

From Information to Inheritance: Tree, Web and Viruses

QTC-027 · From Quarks to Consciousness

The Universe, Life, and How We Found Out

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Article Abstract

Inheritance is not the passive storage of a code. It is a physical history of molecules copied with high but finite fidelity, repaired, rearranged and sometimes moved between lineages. Vertical descent produces genuine trees, yet different genes can follow different routes through duplication, loss, recombination, horizontal transfer and endosymbiosis. Viruses complicate the picture further: they depend on cells, exchange genes with them, leave inherited fossils in genomes and provoke defences that acquire molecular memories. None of this makes ancestry an undifferentiated web. Conserved cellular histories still supply a branching scaffold, while transfers add traceable connections. This article follows information from DNA replication through mutation, phylogenetic reconstruction, mobile elements and viral conflict, showing why life preserves its past through both continuity and controlled opportunities for change.

In 60 Seconds

Genetic information has a physical carrier. In cellular life, DNA sequences are copied semiconservatively, proofread and repaired, yet some differences survive as mutations. If copying were perfect, novelty would stop; if it were too inaccurate, inherited adaptations would dissolve. Parent-to-descendant transmission makes branching genealogies, and conserved genes reveal deep cellular ancestry. But genes also move laterally through transformation, conjugation and phage-mediated transduction. Viruses add DNA or RNA histories that do not fit one universal viral tree. Some entered germ lines and now occupy recognizable parts of animal genomes; captured viral envelope genes help build mammalian placentas. Cells answer with restriction systems and CRISPR memories, while phages evolve counter-defences. Transposons and plasmids add further replicating interests inside genomes. The result is neither one perfect tree nor shapeless web: cellular descent supplies a scaffold, and gene-specific transfers form dated grafts. Inheritance works because continuity and change are coupled, not because either one acts alone.

Why This Question Matters

Every genome is both a mechanism and an archive. It must copy enough structure to keep an organism viable, yet it also bears mutations, borrowed genes, ancient viruses and mobile elements whose histories do not coincide. Reading that archive changes how we understand ancestry, antibiotic resistance, immunity and even placental development. It also prevents a false choice. Horizontal

transfer does not prove that evolutionary trees are meaningless, and a cellular tree does not make every gene vertical. The scientific task is to identify which process produced each pattern and how confidently its direction can be reconstructed.

1. Information Needs a Physical Carrier

The Question

If genetic information is more than a metaphor, where is it physically located, what exactly is copied, and how can a molecular sequence preserve a difference across generations and cellular contexts without determining an organism by itself?

Information in genetics is not an invisible fluid poured into chemistry. It names measurable relations: one molecular sequence can be copied, compared with another and causally alter a product or regulatory process.[1,2,4] Context still matters. The same DNA can behave differently across cells, environments and developmental stages, so sequence is a carrier of inherited differences, not a complete instruction manual detached from a living system.

The carrier became an experimental question before it became textbook certainty. Avery, MacLeod and McCarty purified a fraction from pneumococci that transmitted an encapsulated, heritable type and found its activity consistent with DNA.[1] Hershey and Chase later labelled phage protein and DNA separately; most DNA entered bacteria while most protein remained outside.[2] These were converging experiments, not one magical moment when every exception disappeared.

Once DNA was identified as hereditary material in cells, copying itself had to be tested. Meselson and Stahl grew bacteria with heavy nitrogen, shifted them to light nitrogen and followed DNA density across generations.[3] The bands matched semiconservative replication: each daughter duplex retained one old strand beside one new strand. Inheritance was revealed as a material continuity, not the manufacture of wholly new copies without parental structure.

Crick's central dogma is often shortened until it becomes wrong. His 1970 formulation concerned transfer of sequential information: DNA and RNA can template nucleic acids, nucleic acids can specify protein, but protein sequence does not template nucleic-acid sequence.[4] Reverse transcription is therefore not a violation. Nor does the dogma deny feedback, regulation or environmental causation. It limits one kind of copying relation.

This article begins where several earlier ones stop. QTC-010 explained mutation and selection; QTC-023 reconstructed common cellular ancestry; QTC-026 followed the merger that built the eukaryotic cell.[16,17] The present question is how inherited information crosses generations and sometimes crosses lineages. That shift turns a stored sequence into a history whose fidelity must be measured before its branching can be trusted.

Where That Leaves Us

Genetic information is physical enough to label, separate and follow through replication, yet contextual enough that sequence never acts alone. Semiconservative copying supplies continuity, but continuity is useful only if errors are mostly corrected and some still survive. The next problem is therefore not whether copying is accurate, but how life builds accuracy without eliminating change.

