Abiogenesis as the origin of adaptive evolution. An alternative to the Oparin-Haldane model

Juan Rivas-Santisteban[1,2]

[1]Department of Biodiversity and Evolutionary Biology, National Museum of Natural Sciences, CSIC, Madrid, Spain
[2]Milner Centre for Evolution, Department of Biology and Biochemistry, University of Bath, Bath, UK

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Correspondence: juan.rivas@mncn.csic.es

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Abstract

The emergence of life from non-living matter remains one of the most profound unresolved questions in natural philosophy. Current paradigms largely inherit the Oparin-Haldane assumption that abiogenesis is preceded by a prolonged accumulation of traits through both nonadaptive (e.g. self-organisation) and adaptive (i.e. Natural Selection) processes. Yet this raises a legitimate question: how can adaptive evolution occur before abiogenesis? Natural selection (NS) requires both a persistent resemblance between parental and offspring characters (i.e. heredity) and a source of transmissible variation (i.e. mutation); however the emergence of these properties is itself the central problem of abiogenesis. Here, instead, I describe a spontaneous adaptation model in which heritable variation arises nonadaptively, and therefore prior to the action of NS. The Price equation, although merely a mathematical tool, can help us think about the evolvability of NS itself. NS is best understood as the systematic non-null covariance between a trait value and its fitness, a quantity that itself evolves. Prior to abiogenesis, this covariance should be effectively zero. This model defines a null expectation in which adaptiveness is absent from prebiotic systems. It would be experimentally falsified by demonstrating adaptive evolution in a system that remains independently non-living.

Keywords: origins of life, abiogenesis, dissipative structures, Price equation, molecular bricolage, Oparin-Haldane hypothesis, gradualism, evolutionism, spontaneity

Introduction

L. Pasteur first showed that the spontaneous generation of complex life (*generatio aequivoca*) was impossible *de facto*. Every biological form known required a pre-existing living propagule (*generatio univoca*) [1–4]. After some intense debate, the experiments were certainly replicated, and the question settled [5]. R.L.C. Virchow's *omnis cellula e cellula* [6], together with the "immortality of the protoplasm" [7], remained dominant principles of biological reproduction for decades thereafter. Thus, although Pasteur's discovery filled an important gap in knowledge, an intriguing question emerged: whence do living things arise, if not from inert matter? [8, 9]

Besides a subtle margin for vitalistic responses –*à la* "life is eternal" [10]–, many experimentalists in that era confronted the question seriously. The general materialist answer was that evolutionism, not spontaneity, led to abiogenesis [3, 7, 11–14]. They argued that only a prolonged chemical evolution could explain the inert-living interface. As stated by A.I. Oparin: "(...) *these polymers, which were already capable of forming poly-molecular systems, were, of course, simpler than living protoplasm. Only later, due to the prolonged evolution of these systems, their interaction with the environment, and natural selection, did the types of organization that characterize living beings appear.*" [13], pg. 230. J.B.S. Haldane shared a similar view [15]. The Oparin-Haldane hypothesis [16, 17] rendered the best explanation available at the time: a prolonged phase of both, nonadaptive and adaptive evolution, prior to the origins of life (hereafter abiogenesis).

Here I argue, however, that this generalised response reflected a false dichotomy, i.e. the spontaneous emergence of life could not be accepted as a valid materialist explanation at the time [3]. I argue that this historical contingency is still influencing current theoretical and experimental frameworks to tackle the question (see the Section below).

Could we render alternative materialist models? In this piece, I elaborate on the feasibility of a spontaneous adaptation model, in which abiogenesis coincides with the origin of Natural Selection (hereafter NS). In contrast to the Oparin–Haldane model,

where pre-biotic adaptiveness is a prerequisite to reach abiogenesis, this alternative model forbids adaptive evolution in inert systems, as no mechanism exists to preserve heritable differences. Rather, it emerges *ex novo* when a chemical system spontaneously acquires a channel of inheritance that allows for variation, rendering the transition between non-life and life a discrete (albeit undirected) event.

Box 1. Evolution by Natural Selection?

