

Do Pseudogenes and Endogenous Retroviruses Prove Evolution¹?

Some Literature References

- 1) <https://evolutionnews.org/2024/05/heres-a-far-from-exhaustive-yet-still-exhausting-list-of-papers-discovering-function-for-junk-dna/>
- 2) <https://evolutionnews.org/2021/07/junk-no-longer-paper-reports-endogenous-retroviruses-are-integral-and-important-components-of-immune-responses/>
- 3) <https://evolutionnews.org/2019/09/waste-not-research-finds-that-far-from-junk-dna-ervs-perform-critical-cellular-functions/>
- 4) https://evolutionnews.org/2013/04/an_icon_of_the_/
- 5) <https://www.sciencedirect.com/science/article/pii/S153458072031025X>
“Genome-wide analysis of pseudogenes reveals *HBBP1*’s human-specific essentiality in erythropoiesis and implication in β -thalassemia”
- 6) Weitere Details: <https://www.ncbi.nlm.nih.gov/gene/3044>
- 7) <https://www.genecards.org/cgi-bin/carddisp.pl?gene=HBBP1>
- 8) <https://answersresearchjournal.org/human-beta-globin-pseudogene/>
- 9) Excerpt from p. 9 of <https://www.weloennig.de/PANDA.Part1.pdf> (***Apply please the following Evolutionary Contradiction to Pseudogenes and Retroviruses***):

A Massive Contradiction Within the Theory of Evolution Itself

On the other hand, I would like to emphasize that – in utter contrast to all the assertions on the panda’s imperfection cited above – now *according to the evolutionist’s own presuppositions on the limitless powers of natural selection*, the panda’s thumb should already be the best solution possible, i.e. it cannot be designed more elegantly, more efficiently and perfectly, so that any redesign would be entirely superfluous – representing a massive contradiction/conflict/inconsistency/incongruity within the theory of evolution itself, for example (just a few keywords):

“...natural selection is daily and hourly scrutinizing, throughout the world, every variation, even the slightest; *rejecting that which is bad, preserving and adding up all that is good*; silently and insensibly working, whenever and wherever opportunity offers, at the improvement of each organic being in relation to its organic and inorganic conditions of life” ... “**I can see no limit to this power**” ... “natural selection ... always *intently watching* each slight alteration in the transparent layers [of the eye]; and carefully preserving each which ... in any way or in any degree, tends to produce a distincter image” – Darwin.

Prof. John Avise: “Natural selection comes *close to omnipotence*”. Prof. Christopher Exley is, indeed, convinced that “both the beauty and the brilliance of natural selection are reflected in *its omnipotence* to explain the myriad

¹ Macroevolution

observations of life” (virtually/vitally in agreement with Dawkins, Coyne, Futuyma, Todd, Ayala, Mayr and many other renowned evolutionary authors)

“The genetic message, the program of the present-day organism...resembles a text without an author, that a proof-reader has been correcting for more than two billion years, *continually improving, refining and completing it, gradually eliminating all imperfections.*” (Nobel laureate Francois Jacob)²

And as result of this limitless, omniscient and omnipotent natural selection “gradually eliminating all imperfections” now this “crude”, “clumsy”, “highly inefficient”, “imperfect”, “suboptimal” and “bad design” of the panda’s thumb?

So, you can choose: Imperfect or perfect, bad design or excellent design? There are evolutionists on both sides. Whatever the case – Evolution is always right.

10) Another now completely different aspect (assuming/presupposing the whole evo-story):

Overview with AI (18 August 2025)

"Retroviral integration³ into a host genome is **not random; different retroviral types exhibit preferences for specific genomic features and nucleotide sequences.** These preferences are influenced by the viral integrase protein and host cell cofactors that guide the integration machinery to particular chromatin environments."

Or similarly (19 August 2025):

“Retroviral integration into a host cell's genome is indeed not random. While retroviruses can insert their DNA into various locations, **they exhibit specific preferences for certain genomic regions.** These preferences vary between different retroviral types, but generally involve a bias towards active genes, regulatory regions, and areas with specific chromatin structures.”

See there also the more detailed explanation like **“Non-random integration:**

“Retroviruses don't insert their DNA randomly throughout the genome; they show biases towards certain regions.”