2. Fidelity Is Built, Not Perfect

DNA replication begins with chemical discrimination, not certainty. Polymerases select among nucleotides, some remove a mismatched end, and mismatch-repair systems inspect newly copied DNA afterward.[6,8] Each layer rejects errors missed by the previous one. Their combined performance makes genomes stable across generations, while differences among organisms and conditions show that fidelity is a biological construction with parts, costs and failure modes.

The errors that remain are not uniformly scattered. Whole-genome accumulation and maximum-depth sequencing reveal different substitution probabilities among sites, sequence contexts and physiological conditions.[6,7] Some misincorporations occur far more often than the inherited mutation rate because repair removes them. A mutation spectrum is therefore the filtered output of damage, replication and repair. Random with respect to benefit does not mean chemically featureless.

Luria and Delbrück made that distinction visible before genes could be sequenced. Independent bacterial cultures exposed to phage produced wildly different numbers of resistant colonies, including jackpots from mutations that occurred early.[5] The distribution fit resistance arising before exposure better than phage instructing each cell to mutate usefully. Modern stress responses complicate rates and spectra, but they do not turn future adaptive value into a molecular template.

Too little fidelity can also destroy inheritance. Eigen's quasispecies models link error rate, selective advantage and sequence length: beyond a threshold, a privileged sequence loses localization in mutant space.[9] That is a precise result within a model, not a measured universal cliff for all life. Real populations recombine, repair, compartmentalize and inhabit rugged fitness landscapes. The theory identifies a constraint while leaving its empirical boundary system-specific.

Nor is maximal fidelity automatically favoured. Proofreading and repair require molecular machinery, while selection has difficulty improving tiny error differences in small populations. The drift-barrier framework connects achievable mutation rates to effective population size and molecular cost.[7,10] It remains a framework rather than one number for life. The balance among error, repair and selection must be measured lineage by lineage.

Where That Leaves Us

Copying fidelity is neither accidental nor absolute: molecular layers suppress most errors, population processes limit further improvement, and surviving mutations keep histories from becoming identical loops. Once differences can persist, descent acquires a geometry. The next step is to ask why parent-to-descendant transmission creates real evolutionary trees even when no single gene records every branch perfectly.

3. Vertical Descent Builds Trees

The Question

If genomes recombine and genes can later move between organisms, is an evolutionary tree merely a convenient picture, or does parent-to-descendant inheritance preserve a branching history that data can recover despite later exchanges among descendants?

A genealogy branches whenever descendants separate from a parent lineage and then accumulate different histories. That relation is tree-like even if later contacts add connections.[11,12] The tree is not a claim that organisms never exchange genes. It is a claim about vertical descent: cells come from cells, and reproductive lineages divide. The evidence for a branch depends on characters that have survived enough copying to retain shared ancestry.

Ribosomal RNA supplied unusually deep characters. Woese and Fox used it to distinguish a major archaeal lineage from familiar bacteria; Woese, Kandler and Wheelis later formalized Bacteria, Archaea and Eucarya as domains.[11,12] Later work often nests eukaryotes within Archaea rather than retaining three primary branches. That revision demonstrates what a scientific tree is for: topology can change while measured homology and archaeal distinctness remain.

No rule says every gene must display the same topology. Duplication creates paralogues, loss deletes branches, transfer changes owners, recombination joins segments and incomplete lineage sorting lets ancestral variants cross successive speciation events.[14,15,17] In some population ranges, the most probable gene tree can even differ from the species tree. Discordance is therefore evidence to explain, not automatic proof that phylogeny has failed.

Universal common ancestry rests on a network of conserved molecular correspondences and explicit statistical comparisons, not on visual resemblance to one diagram.[13,16] Theobald's formal test strongly favoured common ancestry over independently originated alternatives within its model set.[16] That result does not reconstruct LUCA's body, metabolism or environment. It addresses shared descent, while QTC-023 owns the harder phenotype and root questions.

Modern phylogenomics therefore combines many markers and models processes that separate their histories.[13–15,18] A species framework is an inference with support values, taxon sampling and assumptions, not a sacred tree. The same discipline that protects vertical history also prevents forcing a transferred or duplicated gene onto the wrong branch. Tree thinking becomes stronger when discordance is modelled rather than hidden.