The term "Natural Selection", or "selection" alone, is at times invoked to describe the differential elimination of chemical types [18]. Yet, in formal terms, differential survival of types does not equate to Natural Selection. Instead, it is regarded as the complete mechanism driving adaptation to a changing environment, often referred as Darwinian evolution [19–25]. Equating these two notions is misleading, because Natural Selection additionally requires replication, heredity, and variation among produced replicators.

Confusion may arise from the fact that even banal enrichment of types is both a natural and a selective process. Indeed, prebiotic mineral types available on Earth were determined by extrinsic constraints. That is to say, the space of chemical species realisable on Earth suffered a continuous, non-Darwinian evolutionary filtering prior to abiogenesis [26]. Thus, a system can be considered evolvable, natural and selective [27], but not in the Darwinian sense applied in this work.

For example, drastic acidification may alter the composition of a chemical pool in a primaeval ocean through selective elimination of the more unstable types, but such a process does not entail adaptation, or memory, at any level [25].

Finally, evolution in Darwinian systems is also subjected to non-Darwinian changes (e.g. drift via random sampling, random mutation, etc.) [28–31], but non-Darwinian systems cannot evolve by Darwinian rules.

The assumption of prebiotic chemical adaptiveness in current frameworks

Since Oparin–Haldane, prebiotic chemistry has remained, to varying degrees, conceptually rooted in the idea that adaptive chemical evolution precedes abiogenesis, from the classic experiments (e.g. Miller–Urey, Oró, Ponnamperuma, etc.) to modern prebiotic synthesis frameworks [32–53].

On the one hand, current perspectives that largely reminisce the Oparin–Haldane view commonly identify NS with changes in the frequency of prebiotic types (Box 1).

Here we find autocatalytic chemical ecosystem (ACE) models, where "molecular competition" and "predation" shift the frequencies of chemical types [54]. Such frequency changes are treated as NS-like processes, where Darwinian evolution is viewed as a gradually-emerging property rather than as a discrete transition [55]. As made explicit by Baum: "*Darwinian evolution is not a discretely delimited mechanism of change*."

Yet in a strict Darwinian sense, adaptiveness requires heritable variation associated with differential reproductive success. Therefore contingent enrichment of chemical types does not equate to NS (Box 1).

The GARD (Graded Autocatalysis Replication Domain) model provides an excellent complementary case. Compositional chemical assemblies were initially treated as pre-genetic units of selection [56], but later analysis showed that such a system may lack sufficient heritable variation to sustain adaptive evolution [42]. A system may be autocatalytic and self-sustaining without being adaptive [42].

On the other hand, current perspectives that substantially depart from Oparin–Haldane assumptions strictly adhere to the Darwinian sense of selection even in prebiotic settings, stating that NS cannot precede adaptiveness. For example, [57] argued that NS may operate in autocatalytic sets, but only where self-reproduction, heredity

and variation are granted. This distinction has also been captured by the term "auto-catalytic selection" [58], where NS "*can only operate on living systems and thus cannot be applied to the origin of life*". Likewise, [59] explicitly distinguished "chemical selection" from NS. Following the "Darwinian threshold" [60], the authors state that cellular structure is required for NS [59].

