genes are expressed when and where they are in the spatial domains of the developing organism are revealed in network "architecture," that is, in the total aggregate pattern of regulatory linkages.

Definitive regulatory functions emerge only from the architecture of intergenic linkages, and these functions are not visible at the level of any individual genes …
- Gene regulatory networks are inhomogeneous compositions of different kinds of subcircuits, each performing a specific kind of function. This concept is important, because it holds the key to network design principles."
- http://www.pnas.org/cgi/content/full/102/14/4935

By Chance, What Made Unrelated ERVS in Unrelated Species Create Almost the Same Gene (Convergent Evolution)?

- "ERVWE1/Synctin-1 and ERVFRDE1/Syncytin-2 are specific to primates and thus do not exist in other placentae. However, this apparent endogenous retrovirus hijacking for placentation use is not restricted to the primates. Indeed two unique endogenous envelope genes of retroviral origin have been found in the mouse, i.e. Syncytin-A and –B … Altogether the date strongly argues for convergent evolution of endogenous retroviral envelopes to serve for placentation in mammals."
- http://atlasgeneticsoncology.org/Genes/ERVWE1ID40497ch7q21.html

By Chance, What Made Two Unrelated ERV - LTRS Evolve Independently in Creating the Same Regulatory Roles for the Same Gene (Convergent Evolution)?

- "We demonstrate that both the human and rodent neuronal apoptosis inhibitory protein (NAIP) genes, involved in preventing cell death, use different ERV sequences to drive gene expression. Moreover, in each of the primate and rodent lineages, two separate ERVs contribute to NAIP gene expression. This repeated ERV recruitment by NAIP genes throughout evolution is very unlikely to have occurred by chance. We offer a number of potential explanations, including the intriguing possibility that it may be advantageous for anti-cell death genes like NAIP to use ERVs to control their expression. These results support the view that not all retroviral remnants in our genome are simply junk DNA."
- http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1781489#id2960616

By Chance, What Made ERV LTRS Immediately Turn into Essential Gene Regulators Upon Insertion?

- "We also present evidence that the functional TF binding sites of the human b3GAL-T5 LTR promoter were present in the original consensus sequence for this class of LTRs. Upon similar analysis of other ERV sequences, we have concluded that this evolutionary history is shared by certain other LTR gene promoters, and may be a general phenomenon."
- http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6T39-
- 4GXVG6Y-1&_user=10&_rdoc=1&fmt=&_orig=search&_sort=d&view=
- c&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=
- 19c2ea074bec5e7313b36ce1630e38a8

By Chance, What Made LTRS Acquire Transcription Abilities for Essential Genes?

- "Endogenous retrovirus (ERV) elements have been shown to contribute promoter sequences that can initiate transcription of adjacent human genes …
- These data illustrate the potential of retroviral sequences to regulate human transcription on a large scale consistent with a substantial effect of ERVs on the function and evolution of the human genome."
- http://www.ncbi.nlm.nih.gov/pubmed/18535086
- "Despite the general selection against ERVs near genes, the number of examples of promoters and enhancers derived from ERVs is steadily increasing. The evolutionary process of "exaptation" of noncoding functional elements from viruses and transposons to benefit the host is not well understood beyond a few examples."
- http://www.pnas.org/cgi/content/full/104/47/18613
- "HERVs have been found to contribute physiologically in other human tissues. In particular the vast numbers of residual LTRs contain regulatory elements known as promoters, enhancers, silencers and polyadenylation signals that can specifically interact with the cellular expression of proteins. HERV-derived promoters have been found in roughly a quarter of all the human promoter regions so far examined. In many such instances, the viral promoter has only a minor function. However, in 2003, Dunn and colleagues demonstrated that an LTR of the ERV-L family is the dominant promoter of the gene responsible for the enzyme galactosyltransferase in the human colon and small intestine.31 Andersson and her colleagues have produced clear evidence for developmental and physiological roles for HERV-R (also known as ERV-3), showing that it is highly expressed in many human fetal tissues including adrenal cortex, kidney tubules, tongue, heart, liver and central nervous system as well as in the sebaceous glands of normal skin."