Where That Leaves Us

Vertical inheritance gives cellular ancestry a branching scaffold, while gene-tree disagreement reveals duplication, loss, sorting and exchange around it. The tree survives because it represents a specified process, not every process at once. The next section follows the most dramatic exception to strict verticality: genes crossing between contemporaries through mechanisms that experiments can separate and genomes can detect.

4. Genes Also Move Sideways

Horizontal transfer is not one mechanism. Natural transformation takes up genetic material from the environment; conjugation uses cell-associated transfer machinery; transduction packages host DNA in phage particles.[1,19,20] The classic experiments distinguished outcomes before every component was known. Their modern labels summarize later mechanistic work, so historical fairness requires neither denying what they showed nor pretending their authors already possessed today's molecular diagrams.

Avery's transforming principle changed a heritable bacterial character without reproduction.[1] Lederberg and Tatum recovered recombinant *E. coli* from mixed cultures, establishing genetic

exchange between cells.[19] Zinder and Lederberg then found a filterable agent mediating exchange in Salmonella and traced it to bacteriophage, revealing transduction.[20] Three routes made one lesson unavoidable: a lineage can acquire inherited material from somewhere other than its parent.

Genome comparisons expanded isolated mechanisms into an evolutionary force. A 2024 analysis reconciled gene trees with a species framework across 8,790 prokaryotic species and detected millions of putative transfer events.[21] The precise fraction is method-dependent and older events are harder to recognize. The robust conclusion is heterogeneity: transfer varies with ecological contact, phylogenetic distance, gene function and the opportunity for foreign DNA to persist.

Recent transfers in that survey were enriched in accessory functions, mobilome components and antimicrobial resistance, whereas some older transfers had become widespread metabolic genes. [21,23] This does not mean resistance began with medicine or that every metabolic pathway was imported. It shows a sorting process: incoming genes arrive, many disappear, and a subset persists because ecology, compatibility and selection make them useful or at least tolerable.

Sideways movement can even clarify vertical chronology. Reconciliation places a transferred gene on donor and recipient branches, implying that the donor lineage existed before the recipient acquired it.[18,22] Many such constraints can order nodes and test molecular clocks. A transfer therefore has two epistemic faces: it can make one gene misleading as a species marker, yet become evidence once its nonvertical route is correctly identified.

Where That Leaves Us

Transformation, conjugation and transduction turn horizontal inheritance from metaphor into mechanism, while phylogenomics shows that its frequency and persistence are structured rather than universal. Phages already entered that account as carriers, but viruses do more than shuttle bacterial genes. They possess their own diverse histories, enter germ lines and sometimes become indispensable parts of host biology.

5. Viruses Are Vectors and Archives

The Question

Viruses cannot reproduce without cells, yet they cross lineages and leave genes that descendants inherit for millions of years. Are they external parasites, branches of life, mobile genomes, or different things in different histories today?

Viral diversity defeats one tidy definition. Viruses use DNA or RNA, single or double strands, segmented or continuous genomes, and depend on host translation for reproduction.[24,25] That dependence makes them boundary cases in QTC-009's question of life, but not biologically marginal. Environmental sequencing uncovered immense numbers of previously unknown viral sequences, exposing a genetic world that cultivation had sampled only thinly.[24]

No gene is known to unite every virus in the way conserved translation genes connect cellular life. [25,26] Some viral groups share capsid or replication hallmarks; others do not. Escape from cells, reduction from cellular parasites and origins among pre-cellular replicators each explain portions of the evidence.[25,26] The open conclusion is plural: virus origins may be multiple, and one scenario should not be promoted into a recorded universal history.

Viruses also become fossils. When a retrovirus enters a germline cell and its DNA persists, descendants can inherit the insertion. The human reference genome contains recognizable endogenous-retroviral material amounting to roughly eight percent, nested within a much larger transposable-element fraction.[27] Exact percentages depend on annotation and reference sequence. The conclusion is historical: some apparent human DNA arrived through infections of ancestors.

Occasionally, a fossil becomes machinery. Human syncytin is a captured retroviral envelope protein with fusogenic activity in placental tissue.[28] In mice, knocking out Syncytin-A disrupted placental architecture and caused embryonic death, providing causal evidence rather than sequence resemblance alone.[29] Different mammal groups captured different envelope genes. Viruses did not invent one universal placenta; evolution repeatedly recruited viral fusion tools into lineage-specific development.