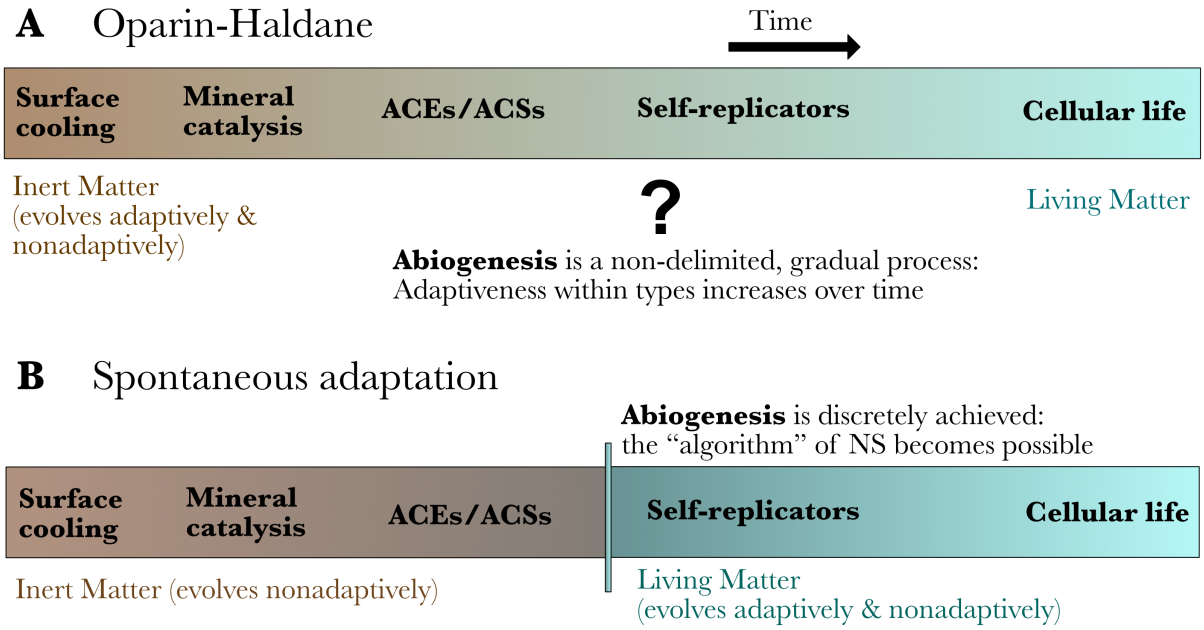

Figure 1: Contrasting views of the living-inert interface. (A) The Oparin-Haldane framework adopts a gradualist perspective in which life-like properties are acquired progressively over extended timescales. Under this view, both adaptive evolution and nonadaptive transformation of types are assumed to operate prior to the emergence of life. Abiogenesis is, at times, identified with the origin of cellularity; other accepted definitions instead emphasize metabolism or replication [61, 62]. (B) The spontaneous adaptation as a legitimate materialist model. Here, the inert–living transition occurs on the timescale of a single chemical event that establishes the requirements for NS and is itself undirected by NS. Adaptive evolution is therefore forbidden before abiogenesis, although matter may still evolve through nonadaptive processes (e.g. mineral evolution [26]). Importantly, the chronology of material events need not differ between the two views. The distinction is that Oparin–Haldane decouples abiogenesis from the origin of NS, whereas the spontaneous adaptation model identifies abiogenesis with the discrete advent of NS, when matter first acquires an evolutionary regime previously unavailable and from which later biological complexity –eg. cells– can arise, thereby allowing that transition itself to be explicitly interrogated.

Thus, such views implicitly assume that, while the evolution and complexification of matter were of course gradual processes, the onset of NS itself was not (Fig. 1). NS is, by definition, an emergent property that begins discretely when its enabling conditions first coexist. Whether these conditions are first achieved at the cellular [60],

self-replicator [24], or ACE level [25], each case represents a discrete, spontaneous transition toward adaptiveness.

The aim of this piece is to provide a general conceptual guideline and a formal basis for models that depart from the Oparin–Haldane view.

Evolvability of trait-fitness covariance

An implication of the Oparin–Haldane view is that NS operates continuously across the chemistry–biology transition, generating progressively increasing adaptiveness from the earliest stages of prebiotic evolution. But how could adaptiveness itself be established in such early systems?

First, adaptiveness requires heredity: the capacity of a material type to transmit information beyond its own persistence [24]. Heredity may be perfect or imperfect. Second, perfect heredity cannot generate heritable variation, thus long-term adaptation to ever-changing conditions requires a source of transmissible variation (imperfect heredity). Third, imperfect heredity requires information to be copied into newly sequestered material types (replication). Any system capable of adaptation must therefore be a replicator in a broad sense. Replication may be multiplicative ($N' > N$), conservative ($N' = N$), or partial ($N' = xN, ; 0 < x < 1$).

