Al-Rafidain J Med Sci. 2026;10(1):90-92.
DOI: https://doi.org/10.54133/ajms.v10i1.2726

AJMS

Online ISSN (3219-2789)

Editorial Letter

Can Darwin's Natural Selection Alone Explain the Origin of Species? An Information-Theory and Computer-Science Perspective

Ahmed Al-Imam[1,2]*, Riccardo Lora[3], Sinan Subhi Farhan[1]

[1]*Department of Anatomy, College of Medicine, University of Baghdad, Baghdad, Iraq;* [2]*Barts and the London School of Medicine and Dentistry, Queen Mary University of London, London, United Kingdom;* [3]*Section of Pharmacology, Department of Diagnostics and Public Health, University of Verona, 37134 Verona, Italy*

Received: 2 January 2026; Accepted: 16 January 2026

* ***Corresponding author***: Ahmed Al-Imam, Department of Anatomy, College of Medicine, University of Baghdad, Baghdad, Iraq; Email: ahmed.mohammed@comed.uobaghdad.edu.iq

Article citation: Al-Imam A, Lora R, Farhan SS. Can Darwin's Natural Selection Alone Explain the Origin of Species? An Information-Theory and Computer-Science Perspective. *Al-Rafidain J Med Sci.* 2026;10(1):90-92. doi: https://doi.org/10.54133/ajms.v10i1.2726

Dear Editor-in-Chief,

In biomedical research, many disease risks, pathways, and genetic mechanisms remain unexplained. Likewise, the universe holds deep puzzles, such as cosmic origin and dark energy, the origin of life, and consciousness, which test the limits of science and challenge our modest standing on Kardashev's scale of civilizations [1]. Similarly, while natural selection explains much, it may not account for all stochastic and deterministic forces behind genomic innovation and speciation. Rather than answer, this article invites interdisciplinary thinking: prebiotic chemical evolution, deterministic and stochastic models, genomic informational entropy, computer science analogies (code modularity, "spaghetti" architectures, semantic versioning), and gene regulatory networks. Integrating math and programming may reveal mechanisms that promote, constrain, or block speciation.

Chemical evolution and the origin of life

The debate over abiogenesis, life arising from non-living matter, is even tougher than those over species origins. Professor James Tour, a chemist and nanotechnology researcher, says that current chemical-evolution theories have five huge problems: making stable, sequence-specific polypeptides; making complex polysaccharides; making long polynucleotide chains; putting specific information into the first nucleic acids; and putting together membranes, proteins, and nucleic acids into the first living cell [2]. These obstacles underscore the extraordinary complexity required for the creation or emergence of life.

The stochastic nature of speciation, according to Darwin

Darwin's theory assumes that natural selection is the primary driver of speciation. Random genetic mutations that enhance an organism's fitness within a given environment increase its likelihood of survival and reproduction. Over generations, advantageous traits accumulate, leading to reproductive isolation and new species. Evolution leads to speciation, which is a mostly random process. It is caused by adaptive radiation, isolation (geographic, ecological, or reproductive), and changing selective pressures such as climate, competition, and predation. Classic examples are the Galápagos finches, where one ancestor produced diverse beak forms adapted to different diets [3], and the rapid diversification of mammals after the dinosaurs' extinction as empty niches were filled [4].

Speciation in the context of deterministic and non-deterministic models

There are two types of computer models: deterministic and non-deterministic. Non-deterministic models use randomness or quantum effects to produce different valid outcomes across runs. These methods are used in optimization models, Monte Carlo simulations, and randomized algorithms (for example, Randomized Quicksort) [5]. Speciation plausibly blends both: deterministic, in which persistent environmental pressures favor certain traits, while random mutation, drift, and chance events introduce novel variation, and randomness supplies innovation.

Are mutations strictly random and stochastic?