- http://www.jrsm.org/cgi/content/full/97/12/560
- "Here, we describe mapping of transcriptional start sites within solitary and proviral LTRs of the HERV-K (HML-2) human-specific subfamily of endogenous retroviruses. Surprisingly, the transcription was initiated predominantly from the very 3¢ termini of the LTR R regions. The data presented here may shed light on adaptive coevolution of human endogenous retroviruses with their host cells."
- http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WXR-
- 4HRMTVY-1&_user=10&_rdoc=1&_fmt=&_orig=search&_sort=d&view=c&_acct=
- C000050221&_version=1&_urlVersion=0&_userid=10&md5=
- 9a8359dcf66d451223e17972617f87c3
- "While both LTRs were shown to promote transcription in vivo and in vitro, their respective activity and tissue specificity **appeared to differ even though they shared a high degree of sequence identity** … The results from this study illustrate how slight variations in transcriptional regulatory sequences can have a profound effect on promoter activity and demonstrate the complex regulatory effects of human endogenous retrovirus elements on human gene expression." http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=164795
- "By germline insertion, a long terminal repeat (LTR) of an intracisternal A-particle type IAP retrovirus has overtaken the transcriptional control of the rat oncomodulin (OM) gene, which codes for a high affinity Ca2+-binding protein with modulatory capacity."
- http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WB5-4J3P0MR-5&_
- user=10&_origUdi=B6T39-4GXVG6Y-1&_fmt=high&_coverDate=03%2F01%2F2006&_
- rdoc=1&_orig=article&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5=
- db4a9ec49fd48c70f7bdbf5d97495bb0

Where is the Proof that ERV or LTRS can "Self-Replicate" and Why Don't We See them Doing it Now?

- "During primate evolution, the LTRs were self-replicated and stably integrated into various host chromosomal site."
- http://www.jbc.org/cgi/content/full/280/42/35184

By Chance, What Made the Same ERV Transcribe Differently Between Supposedly 'Closely Related' Species?

- "Based on analysis of finished BAC chimpanzee genome sequence, we characterize a retroviral element (Pan troglodytes endogenous retrovirus 1 [PTERV1]) that has become integrated in the germline of African great ape and Old World monkey species but is absent from humans and Asian ape genomes … Six out of ten of these genes, for which there are expression data, show significant differences in transcript expression between human and chimpanzee."
- http://biology.plosjournals.org/perlserv/?request=get-document&doi=
- 10.1371/journal.pbio.0030110&ct=1
- "We report that human ERVs actively shape the p53 transcriptional network in a species-specific manner."
- http://www.pnas.org/cgi/content/full/104/47/18613
- "Most HERVs are active in at least some tissues, though tissue specificity is common for most elements. We analyzed multiple tissues from several Old World monkeys using retroviral pol-based DNA microarrays and quantitative PCR methods to determine their ERV expression profiles. The results demonstrate that while many ERVs are active in nonhuman primates, overall the tissue expression specificity is unique to each species. Most striking is that while the majority of HERVs analyzed in this study are expressed in human brain, almost none are expressed in Old World monkey brains or are only weakly expressed."
- http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1472034

By Chance, What Made the Same ERV Transcribe Differently Among Different Cell Types Within the Same Organism?

- "Furthermore, there is evidence that transcription of at least some HERV families may be differentially regulated depending on the cell type. Characterization of promoter activities of HERV-K, HERV-H, HERV-E, ERV9, and HERV-W families, the most intensively studied HERVs, revealed specific cell type preferences for each HERV family, and even individual elements of one family showed significant variation in transcription pattern. In some cases, transcription factor binding sites that interact with cell type-specific nuclear factors could be identified, demonstrating that the expression of HERVs is regulated in a complex and diverse manner comparable to cellular genes."
- http://jvi.asm.org/cgi/content/full/79/1/341

Where is the Evidence that Genetic Recombination Creates New Exogenous and Endogenous Retroviruses as well as Novel Beneficial Genetic Elements?