To merely assist our thinking, I make use of the Price equation, a formalism with the least assumptions, to explore whether adaptiveness is possible prior to NS. The Price equation [63] is commonly expressed as

$$\Delta\bar{z} = \mathrm{Cov}\left(\frac{w}{\bar{w}}, z\right) + \mathbb{E}\left(\frac{w}{\bar{w}}\Delta z'\right), \tag{1}$$

where $\Delta\bar{z}$ is the change in mean character value z between parental and descendant populations of chemical replicators. The first term measures the association between parental character value and relative replication success, $w/\bar{w}$. Under NS, systematic

differences in replication associated with trait z contribute to this covariance. The second term captures change during transmission, for example through replication errors.

The Price equation is therefore a statistical identity rather than a causal criterion for NS [64]. A related partition [65] is

$$\Delta\bar{z} = \mathrm{Cov}\left(\frac{w}{\bar{w}}, z'\right) + \bar{\delta}, \tag{2}$$

where the covariance relates relative replication success to descendant character value z', and $\bar{\delta}$ is the mean change during transmission. Eq. (2) separates replication-associated change from transmission change, but does not establish their cause.

This distinction becomes critical when considering the advent of NS. Before NS, some replicators may leave more descendants than others, but these differences are not systematically associated with inherited character state z. A particular population may therefore show positive or negative covariance between z' and w purely by contingency. Consequently,

$$\mathbb{E}\left[\mathrm{Cov}\left(\frac{w}{\bar{w}}, z'\right)\right] = 0, \tag{3}$$

where the expectation is taken over repeated realisations of the same system. With the onset of NS, inherited character states begin to systematically predict replication success, producing an expected non-zero covariance.

The onset of adaptiveness due to NS thus corresponds to the appearance of a systematic association between inherited character state and replication success, where the trait-fitness covariate begins to be controlled by such a NS-capable system, granting the expectancy

$$\mathbb{E}\left[\mathrm{Cov}\left(\frac{w}{\bar{w}}, z'\right)\right] \neq 0. \tag{4}$$

NS therefore introduces a persistent bias in the differential propagation of inherited states. This bias corresponds to adaptiveness. Adaptiveness, formally, cannot precede

NS.