The study "A Possible Information Entropic Law of Genetic Mutations" compares the buildup of mutations to the second law of thermodynamics. It suggests that mutations increase informational entropy in genetic sequences, which can be tested by looking for changes in directional entropy in genomic datasets. Still, studies of the SARS-CoV-2 genomes showed that entropy decreased over time. This suggests that evolution can favor less informational disorder and that genetic variation may be shaped by non-random, possibly

deterministic, forces [6,7]. These findings challenge the Darwinian view of purely random mutations, implying that unknown deterministic mechanisms may shape genetic variation. Many studies showed that stochastic and deterministic forces jointly shape evolution across scales [5]. Rouzine and colleagues (2001) argue that viral evolution reflects an interplay of random genetic drift, selection, and mutation, with drift dominating small populations, natural selection large ones, and both acting at intermediate sizes; their Wright-Fisher diffusion model captures these dynamics and approximates viral behavior [8]. Russo *et al*. (2021) reviewed how stochastic and programmed DNA-methylation changes drive cancer heterogeneity and clone expansion [9]. On another note, Fleming (2017) argues that biology's principal constraint is not reductionism but a prevailing determinism, the belief that processes follow fixed, preordained paths, which obscures biological stochasticity [10]. The author cited the protein-folding research problem, which was long pursued as a strict "sequence → structure → function" problem until probabilistic methods achieved breakthroughs, and also, in cancer research, where the hope of isolating a few "driver" mutations gave way to appreciating tumors' chaotic, multilevel evolution.

Coding blocks and genes

In programming, snippets and blocks serve distinct roles: a snippet is a small fragment of source code, while code blocks (functions, classes, and libraries) provide abstraction, coherence, and reuse [11]. Analogously, a function resembles a single gene performing a discrete role, whereas classes or libraries map to gene clusters or pathways that encode complex traits. Biological regulation also uses genomic networks that talk to each other and give feedback, like endocrine circuits. One gene's effect depends on the effect of another gene, and co-regulation coordinates gene groups during processes like embryonic development. A notable example is the BRCA1 and BRCA2 genes, which cooperate in homologous DNA repair, and mutations increased in either breast or ovarian cancer risk [12]. Hox genes regulate one another to pattern the anterior-posterior axis during embryogenesis, specifying segment identity [13]. Sex determination depends on SRY activating SOX9, which reinforces SRY in a feedback loop that drives testis formation [14]. Such regulatory networks parallel modular code components coordinating to produce complex functions.

Biological "spaghetti" and modular code

"Spaghetti code" refers to poorly structured, tangled, and difficult-to-read or maintain software, often resembling a messy bowl of spaghetti [15]. It typically arises from a lack of planning, frequent quick fixes, deep nesting, and confusing jumps between sections, such as uncontrolled "Go to" statements in older languages. As a codebase grows, small changes cause unexpected failures and fragility. By contrast, modular design isolates functionality into testable components; repairing spaghetti code demands deliberate, deterministic effort rather than "lucky" random tweaks. In other words, accumulated random mutations can disorder genomes into "biological spaghetti" that resist further change. Early speciation may have been caused by random events, but as genomic entanglement grows, random or planned mutations become less able to create new phenotypes that can survive. Over time, this yields a "speciation plateau," constraining lineages within established taxonomic bounds much like spaghetti code that becomes fragile and hard to modify.

Semantic versioning and speciation plateau

Semantic versioning (SemVer) defines rules for numbering software releases with MAJOR. MINOR.PATCH to convey change scope [16]. MAJOR increases for incompatible or milestone changes, MINOR for added backward-compatible features, and PATCH for bug fixes. Seeing a version jump (e.g., 1.2.3 → 1.3.0 *vs*. 1.2.3 → 2.0.0) immediately signals whether updates are compatible or breaking. SemVer therefore simplifies dependency management, update assessment, and integration across projects. Semantic versioning mirrors biological evolution: MINOR updates (backward-compatible tweaks) resemble small genetic adaptations within a species, while MAJOR updates (backward-incompatible shifts) mirror speciation from substantial genetic change. Both hinge on compatibility and functional impact, in which small changes refine existing systems; large ones create new, incompatible lineages. As divergence occurs, further major transitions become rarer, limiting additional speciation.

Conclusion

Natural selection removes harmful variants and favors beneficial ones, but in tightly linked gene networks, "biological spaghetti," mutations are more likely to break function than create novelty. Genomic informational entropy analyses suggest limits to natural selection's "creativity." Finally, framing evolution as biological "semantic versioning" highlights that speciation demands a radical change analogous to a major software update.