- "The question arises as to whether HERV elements can continue to change our genomic landscape through active retrotransposition or recombination events. While no direct evidence indicates that such events are ongoing in the human genome, members of the HERV-K family appear to be the most likely candidates for playing such a role."
- http://www.genetics.org/cgi/content/full/171/3/1183
- "Although repeated sequence elements such as HERVs have the potential to lead to chromosomal rearrangement through homologous recombination between distant loci, evidence for the generality of this process is lacking."
- http://www.ncbi.nlm.nih.gov/pubmed/11704760?ordinalpos=1&itool=EntrezSystem2.
- PEntrez.Pubmed.Pubmed_ResultsPanel.Pubmed_RVAbstractPlus
- "As for the elimination of the numerous copies produced by such rapid and extensive bursts, it is not yet clear whether recombination occurs continuously through time, thus slowly and regularly decreasing large amounts of DNA, or if there is any mechanism that would activate large recombination events following bursts of amplification, as proposed by some authors (Rabinowicz 2000)."
- http://mbe.oxfordjournals.org/cgi/content/full/20/4/528

Why are there NO Examples of an ERV that Has Been Recently "Endogenized" or Examples of ERVS that Have A Direct Exogenous Counterpart?

(Note: Go to "Examples of biased research/articles" regarding KORV Koala virus.)

- "No current transposition activity of HERVs or endogenization of human exogenous retroviruses has been documented so far." http://www.pnas.org/cgi/content/full/101/suppl_2/14572
- "Most of these elements represent ancient retroviral infections, as evidenced by their wide distribution in primate species, and no infectious counterparts of human endogenous retroviruses (HERVs) are known to exist today."
- http://www.pnas.org/cgi/content/abstract/101/6/1668

If ERV Elements Deteriorate Rapidly, Why Did Only One ERV Remain Active and Continue to Infect All Placental Mammals Over a 70 Million Year Period?

Evolutionists point to "lack of selection pressure" to explain why ERVs deteriorate rapidly, but then point to "bursts" of copy numbers when they need ERVs to be conserved: "Southern blot analysis of a large series of mammalian genomic DNAs shows that HERV-L-related elements—so-called ERV-L—are present among all placental mammals, suggesting that ERV-L elements were already present at least 70 million years ago. … Phylogenetic analyses allowed the establishment of a plausible evolutionary scheme for ERV-L elements, which accounts for the high level of sequence conservation and the widespread dispersion among mammals … One rather striking feature of our results is the observation of a high level of sequence conservation among ERV-L elements. It is generally admitted that such sequence conservation is a common feature of functional genes, whereas noncoding sequences as well as pseudogenes diverge more rapidly, due to the lack of any selection pressure …

Rather, a scheme in which sequence conservation is the unnecessary consequence of the transpositional activity of the transposable element itself (which requires both transcription activity and coding capacity), independent of any possible selective pressure imposed by the host, appears more plausible and would account for the data. According to this scheme, a functional sequence present in an ancestor of mammalian species would survive only if active, simply by generating a sufficiently high number of copies, so that despite the fact that such elements are submitted to genetic drift, as any pseudogene-like sequence, and also to elimination through transposon excision, there still remain functional copies of the founder element. Evidence for the transpositional activity of ERV-L elements is provided by the occurrence of bursts in both the primate and mouse branches, which resulted in large increases in ERV-L copy numbers."

- http://jvi.asm.org/cgi/content/full/73/4/3301

Why Are Closely Related ERV Elements Found in Supposedly Non-Related Species?

"… and two closely related ERV genomes are found in a carnivore (fox) and a ruminant (sheep)." http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1617120

"We have sequenced and characterized an endogenous type D retrovirus, which we have named TvERV(D), from the genome of an Australian marsupial, the common brushtail possum (Trichosurus vulpecula). Intact TvERV(D) gag, pro, pol, and env open reading frames were detected in the possum genome. TvERV(D) was classified as a

type D retrovirus, most closely related to those of Old World monkeys, New World monkeys, and mice, based on phylogenetic analyses and genetic organization."
http://jvi.asm.org/cgi/content/full/75/5/2499

If ERVS Are Intrinsic, Essential and Functional Genetic Elements, Why Wouldn't Integration Sites Be identical?