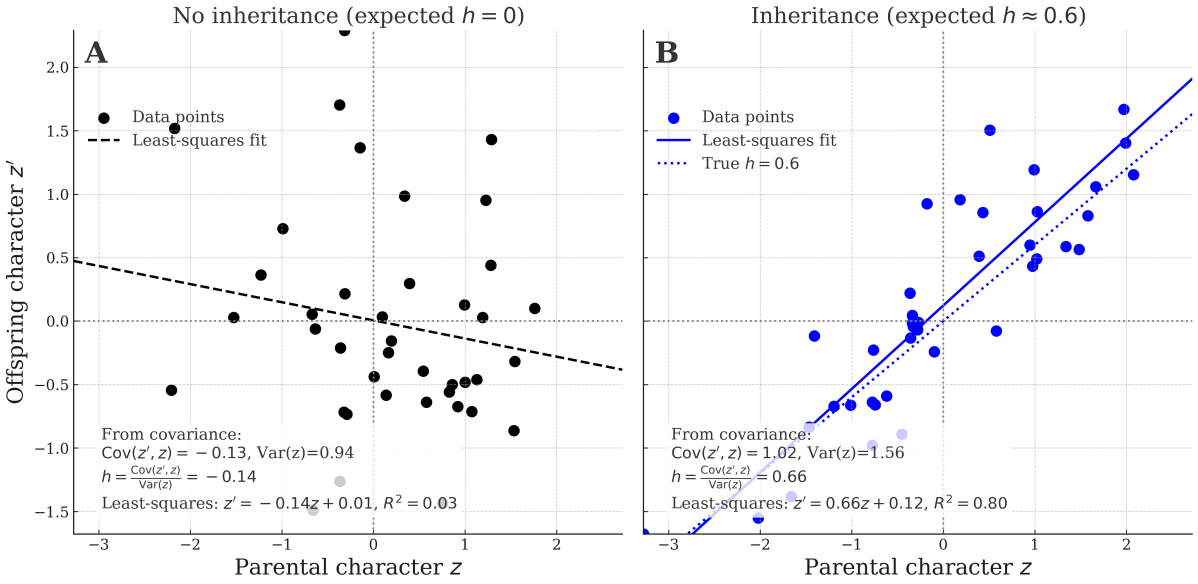

Figure 2: Two illustrative cases of weak (A) and strong (B) linear parent–offspring resemblance, quantified by the regression coefficient h. In both cases, replication and parent–descendant relationships are assumed to have been independently established. A population of parental character values (z) gives rise to descendant values (z'). In (A), parental values have little predictive power for descendant values, as expected when transmission of the focal character is weak or disrupted, for example beyond an error threshold [24, 66]. In (B), parental values predict descendant values, yielding positive covariance between z and z'.

A separate question concerns the emergence and identification of a persistent inheritance channel. Linear inheritance of character z can be quantified by

$$h = \frac{\mathrm{Cov}(z', z)}{\mathrm{Var}(z)}. \tag{5}$$

where h is the regression slope of descendant types on parental character values. See an example in Figure 2.

A non-zero h indicates linear parent–offspring dependence within an established inheritance channel: $h > 0$ denotes positive resemblance, whereas $h < 0$ denotes an inverse relationship. Conversely, $h = 0$ indicates only the absence of linear covariance and does not exclude nonlinear transmission. For example, if $z' = z^2$ and parental z is symmetrically distributed around zero, offspring state is fully determined by parental state while $\mathrm{Cov}(z, z') = 0$, because positive and negative parental values cancel in the

linear covariance.

Variation in h among true replicator populations may reflect changes in how character states are transmitted from parent to descendant types. Figure 3 illustrates this variation. Here, NS provides a statistical association between inherited character states and replication success.

However, as said, h quantifies the linear component of heredity but cannot by itself establish its presence or absence. Parent–offspring relationships must first be established independently of chemical persistence. For example, simple decay can generate strong time-lag correlations among chemical types, $z(t + \Delta t) = e^{-k\Delta t} z(t)$. The corresponding regression coefficient is positive and approaches unity when the sampling interval is short relative to the decay timescale. This may occur without replication.

Experimentally, the problem reduces to two questions. First, *is the character transmitted?* Replication and parent–offspring descent must be mechanistically established. Where such a mechanism exists, h can help quantify the linear component of heredity within the population (Eq. 5). Second, *does the inherited character predict replication success?* Replication output must then be quantified independently to test whether inherited states systematically alter offspring production, fulfilling the expectation in Eq. (4) and providing the signature of NS.

In this sense, adaptiveness is a consequence of the emergence of NS rather than a prerequisite for it.

Why does imperfect heredity allow for adaptiveness among offspring types?

Artificial replicators such as the Rebek system [67] illustrate more clearly why this is so. These are template-based autocatalytic replicators, but behave closer to perfectly faithful than Darwinian replicators, storing little information and, in theory, generat-

