

# Memory about the past is beneficial for survival and reproduction (reply to comment

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by V I Klyatskin on “21st century: what is life from the perspective of physics” (*Usp. Fiz. Nauk* 180 337 (2010) [*Phys. Usp.* 53 327 (2010)]) by G R Ivanitskii

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**Abstract.** The fact that the first living structures were learning in the process of evolution resulted in no evolutionary step being completely random. This learning was impossible without ‘memory’ about the past: survival-enhancing situations should be remembered to accumulate experience for survival and reproduction in the future.

V I Klyatskin notes in his letter [1] that the processes proceeding in chaotic media are characterized by the emergence of specific structures in which a growing quantity frequently reaches high values. This assertion is correct: the formation mechanism of such structures is known [3]. Such events are characteristic of both *living* and *nonliving* systems, as also mentioned in my paper [2, p. 344]. According to V I Klyatskin, characteristic clustering time in space is determined by statistical Lyapunov index  $\alpha$  and diffusion coefficient  $D_f$  in the phase space of a positive field function  $f(\mathbf{r}, t)$ . We both agree on this point. The main riddle of the origin of life is not the fact of structure formation itself but *the time intervals from cluster formation till disappearance, i.e., cluster lifetime  $\tau$ . A newly formed cluster must remember conditions of its birth to become a ‘living structure’ and transfer this memory to the progeny. The ‘intermittency’ concept [3] explains neither the appearance of the genome nor the mechanism of accelerated biological evolution.*

## Time deficit

Suppose we need to assemble a certain device having only two hierarchical levels, i.e.,  $k$  nodes composed of  $n$  pieces each. The total number of assembly parts is  $kn$ . Let us consider three variants of assembly work.

(1) Given the assembly instruction is provided, one needs to perform only  $(k-1)(n-1)$  operations.

(2) In the absence of instruction, the assembly is performed independently at two hierarchical levels by the rule-of-thumb method, sorting out constituent parts inside the nodes and thereafter the nodes themselves. In this case, the number of required operations per node is  $n!$ , and their total number reaches  $\langle k!n! \rangle$ .

(3) In the absence of such a criterion and the necessity to estimate the success of assembly only from the final result (obtained by a random combination of all pieces and blocks), complete sorting of all combinations is needed. For example, in order to open a lock with a  $kn$ -digit code, the number of operations to be performed comes to

$$(kn)! \approx (kn)^{kn}.$$

Suppose a device consists of 4 blocks, and each block contains 4 pieces ( $k = n = 4$ ). Let each operation take only 5 s to be performed; then 9 operations and 45 s are needed to assemble a device according to instruction. Assembly by hierarchical sorting takes 576 operations and 48 h, and by completely random sorting  $16! = 2 \times 10^{19}$  operations and approximately  $5 \times 10^{11}$  years. These figures illustrate increased time expenditures upon transition from a totally determined to a totally random assembly.

Certain authors adhere to the former scenario of the origin of living matter [4, 5]. I discard the former scenario as leading to creationism. The controversies between V I Klyatskin and myself arise from his giving preference to the latter scenario, and my preference for the second one. When improving the genetic text in molecular biophysics, we called this process ‘the block-hierarchical selection (BHS)’ [6, 7]. Over 20 hierarchical levels can be distinguished in biological structures exemplified by *molecular structures*  $\rightarrow$  *genome*  $\rightarrow$  *cells*  $\rightarrow$  *individuals*  $\rightarrow$  *populations*  $\rightarrow$  *species*. Biological structures of each level possess a definite lifespan limit  $\tau$  and reproductive potential.

Paleontological findings give evidence that forming structures remembered past successful experiences in the process of biological evolution. Existing living systems have a genome. Otherwise, no further complication of these systems would be possible, as creationists rightfully assert [4, 5]. This argument is refuted on the assumption that biological structures and organisms arising from them could ‘learn’ in the course of evolution and each new evolutionary step was not random, but rather a quasirandom or deterministic

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stochastic process. Otherwise, we arrive at the *time deficit paradox* (see paradox 3.5 in my paper [2]). *Memory must fix successful variants and thereby prolong their influence on subsequent development.*

The ability to learn and remember is a specific property of living matter. It results in a ‘development flow’ in the phase space that collapses *with acceleration* into a certain region corresponding at each hierarchical level to an improved new living structure. V I Klyatskin [1] maintains that structure formation is also a region in the phase space, but an arbitrary one rather than concrete, and the process *slowly* collapses into it. In other words, his scenario implies neither ‘learning’ nor selection, and structure formation is only a property of the environment.