Phages can transform hosts without waiting for geological time. CTXphi carries genes encoding cholera toxin and lysogenic conversion turns susceptible *Vibrio cholerae* into toxin producers.[33] The disease is not 'a virus' in isolation: bacterial regulation, phage cargo, ecology and the human host all matter. Yet the example makes inheritance concrete—a viral genome can add a trait that then travels with a bacterial lineage.

Where That Leaves Us

Viruses are parasites, vehicles and inherited archives, sometimes all within one long history. Their lack of a universal gene leaves deep origins open, while endogenous sequences and lysogenic conversion make their genomic effects observable. Hosts are not passive recipients, however. The next step is the reciprocal history: molecular defences that remember invasion and viral countermeasures that defeat them.

6. Cells Defend, Viruses Counter

Bacterial defence begins with molecular discrimination. Restriction systems cut susceptible DNA, while host-controlled modification protects matching resident DNA.[31] The distinction is biochemical rather than philosophical: recognition sites, enzymes and modification states determine survival. These systems can block phages and incoming genes, yet their own mobility and maintenance costs complicate the simple image of a genome defending a fixed, permanent self.

CRISPR-Cas adds a different kind of inheritance. Barrangou and colleagues challenged *Streptococcus thermophilus* with phages and observed new spacers derived from viral sequences; adding or removing specific spacers altered resistance.[30] A cell lineage can therefore inherit a molecular record of exposure and use sequence complementarity against a related invader. The memory is acquired and specific, but it is built from encountered DNA, not foresight about future needs.

No defence ends the conflict. Bondy-Denomy and colleagues identified phage genes that inactivate particular CRISPR-Cas systems, now called anti-CRISPRs.[32] A host innovation changes the environment of the virus; a viral countermeasure changes it back. Receptors, restriction sites, spacers and inhibitors can all evolve. What looks like a stable immune mechanism at one moment is one move in a longer reciprocal history.

'Arms race' is useful only if it is not made universal. Some host-virus systems show escalating defence and escape; others cycle through genotypes, tolerate infection or settle into coexistence. [30,32,33] The appropriate model depends on ecology, population structure and costs. Evolution has no obligation to maximize resistance. A defence that blocks every mobile element may also block genes that would benefit the host under a new condition.

That trade-off joins immunity to inheritance. CRISPR, restriction and other defence systems change which DNA can cross a cell boundary, while phage counter-defences reopen routes.[21,30,32] The genome is therefore neither an open library nor a sealed vault. It is a selectively permeable history whose filters also evolve. To understand the resulting conflicts, the unit being copied must be allowed to vary from chromosome to plasmid to transposon.

Where That Leaves Us

Defence systems make inheritance selective, and viral counter-defences make that selectivity temporary. The same genome can reject one sequence, archive another and later depend on a captured viral gene. That instability shifts attention from organisms alone to the smaller replicators they contain. The next section follows transposons and plasmids, whose persistence can align with host success or oppose it.

7. Inheritance Has More Than One Channel

The Question

A chromosome, a plasmid and a transposon can all be copied, but their chances of persistence need not coincide across generations or environments. Whose inheritance are we describing today when several replicators share one cell?

Barbara McClintock inferred mobile loci from changing pigmentation and chromosome behaviour in maize before transposons had a molecular name.[34] Her experiments showed that inherited DNA could alter position and regulation within a genome. Modern transposable elements use several mechanisms, but the core lesson remains: movement need not cross an organismal boundary to reshape inheritance. A genome has internal traffic as well as parent-descendant transmission.

The human reference genome made the scale visible. Recognizable transposable-element-derived sequence occupies roughly forty-five percent, including endogenous retroviral remnants near eight percent.[27] These are annotation-dependent lower-level categories, not a census of active jumping genes. Most copies are damaged or immobilized. Sequence abundance records past replication and insertion; it does not by itself prove present function, harm or adaptation.

Plasmids make replicator interests easier to measure. They copy within cells, segregate into descendants and sometimes transfer between cells.[19,35] Carrying one can slow growth when its genes are unnecessary, yet selection for a plasmid trait or compensatory host evolution can stabilize the association. Experiments show that a costly arrival need not remain costly. Persistence emerges from both within-cell and between-cell dynamics.

Calling a mobile element selfish is shorthand, not psychology. An element can increase its own transmission while harming host reproduction, become nearly neutral, or carry a function that benefits both under particular conditions.[27,33,35] The sign changes with environment and scale.

Cholera-toxin phage, a resistance plasmid and a silent retroviral remnant are all inherited sequences, but their relations to hosts are not interchangeable.