- "Integration into the host-cell genome is a critical step in the retrovirus life cycle. In particular, the choice of the integration site is crucial for retroviral replication, since integration at a site incompatible for high-level transcription may impair production of the progeny virus … Target site selection might be influenced by several factors, including the function of cellular proteins that interact with integrase, the viral protein that catalyzes the integration reaction. Interestingly, a common functional feature that unifies these cellular co-factors is that, to a different extent, they are all involved in the regulation of chromatin structure or transcription. Inappropriate retroviral integration might lead to insertional mutagenesis and cellular transformation, as recently observed in a gene therapy clinical trial exploiting retroviral vectors for gene transfer into hematopoietic progenitors. Thus, the deeper understanding of the molecular mechanisms regulating integration site selection is also essential for the design of safer and more effective gene transfer vectors."
- http://www.ncbi.nlm.nih.gov/sites/entrez?Db=PubMed&Cmd=ShowDetailView&
- TermToSearch=15168737&ordinalpos=1&itool=EntrezSystem2.PEntrez.Pubmed.
- Pubmed_ResultsPanel.Pubmed_RVAbstractPlus
- "Thus, each of the three retroviruses studied showed unique integration site preferences, suggesting that virus-specific binding of integration complexes to chromatin features likely guides site selection."
- http://www.iscid.org/boards/ubb-get_topic-f-1-t-000167.html
- "While the PIC is capable of directing integration of the viral DNA into any chromosomal location, different retroviruses have clear preferences for integration in or near particular chromosomal features. The determinants of integration site selection are under investigation but may include retrovirus-specific interactions between integrase and tethering factors bound to the host cell chromosomes."
- http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B7CT7-4HJ20NX-5&_user=
- 10&_origUdi=B6WSN-49TP1CG-5&_fmt=high&_coverDate=12%2F31%2F2005&
- _rdoc=1&_orig=article&_acct=C000050221&_version=1&_urlVersion=0&_userid=10&md5
- =373115706749716ff8fdc6aa26485094
- "Replication of retroviruses and retrotransposons depends on selecting a favorable chromosomal site for integration of their genomic DNA. Different retroelements meet this challenge by targeting distinctive chromosomal regions. Despite these differences, recent data hints at a common targeting mechanism —tethering of integration complexes to proteins bound at favorable sites."
- http://www.sciencedirect.com/science?_ob=ArticleURL&_udi=B6WSN-49TP1CG-5&_user=10&_rdoc=1&_fmt=&
- _orig=search&_sort=d&view=c&_acct=C000050221&_version=1&_urlVersion=
- 0&_userid=10&md5=5f51596c964fb5c3abe56a9e60d7ae24
- "Each retrovirus, they discovered, showed distinct preferences for genome integration."
- http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=509317

Do ERV Elements Have Random Integration Sites?

The premise that ERV integration would prove evolution to be true is purely based on presupposition, instead of objective research and empirical evidence. Random or non-random insertion (if it was the case) is impossible to conclude due to an utter lack of realistic testing. To replicate the supposed original insertion, the EXACT genetic sequence of the original integrated ERV must be inserted in closely several related species. This has never been done, nor is it plausible:

- There are no exogenous retroviral counterparts of any ERV. Therefore, it is impossible to reconstruct a sequence from one that supposedly was degraded by deleterious mutations, deletions, recombination, and insertions of other repetitive elements.
- Even if a precise ERV sequence was known, cloning techniques using vectors do not precisely replicate anything without problems:

- “Scientists have tried to take advantage of the virus’s biology and manipulate its genome to remove human disease-causing genes and insert therapeutic genes. However, viruses, while effective, introduce other problems to the body, such as toxicity, immune and inflammatory responses, and gene control and targeting issues.”
- http://www.genome.gov/10004764
- Regulation, mainly activation, of neighboring (downstream) genes during embryonic development of the mouse. -- Peaston A.E., Evsikon, A.V., Graber, J.H., de Vries, W.N., Holbrook, A.E., Solter, D., and Knowles, B.B., Retrotransposons regulate host genes in mouse oocytes and preimplantation embryos, Developmental cell 7:597-606, 2004.