A natural question arises: how was learning realized in primitive biological structures? I called the phenomenon of learning the collecting term ‘memory’. To clarify our positions on the controversy, it should be explained what is meant by the word ‘memory’. Usually, ‘memory’ is defined as *the process of storage of past successful experience, making possible its reuse in the present and future.*

### What unites and disunites our models

Evolutionary genetics is usually described in the language of mathematical linguistics, although other languages (probably more comprehensible) can be used as well, e.g., the language of growth and death of random and quasirandom graphs.

Let us denote the progeny of the  $i$ th reproducing prastructure of the  $j$ th level by  $x_{ji}(t)$ :

$$x_{ji}(t) = n_{ji}(t) \omega_{ji}(t) \tau_{ji}(t), \quad (1)$$

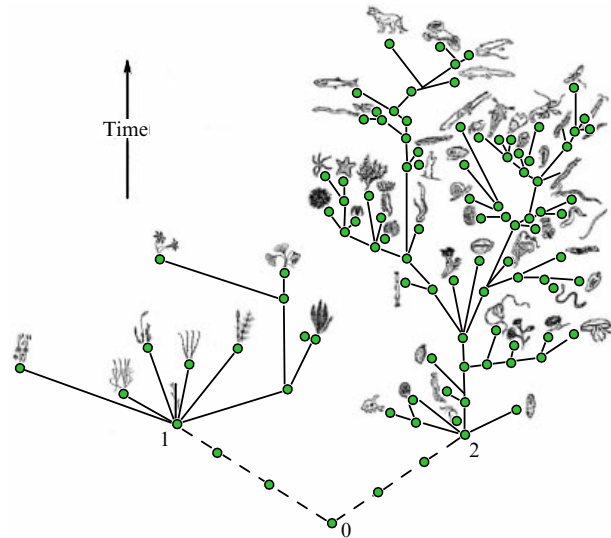
where  $t$  is time,  $n$  is the number of ‘children’ per litter,  $\omega$  is the birth rate, and  $\tau$  is the lifespan of the ‘parents’.

Biological evolution is a *branching process* with propagation of objects. The current great interest in the formation of branched networks and processes under different conditions is supported by Internet activities and teaching living organisms through intracerebral bond formation [8]. Let us denote by  $x_0$  the structure that was the first to form, e.g., via the mechanism described in Klyatskin’s comment. The result of its successful experience is denoted by  $M(x_0)$ . It is determined only by the fact that the structure exists at the time instant  $t = 0$  under given environmental conditions and reproduces, say, by division into two structures. Reproduction is the main property of living systems. Thus, let  $x_1, \dots, x_t, \dots$  be the result of survival secured by reproduction; then, one can write

$$x_0 = 1, \quad x_t = x_{t-1} + x_t - 1. \quad (2)$$

Such a description is isomorphic to the reproduction of successful experience.

If a structure brought forth offspring, the experience  $M(x_1)$  was reproduced, while the prastructure itself disappeared (died) after a time period  $\tau$ . Notice that it could die leaving no progeny if its previous experience  $M(x_1)$  was unsuccessful. In this case,  $M(x_1) = 0$ . This process repeated at the next stage with  $M(x_2)$ ,  $M(x_3)$ , ..., etc. Evidently, certain structures having poor experience  $M(x_i)$  represented the developmental dead end. They degraded while their successful counterparts could live forever (at least as long as the substrate necessary for their offspring to develop was available). Environmental changes promoted selection of successful structures. Such biological evolution was described by Charles Darwin [9] (variability, heredity, selection). Later



**Figure 1.** A hypothetical phylogenetic tree: branch 1 — terrestrial plants, and branch 2 — fauna objects.

on, numerous attempts were undertaken to describe this process mathematically (see Ref. [10]). But a more serious thing is how to explain the acceleration of biological evolution from arising to dying structures at the population level.