Keywords: Biological evolution; Gene regulatory networks; Information entropy; Semantic versioning; Spaghetti code.

ACKNOWLEDGEMENTS

The authors would like to thank Mr. Abdul-Rahman Shukoor (second-year undergraduate medical student) at the College of Medicine, University of Baghdad, for his help as a programmer in clarifying the concept of "spaghetti code" discussed in this article.

Conflict of interests

The authors declared no conflict of interest.

Funding source

The authors did not receive any source of funds.

Data sharing statement

N/A

REFERENCES

1. Al-Imam A, Motyka MA, Jędrzejko MZ. Conflicting opinions in connection with digital superintelligence. *IAES Int J Artif Intell IJ-AI*. 2020;9(2):336-348. doi: 10.11591/ijai.v9.i2.pp336-348.
2. Evolution/Creation – Dr. James Tour [Internet]. [cited 2025 Apr 28]. Available from: https://jmtour.com/evolution-creation/
3. Darwin C. On the origin of species by means of natural selection, or the preservation of favoured races in the struggle for life. *Br Foreign Medico-Chir Rev*. 1860;25(50):367–404. PMID: 30164232.
4. Bromham L, Phillips MJ, Penny D. Growing up with dinosaurs: molecular dates and the mammalian radiation. *Trends Ecol Evol*. 1999;14(3):113–118. doi: 10.1016/s0169-5347(98)01507-9.
5. Joober R, Karama S. Randomness and nondeterminism: from genes to free will with implications for psychiatry. *J Psychiatry Neurosci*. 2021;46(4):E500–505. doi: 10.1503/jpn.210141.
6. Vopson MM. A Possible information entropic law of genetic mutations. *Appl Sci*. 2022;12(14):6912. doi: 10.3390/app12146912.
7. Motyka MA, Al-Imam A, Aljarshawi MHA. SARS-CoV-2 pandemic as an anomie. *Przestrzeń Społeczna (Social Space)*. 2020; 20(2):111-144.
8. Rouzine IM, Rodrigo A, Coffin JM. Transition between stochastic evolution and deterministic evolution in the presence of selection: General theory and application to virology. *Microbiol Mol Biol Rev*. 2001;65(1):151–185. doi: 10.1128/MMBR.65.1.151-185.2001.
9. Russo G, Tramontano A, Iodice I, Chiariotti L, Pezone A. Epigenome chaos: Stochastic and deterministic DNA methylation events drive cancer evolution. *Cancers*. 2021;13(8):1800. doi: 10.3390/cancers13081800.
10. Fleming L. Two dogmas of biology. *Philos Theory Biol*. 2017;9(2):1-14. doi: 10.3998/ptb.6959004.0009.002.
11. Zhang B, Fu L, Liang P, Yu J, Wang C. Demystifying code snippets in code reviews: a study of the OpenStack and Qt communities and a practitioner survey. *Empir Softw Eng*. 2024;29(4):78. doi: 10.1007/s10664-024-10484-2.
12. Yoshida K, Miki Y. Role of BRCA1 and BRCA2 as regulators of DNA repair, transcription, and cell cycle in response to DNA damage. *Cancer Sci*. 2004;95(11):866–871. doi: 10.1111/j.1349-7006.2004.tb02195.x.
13. Hubert KA, Wellik DM. Hox genes in development and beyond. *Dev Camb Engl*. 2023;150(1):dev192476. doi: 10.1242/dev.192476
14. Hawkins JR. The SRY gene. *Trends Endocrinol Metab*. 1993;4(10):328–332. doi: 10.1016/1043-2760(93)90075-p.
15. Politowski C, Khomh F, Romano S, Scanniello G, Petrillo F, Guéhéneuc YG, et al. A large scale empirical study of the impact of Spaghetti Code and Blob anti-patterns on program comprehension. *Inf Softw Technol*. 2020;122:106278. doi: 10.1016/j.infsof.2020.106278.
16. Preston-Werner T. Semantic Versioning. [cited 2025 Apr 28]. Semantic Versioning 2.0.0. Available from: https://semver.org/