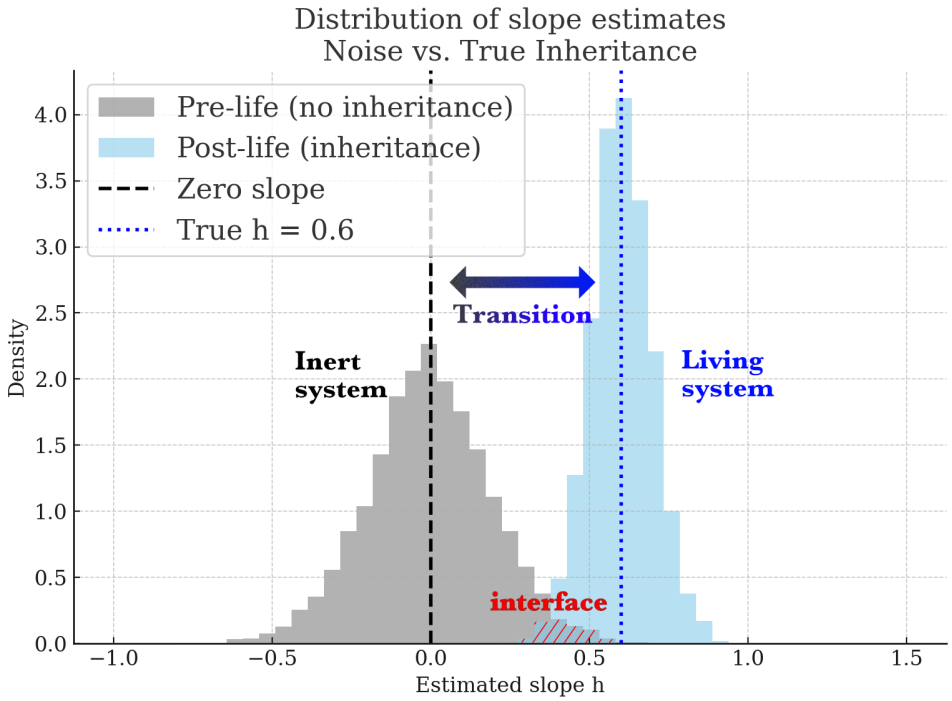

Figure 3: Illustrative distributions of h corresponding to the cases in Figure 2. Values are arbitrary and have no direct biological significance. Overlap between distributions may occur. The distributions represent differences in linear transmission fidelity among replicating systems and do not by themselves identify the onset of NS.

ing no heritable variation [68]. They are therefore *trivial* replicators, as their encoded information cannot readily generate alternative transmissible character states [69, 70]. Their expected trait–fitness covariance is therefore that of Eq. (3). Frequencies may still change contingently (e.g. through kinetics, precursor availability, environmental change, etc.) but such changes do not entail adaptation of surviving types. Without heritable variation, successful types cannot accumulate adaptive information about previously experienced environments.

Conversely, imperfect heredity is a prerequisite for NS, because adaptation requires heritable variation. This follows from the Fundamental Theorem of Natural Selection [20, 23] and is reflected in the Price equation. Yet excessive transmission error can erode the statistical association between parental and descendant states, eventually crossing an error threshold beyond which information about previously selected states can no longer be reliably maintained [66]. To maintain open-ended Darwinian evolution, abiogenic replicators must therefore have been variable, and capable of un-

limited heredity [24].

Alternative frameworks that formalise selection in a broad sense among populations of early replicative systems, such as the entropic selection principle [71, 72], could in principle be accommodated within the spontaneous adaptation model, provided that variation and heredity are already present.

The spontaneous adaptation model

Considering the above, I reconcile a materialist model of abiogenesis in which living types arise spontaneously. The preceding nonadaptive regime need not be instantaneous, as chemical complexity may accumulate gradually. Yet the transition from nonadaptive to adaptive evolution is discrete. It occurs when a sufficiently reliable inheritance channel is fortuitously established (Fig. 3), allowing parental character states to persist above transmission noise (Fig. 2) and, subsequently, to systematically predict replication success (Eq. 4).

Experimental falsification of the spontaneous adaptation model

Under this model, abiogenesis can be abstracted as a rare, undirected, mineral, spontaneous reaction; and, once it occurs, the descendant of the abiogenic types are forced to store information of the outcomes of the interaction between their characters and the environment. How can we determine whether the latter is a valid model for interrogating the origin of life?

The indefatigable surveyor

Let us consider the classical Oparin–Haldane example [15, 73], where UV radiation and other abiotic processes ensure a concoction of different chemicals within the primaeval oceans and act upon them with certain periodicity (e.g. day–night cycles), while others arrive episodically (e.g. impactors). Together, they continually produce,

transform, and extinguish material types.

Now suppose that we, as modern scientists, had access to an idealised Laplacian daemon devoted solely to the scrutiny of abiogenic conditions. It would possess complete observational access to this Earth-sized prebiotic experiment across time, maintaining detailed surveillance of how material types arise and transform within the soup.

Suppose that the indefatigable surveyor possesses effectively omniscient powers of measurement over this prebiotic world: every material type, its ancestry, instability, and environmental history are fully accessible to us. We may therefore follow any character z of a material type (e.g. amphiphile half-life, aromaticity, molecular weight) and its corresponding value z' in successor types. The Price terms merely provide statistical approximations to these underlying properties, useful for conceptual clarity but unnecessary to their direct observation by such peculiar daemon.