Figure 1 shows a standard graph of biological species evolution. It suggests directional growth and amplification of biological species.

### Hierarchical randomly growing graphs

Randomly growing networks or graphs represent distributions asymptotically leading to power laws. Suppose a graph has  $P(x)$  vertices. At large  $x$  values, function  $P(x)$  is defined by the power law

$$P(x) \sim cx^{-\gamma}, \quad (3)$$

where  $c$  is the normalization constant, and  $\gamma$  is the exponent. Let this graph have  $v$  vertices, i.e.,  $P(x) = v$ . The sequence of powers ( $\deg v$ ) of a graph<sup>1</sup> can be converted into a one-dimensional decreasing series [11]. Function  $f(v) = \deg v$  in the form of a one-dimensional diagram is isomorphic to the graph, since it has the same power sequence  $\deg v$ . Function  $f(v)$  can be considered an ordinary distribution function, much as V I Klyatskin does in his comment [1]. The presence of the gently sloping ‘tail’ in function  $P(x)$  relates the model to this process.

However, we are interested in the network growth or death in time. Let us denote the set of all possible vertices  $\langle v \rangle$  by  $E$ , regarding the network growth as being random [12]. The most important characteristic of scale-free networks is the sum of the vertex powers above the average level. The highest power of the node is the network core, depending not only on the power of a hub<sup>2</sup> but also on its surroundings.

The network lifetime correlates with the total graph power  $S$ :

$$S = \sum_{v \in E} \deg v. \quad (4)$$

<sup>1</sup> The quantity  $\deg v$  denotes the power (valence) of the graph node, i.e., the number of edges reaching vertex  $v$ .

<sup>2</sup> Hubs are referred to as vertices (nodes) with the maximum number of edges (links).

The summarized power decreases when graph edges are torn. Evidently, hub powers exceed the mean graph power and hubs themselves are most stable and long-lived. If environmental changes destroy the edges, the graph may turn into a set of separate minigraphs containing isolated hubs. This network property has been studied analytically with the application of the percolation theory [13]. The opposite statement is also true: if new links (edges) appear between vertices in the node field of isolated minigraphs, the latter gradually combine together into a graph with a large total power  $S$ . However, power sequences do not always determine graph topology unambiguously [14]. The Erdős–Gallai theorem makes it easier to clarify such situations [15].

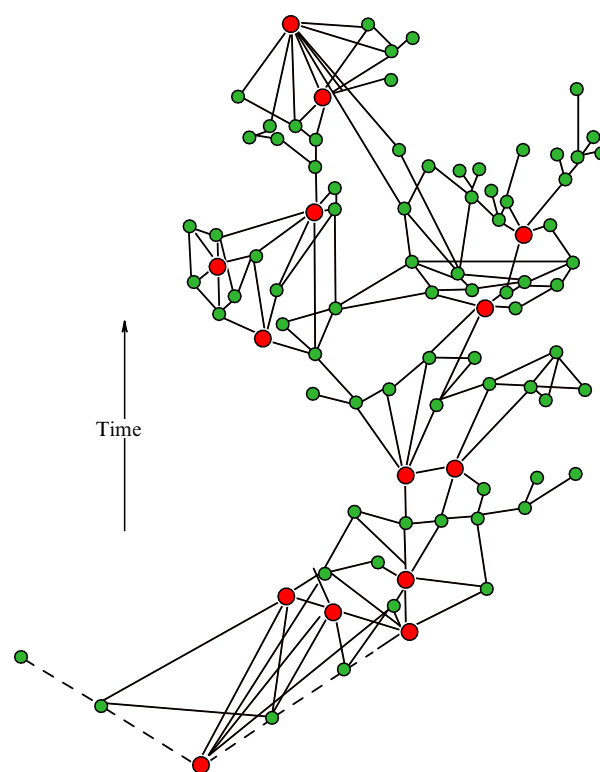
Isolated edge-free nodes possess a zero power. They may serve as a substrate for future graph growth by forming additional links. This possibly accounts for the fact that less than 30,000 genes (instead of the expected 100,000) were identified when the three-billion human nucleotide genome was unveiled. The remaining nucleotides appear to be zero graphs.