  Regulation of human genes expressed in the placenta (e.g. pleiotropin) and somatic tissues (e.g. apolipoprotein C1 in the liver and ß-amylase in the salivary gland). -- Coffin, J.M., Retroviridae: the viruses and their replication; in: Field, B.N., Knipe, D.M., Howley, P.M., Chanock, R.M., Melnick, J.L., Monath, T.P., Roizman, B., and Straus, S. E. (eds.), Fields Virology, 3rd ed, Lippincott-Raven Publishers, Philadelphia, PA, pp. 1767-1847, 1996, also Bannert, N. and Kurth, R., Retroelements and the human genome: New perspectives on an old relation, Proceedings of the National Academy of Science, USA 101: 14572-14579, 2004.

  Immunomodulation, including induction of immunotolerance to self antigens and immunization against exogenous retroviruses -- Coffin, J.M., Retroviridae: the viruses and their replication; in: Field, B.N., Knipe, D.M., Howley, P.M., Chanock, R.M., Melnick, J.L., Monath, T.P., Roizman, B., and Straus, S. E. (eds.), Fields Virology, 3rd ed, Lippincott-Raven Publishers, Philadelphia, PA, pp. 1767-1847, 1996. also Bannert, N. and Kurth, R., Retroelements and the human genome: New perspectives on an old relation, Proceedings of the National Academy of Science, USA 101: 14572-14579, 2004.

  The env protein of HERV-W and HERV-FRD serving as fusogenic factors (syncytins) during human trophoblast development. -- Mallet, F., Oliver, B., Prudhomme, S., Cheynet, V., Oriol, G., Bonnaud, B., Lucotte, G., Duret, L., and Mandrand, B., The endogenous retroviral locus ERVWE1 is a bona fide gene involved in hominoid placental physiology, Proceedings of the National Academy of Sciences, USA 101: 1731-1736, 2004. also Frendo, J.L., Olivier, D., Cheynet, V., Blond, J.L., Bouton, O., Vidaud, M., Rabreau, M., Evain-Brion, D., and Mallet, F., Direct involvement of HERV-W Env glycoprotein in human trophoblast cell fusion and differentiation, Molecular and Cellular Biology 23:3566-3574, 2003.

  Other roles such as mammalian tissue organization. -- Matsui, T., Kinoshita-Ida, Y., Hayashi-Kisumi, F., Hata, M., Matsubara, K., Chiba, M., Katahira-Tayama, S., Morita, K., Miyachi, Y., and Tsukita, S., Mouse homologue of skin-specific retroviral-like aspartic protease (SASPase) involved in wrinkle formation, The Journal of Biological Chemistry Jul 12; [Epub ahead of print], 2006.

  ERVs act as promoters, starting transcription at alternative starting points, which enables different RNA transcripts to be formed from the same DNA sequence.

  “For instance gamma-retrovirus was isolated from trophoblastic cells of the baboon placenta. This virus was found to be very closely related antigenically and by sequence homology to the endogenous RD114 virus in cats (which is itself unrelated to endogenous FeLV). Benveniste and Todaro observed, like we did for jungle fowl, that only certain species of the cat genus, Felis, possessed this endogenous genome related to the baboon ERV. In contrast, all species of baboons carry this virus so it would appear to have been present in the germ line of primates much longer than in cats. Thus it seems evident that a horizontal, infectious event occurred to transfer the virus from baboons to cats, whereupon it became endogenous in the new species.” -- Robin A Weiss, “The discovery of endogenous retroviruses,” Retrovirology, 2006; 3: 67. Published online 2006 October 3. doi:

  ‘We report the existence of 51,197 ERV-derived promoter sequences that initiate transcription within the human genome, including 1,743 cases where transcription is initiated from ERV sequences that are located in gene proximal promoter or 5’ untranslated regions (UTRs).’ -- Conley et al., ref 1, p. 1563.