If the Oparin–Haldane continuum is correct, ours will be a rather tedious watch: adaptive properties should accumulate gradually, with no privileged instant at which inert matter becomes living, since both regimes are governed by essentially the same rules.

If the spontaneous adaptation view is correct instead, the trajectory of these adaptive parameters need not be linear. Different enabling conditions for NS may arise independently, out of sequence, or in combination, and the relevant parameters may change abruptly rather than gradually. The daemon should register multiple local ruptures in which heritable differences in the propagation of material types emerge, opening access to increasingly far-from-equilibrium states that were previously unattainable.

In both cases, chemical types may still accumulate or diversify gradually (Fig. 1). The critical distinction is that, under spontaneous adaptation, the adaptive parameters shift discretely from no systematic signal to a non-zero one. These parameters deter-

mine how material types evolve. At this point, their evolution can be adaptive.

Approximating the indefatigable surveyor

Unfortunately, we are no indefatigable surveyors. Thus, the thought experiment is useful only insofar as it can be reduced to extant experimental conditions. Two practical approximations may suffice.

The first is to approximate the daemon's measurements. Parameters of adaptiveness must be inferred from measurable properties of material types. The Price terms may assist this formalisation, but, for the reasons given above, are not themselves the quantities of interest. Further combined theoretical and experimental work along these lines is therefore required to identify suitable operational proxies.

The second is to constrain the daemon's search space. In the thought experiment, material types were screened across the entire Earth-sized chemical space–time. Experimentally, this must be reduced to tractable systems enriched for conditions enabling replication and imperfect heredity. The aim is to identify restricted regions of chemical space in which transitions to adaptive replication become observable.

A tractable falsification could build on ongoing work by Nghe, Fraccia and colleagues coupling polymer variation to compartment phenotypes [27, 74, 75]. In such systems, vesicle longevity may depend on the internal state generated by polymer replication. Mechanistically coupling this polymer state to stable vesicle replication would represent a critical experiment, because inherited polymer variation z could then generate differences in vesicle persistence that feed back on fitness. Where parent–offspring connection is demonstrated, h could quantify linear heredity, while trait–fitness covariance would hint the emergence of NS.

Under spontaneous adaptation, this coupling should emerge in the absence of prior adaptive states and produce a discrete regime shift in vesicle persistence, accompanied

by the appearance of a systematic trait–fitness covariance. Within a few vesicle generations, NS should rapidly enrich the inherited polymer states associated with greater replication success.

If coupling polymer heredity to vesicle reproduction produces no abrupt qualitative shift in system properties or trait–fitness covariance, this would be more consistent with an Oparin–Haldane continuum, because prebiotic sorting and NS would remain experimentally indistinguishable in this system.

Other recent contributions support the experimental validity of spontaneous adaptation models [76, 77]. I briefly summarize the main findings.

The first experiments show that a variety of semihollow structures, with distinctive morphology and thickness, were achieved spontaneously in a Miller-Urey experiment [76]. The latter consist of silicon-enriched HCN polymers, a kind of compartment not present in modern-day biology. Instead, the second experiments show how photo-RAFTs, or reversible addition–fragmentation chain transfers driven by light pulses, lead to the polymerization-induced self-assembly (PISA) of some components, selected *ad hoc* [77]. These, importantly, are not based on modern-day biological parts, but they spontaneously form micelles that "self-reproduce". Although molecular changes that occur in these micelles over time are the result of contingency rather than NS, as they have no possibly enabled inheritance channel and thus they follow the nonadaptive regime (Fig. 2), this could be an interesting result to be falsified.

Historical decoupling of adaptive evolution and abiogenesis

In the Oparin–Haldane view, the emergence of NS and the emergence of life remain decoupled in time. A.I. Oparin stated that natural selection occurred in non-living molecules [73], but also that the first "living thing" is not a poly-molecule, but probably a "slime": "*with certain reservations we can even consider that piece of organic slime* (...) *as being the first organism*" [73]. J.B.S. Haldane, contrary to A. I. Oparin, recognised that

"*the first living, or half-living things were probably large molecules synthesised under the sun's radiation*" [15], thus treating life as a continuum rather than as a discrete quality. This is an important discrepancy [78] that has been largely neglected in posterior literature. However, Haldane also acknowledged: "*clearly we are in doubt as the proper criterion to life*" [15].