*Retroviral integration could therefore also have occurred later/additionally/subsequently and **independently of each other** in different organisms such as humans and chimpanzees **at specific locations in the genome.***

I had already discussed something similar (“HOT SPOTS OF TE INSERTION”) for transposons (TEs / transposable elements) together with Heinz Saedler in 2002 (Lönnig, W.-E. and H. Saedler: Chromosome rearrangements and transposable elements. *Annual Reviews of Genetics* **36**, 389-410.

And an instructive example for independent integrations from an evolutionary point of view⁴:

“It is perhaps best to elucidate these critical questions on the basis of a renowned retrovirus example: two genes involved in human placenta morphogenesis, *syncytin-1* and *-2*, are supposed to have been obtained from the fusogenic envelope protein (*env*) genes of the HERV-W family of endogenous retroviruses belonging to the copia superfamily of LTR retrotransposons. The process of cooption of one or two of these genes for an essential role in placentation (fusion of cytotrophoblast cells to generate its syncytial layer) *is thought to have happened **at least eight times independently of each other** (in primates, carnivores, rodents, lagomorphs and ruminants) and within the order Rodentia again twice*

² Cf. the references and larger documentation including many more details in <https://www.weloennig.de/OmnipotentImpotentNaturalSelection.pdf>

³ “Endogenous retroviruses (ERVs) are remnants of past retroviral infections that have integrated into the germline (sperm and egg cells) of a host species and are passed down through generations. These viral sequences become a permanent part of the host's genome” (AI 19 August 2025)

⁴ See Lönnig, W.-E.: Transposons in Eukaryotes (Part B): Genomic Consequences of Transposition. Encyclopedia of Life Sciences (2015), from pp. 5 and 7 – colored emphasis added.

convergently (Redelsperger et al., 2014). The latter authors state that their results in the woodchuck *Marmota monax* (whose *syncytin-Mar1* is unrelated to all other *syncytin* genes) ‘extend the range of retroviral envelope gene ‘domestication’ in mammals and show **that these events occurred independently, on multiple occasions during evolution to improve placental development in a process of convergent evolution**’. Extrapolating from the research data published so far, most of the ~30 orders of mammals would have convergently domesticated retroviral envelope genes to improve their placental development. *Also, this could have happened independently several times within different orders.*”

Considering the Paleontological Dimension

“However, checking the paleontological data for the decidedly different time spans calculated in the molecular papers for various cases, **many of these orders had already existed millions of years before their putative syncytin domestications happened – including several groups of living fossils displaying no or hardly any further morphological evolution**. So, a proponent of the selfish DNA hypothesis may possibly ask, how could they have survived and flourished for millions of years without the coopted/domesticated viral genes?”

Further Evolutionary Problems to be Considered

“And a deeper problem: According to neo-Darwinism (synthetic theory), many species of these non-*env*-gene orders were coexisting in extreme competition with species of the remaining mammalian orders, which had already incorporated the *env* genes hundreds of thousands of generations earlier (e.g. in Carnivora *syncytin-Car1* between 60 and 85 Mya, but in Primates *syncytin-1* gene 25 Mya and *syncytin-2* >40 Mya). In other words, **why could so many life forms survive side by side with and without the *env* genes, that is, simultaneously with improved and non-improved placental developments?** And what exactly does ‘improve’ mean in these cases?”

“Moreover, where did endo- and exogenous retroviruses themselves get the range of different *env* genes from? Correctly, Schubert comments that ‘the researchers *speculate* that syncytins arose early during mammalian evolution, but as mammals diverged, the original syncytins were superseded by new syncytin genes from retroviruses in the environment’ (Schubert, 2015; emphasis added). Besides, there is another category of placentas, the **epitheliochorial placentas**, distinguishing **horses, pigs and dolphins that do not display fused cells**. As the latter groups are at least as successful as all the others – why suppose that improved *env* genes are absolutely necessary for mammalian development? It will be interesting to follow-up the future results on research of the *syncytin* genes in these animal forms.

Administering the logic for the evolution of the *syncytin* genes to the putative cases of domesticated TEs, supercedence of earlier gene functions would be the rule. Although many researchers tend to assume without a doubt that such substitutions would have been advantageous functionally and hence selectionally, in an alternative scenario of the neutral theory the replacements could simply be stochastic⁵ events with no or even slightly deleterious effects on the phenotype and yet spread in a population (see below). Even if functionally necessary after takeover of the tasks of the original genes, in such cases, the substitutes would be largely irrelevant for macroevolution.”

See additional points in the original publication.

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⁵ As general occurrences but not necessarily the place.