‘Our analysis revealed that retroviral sequences in the human genome encode tens-of-thousands of active promoters; transcribed ERV sequences correspond to 1.16% of the human genome sequence and PET tags that capture transcripts initiated from ERVs cover 22.4% of the genome.’ -- Conley et al., ref 1, p. 1566.

"Several lines of evidence indicate that chimpanzee and gorilla PTERV1 copies arose from an exogenous source. First, there is virtually no overlap (less than 4%) between the location of insertions among chimpanzee, gorilla, macaque, and baboon, making it unlikely that endogenous copies existed in a common ancestor and then became subsequently deleted in the human lineage and orangutan lineage. Second, the PTERV1 phylogenetic tree is inconsistent with the generally accepted species tree for primates, suggesting a horizontal transmission as opposed to a vertical transmission from a common ape ancestor. An alternative explanation may be that the primate phylogeny is grossly incorrect, as has been proposed by a minority of anthropologists." - Lineage-Specific Expansions of Retroviral Insertions within the Genomes of African Great Apes but Not Humans and Orangutans http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1054887/

“Apoptosis, or programmed cell death, is a normal component of the development and health of multicellular organisms. Cells die in response to a variety of stimuli and during apoptosis they do so in a controlled, regulated fashion. The latter occurs when T-cells recognize damaged or virus infected cells and initiate apoptosis in order to prevent damaged cells from becoming neoplastic (cancerous) or virus-infected cells from spreading the infection.” -- Reproductive and Cardiovascular Disease Research Group.
http://www.sgul.ac.uk/depts/immunology/~dash/apoptosis/

Because apoptosis would kill most ERV-containing cells, why is it that we still have so many ERVs hundreds of thousands of years after supposedly being inserted by retroviruses?

Because these "HERVs" have critical functions in humans demonstrates they are information specific to human functions and anatomy, and are not the genome of viruses.

Members of the HERV-K family are typically found in areas near genes [which code for proteins]. The regulatory role of HERVs has been demonstrated in the liver, placenta, colon, and other locations. Source: Norbert Bannert and Reinhard Kurth, “Retroelements and the human genome: New perspectives on an old relation” [PDF], Proceedings of the National Academy of Sciences, 101:14572–14579, 2004.

One of the 1st stages of ERVs to be expressed from the zygote to the 2 cell stage of development. It was proven in 2004 that if these are disabled the development ceases. Thus, these supposed ERV's cannot be viral DNA incorporated into the genome inserted later because without them life cannot exist. One cannot have offspring without ERV’s

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Conclusion

The word ‘virus’ means toxic or poison, and that is how most evolutionists perceive them. However, if 50% of our DNA is made up of viral elements, wouldn’t that indicate that they might be essential genetic elements and that these viral elements might rather be another regulatory network in organisms? This is just another case why evolutionary-based science is a menace to scientific research, discovery, and progress. It won't break out of its stringent rhetoric of bias thinking of evolution.
In summary, a strong case can be made pointing to the view that ERVs were not inserted by retroviruses. They have function, should have been ridden by apoptosis, are different from their ancestral genomes, and it is incredible that the organisms did not die after being infected with so many viral genes. With so many problems, a lot of explaining must be done before using ERVs as evidence for evolution.

We all have presuppositions that lead to biased thinking. In the area of ERV genetic research, almost all grants start with the presupposition that ERVs began as dangerous viruses, so the natural focus would be on the connection to disease. Because creationists believe that all initial genetic elements were created good, their natural focus would be on the beneficial effects of ERVs.

It's now obvious that the money would have been better spent using a creation presupposition. The 20-30 years of evolutionary biased research on ERVs (commonly referred to as 'junk DNA' by evolutionists) has proven to be one of evolution's biggest blunders. Evolutionists have already begun to spin it and we can't wait to hear their defense!

https://world.wng.org/2014/08/junk_dna_and_darwinian_blind_spots