Present genomes therefore combine channels. Vertical copying supplies most chromosomal continuity; recombination rearranges inherited variants; plasmids and phages cross cells; transposons move within genomes; endosymbiosis joins whole lineages.[18,27,36] These processes do not dissolve into one metaphorical web. Each has different particles, rates and directional evidence. Reconstructing ancestry means assigning a route to a sequence rather than demanding one route for everything.

Where That Leaves Us

Multiple replicators can share a cell without sharing one fate, and multiple inheritance routes can shape a genome without becoming equivalent. That leaves a final representational problem. If branches, transfers and viral insertions are all real, the history of life needs diagrams that preserve their differences—trees where descent dominates, networks where lineages genuinely join, and uncertainty where origin remains open.

8. A Tree with Grafts, Not a Fallen Tree

The choice between tree and web is false when the processes are specified. Cellular reproduction creates a branching scaffold; particular genes depart from it through transfer, recombination, duplication and loss.[13,18,36] A web drawn without direction or mechanism throws away evidence. A tree that forces every sequence onto one topology does the same. The useful representation follows the inheritance route of the object being studied.

Viruses remain the hardest addition because their roots are not secured by one universal gene.[25,26,38] Escape scenarios derive viral genomes from cellular elements; reduction scenarios derive some from more complex parasites; virus-first-like models place replicators before modern cells. Evidence may support different stories for different realms. The honest status is contested plurality, not a single family tree and not permission to invent any origin equally.

Open-ended evolution adds another unresolved layer. Eigen showed why retention can fail when copying becomes too inaccurate, while population genetics explains why perfect fidelity is neither attainable nor always favoured.[9,10] Major-transition theory tracks changes in how information is packaged and transmitted.[37] Yet no formula using fidelity, variation and exchange alone predicts indefinite innovation across cells, ecosystems and artificial systems. Necessary ingredients are not sufficient conditions.

Reticulation can be a source rather than only noise. If a gene passed from one lineage to another, donor and recipient constraints can order events that vertical genes date poorly.[18,20,22] The same anomaly that disrupts a simple gene tree may preserve contact between ancient populations. Scientific reconstruction improves when it asks what process generated discordance before deciding whether to discard or exploit it.

Inheritance is therefore constrained continuity. Semiconservative copying carries old strands forward; repair protects accumulated function; mutation produces differences; selection and drift change their fate; transfer and viruses connect lineages; mobile elements pursue partly independent persistence.[4,16,21,24,36,37] The season closes without a sealed code or shapeless

web. Life keeps a history because copying is reliable, and keeps evolving because inheritance has never meant perfect isolation.

Where That Leaves Us

A tree records vertical descent; grafts record transfers; viral fossils record infections that became inheritance. None cancels the others. Together they show why biological information is historical rather than merely stored: every surviving sequence carries both a copying pathway and a record of change. That coupling closes Season 1—from physical law to life capable of preserving and revising its past.

What We Know, and How

- DNA is copied semiconservatively, and layered proofreading and repair greatly reduce inherited replication errors.[3,6,8]
- Vertical inheritance produces tree-like cellular genealogies, while gene trees can differ through duplication, loss, transfer and lineage sorting.[11–15]
- Transformation, conjugation and transduction are distinct experimentally established routes of horizontal gene transfer.[1,19,20]
- Viruses and mobile elements can enter genomes, transmit traits and be co-opted for host functions.[27–29,33–35]

What We Still Don't Know

- Whether all major viral lineages share any deep common origin.
- Which viral-origin scenarios apply to each realm and in what sequence.
- The quantitative lower limits and costs that set mutation rates across lineages.
- When arms-race, fluctuating-selection or coexistence dynamics dominate host-virus evolution.
- A sufficient, predictive theory of open-ended evolutionary innovation.

What Could Change Our Minds

- A universal homologous core connecting every viral realm.
- Bias-resistant phylogenomic methods that consistently erase or strengthen the vertical cellular scaffold.
- Long-term experiments that isolate universal mutation-rate floors across population sizes and molecular systems.
- Direct reconstructions showing that presumed transfers are instead duplication, loss, contamination or tree error.
- A cross-system theory that predicts when fidelity, variation and exchange produce open-ended evolution.

If You Remember Only One Thing

Inheritance preserves physical differences without sealing lineages. Vertical descent builds trees; mutation, mobile elements and viruses add traceable change, grafts and conflicts to those branching histories.

Related Articles

- QTC-010 — Random Variation and a Filter
- QTC-023 — LUCA: The Ancestor We Reconstructed
- QTC-026 — The Cell Within the Cell

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