Indeed, at the time of elaboration of the Oparin-Haldane narrative, non-adaptive drivers of evolution were largely neglected, maybe with exception of the Fisher-Wright models in population genetics [20, 79]. Now, we do acknowledge that a substantial portion of the variation found in extant replicator populations is due to nonadaptive processes [29, 31]. It is sensible to think that the generation of variability in non-living compounds, which are not purified, was not directed by any adaptive forces (memory of traits that worked better), because there were, by definition, no available channels of inheritance to allow for NS (Eq. 5, Figs. 2& 3). That is to say, variability in the pool of inert types will be determined, contingently, by the local chemistry and the events disturbing that chemistry.

Thus, under a spontaneous adaptation model, the discrete moment at which the conditions enabling NS are attained coincides with abiogenesis. This is because life-defining properties like cellularity, organismality, metabolism, homeostasis, genetic code, compartmentalisation or endosymbiosis are not causes of NS, but collateral consequences of NS-capable replicators with imperfect heredity.

Final notes

I make no claim that this model provides a more correct account of abiogenesis. Rather, I argue that spontaneous abiogenesis models remain comparatively unexplored and poorly formalised, despite representing legitimate materialist alternatives. Importantly, some generate experimentally distinguishable predictions from the Oparin–Haldane framework.

Under the Oparin–Haldane view, abiogenesis follows a prolonged mixture of adaptive and nonadaptive change, leaving its precise occurrence vague. Under spontaneous adaptation, it is instead a spontaneous event from non-adaptive precursors. Before this transition, adaptive parameters among material types should show no persistent signal. Afterwards, inherited types begin to predict replication success. This alternative is therefore useful (1) as a null, (2) to explore chemical routes outside any prior adaptive trajectory, and (3) to trace quantitatively the first departure from the prebiotic expectation.

Resource endowment, for instance, drives important nonadaptive changes in the frequency of prebiotic types [27, 72], but adaptation to resource dynamics is allowed only when those changes become coupled to fitness.

It could be argued that this model merely places the threshold for abiogenesis at very low complexity. A population of simple replicators lacking environmental isolation might satisfy its conditions while remaining "infrabiological" in the sense of [62]. Yet this remains a qualitative position along a continuous complexity gradient. The search for the origin of life is therefore inherently ambiguous under such definitions. By contrast, the present model provides a quantitative criterion for tracing a discrete onset of adaptive evolution experimentally.

Other definitions of life [61] emphasise features such as genetic coding, compartmentalisation, and metabolism. Under the proposed view, these are contingent on prior adaptiveness. NS therefore marks the inflection point from which such higher-order properties become evolutionarily accessible, and thus the defining inert-living boundary.

Interestingly, viruses are not autonomous self-replicators, and under this model would not qualify as living unless cellular machinery enables their reproduction and imperfect heredity [80]. Self-replicators sustain these conditions autonomously.

Finally, two questions are often conflated: (i) how life can emerge in general, and (ii) how life historically emerged on Earth [81]. The second is contingent on local chemistry and historical events, such as impactors or orbital change. The first instead requires general principles, which necessarily depend on an operational account of life [81]. Such principles should nevertheless be theoretically formulable and, in principle, experimentally testable.

This piece addresses question (i). Question (ii), while equally important, is closer to a form of "molecular palaeontology": reconstructing the historical sequence by which life emerged on Earth. By contrast, question (i) asks under what general conditions inert matter can acquire the defining properties of living systems [81]. In this sense, the Oparin–Haldane model provides neither a general nor a quantitative criterion for delineating abiogenesis (Fig. 1).

Here, I propose what I believe is a novel perspective on problem (i). In the end, the quest might still be to figure out the material rules governing a (very) elusive *generatio aequivoca*. What an irony!

Conflict of interest

I declare no conflict of interest.

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