The mechanism of graph growth reduces to re-organization of graph topology in space. Low-power graphs are, as a rule, linked via hubs. The concrete characteristics of the network scale growth vary under the effect of the new link generation mechanism. In random capture, the network growth, for example, of a genetic graph partly disobeys the power law: it sometimes resembles normal distribution [16–19]. As far as biological evolution is concerned, the bond energy in the graph appears to have created kinetic components of the process, and was its main characteristic because temperature fluctuations of the medium were responsible for the selection and growth of genetic graphs. In this case, each node of a graph can be regarded as a set of covalently bound chemical elements, and graph edges as weaker hydrogen or van der Waals bonds. These bond energies are a few orders of magnitude different. Specifically, the hydrogen bond energy ranges 5–10 kcal mol<sup>-1</sup>, while the energy of an O ↔ H covalent bond is 109 kcal mol<sup>-1</sup>. Weak bonds played an important role when the mean temperature of our planet decreased; they stimulated self-assembly of the simplest genetic structures at the early stages of evolution [20].

The node powers in a more or less stable graph gradually decrease when moving along the vector from the network core to periphery. In this case, random damage to the edges does not significantly contribute to the hub death. Such network topology ensures a long life of the hub. Only strong environmental changes can disturb hub connectivity. These properties are lacking in other configurations of the network scale displacing high-power nodes toward the periphery. Therefore, the *lifetime*  $\tau$  of the network may be significantly different depending on its topological features [21]. Figure 2 demonstrates a hypothetical network of one of the branches of the phylogenetic tree at the species level.

Another important characteristic of networks pertains to the mean interhub distance. As shown in Ref. [22], a random network lacking preferred directional growth in space with  $\gamma$  values ranging  $2 < \gamma < 3$  will have a compact structure. The diameter of the increasing scale of such a network may be regarded as constant. Their power density is high compared with that of an ordered network, e.g., a grid.

The growth model of *randomly evolving networks* is based on the random formation of internode links, in which two nodes at each step are selected in a random fashion. A graph created in a certain random probabilistic process is random as



**Figure 2.** A hypothetical network of one of the branches of the phylogenetic tree. Large circles denote 14 hubs for which  $\deg v > 4$ . Clearly, an environmental attack may split the network into 4–5 clusters.

well. The properties of such networks and those formed in a nonrandom fashion are different [23]. The theory of random graphs lies in the region of overlap of the graph theory and the probability theory. Specifying many  $E$  nodes and then randomly adding edges between them give rise to a randomly growing graph. The best known is the formation process of random graphs proposed by Gilbert [24] and designated as  $G(n, p)$  (by the first letter of the author's surname). The periphery of this graph extends independently in all directions with probability  $p$ . Closely related to this is the graph  $G(n, M)$  model (Erdős–Gallai model [25]) in which the probabilities of bond formation at the graph periphery are random but equal. The graph grows in a uniform manner in all directions. Structures from 'chaos' described by Klyatskin [1] grow in a similar way.

In the generalized Gilbert model [24], a directing vector puts in correspondence with each graph vertex. This vector field makes the graph grow in different directions and at different rates. In biology, such growth at the population level corresponds to the self-governing growth mediated through tactic responses (phototaxis, chemotaxis, gravitaxis, etc.). Taxis-governed graphs, unlike random ones, lead to a specific case.

### Growing graphs will memory

Another important feature of graph expansion is the capture of small networks by large ones. This variant of growth is defined by a different model [26]. In this case, accelerated growth of the graph structure proceeds associated with the capture of small networks. It leads to the power-law graph growth typical of processes in which the structure formation rate increases in time proportionally to the already reached level. This model differs from that considered by V I Klyatskin

[1]. Only this model explains the distribution of structural diversity accumulation in biological systems and amplification of organisms in the process of phylogenesis [27].

Such models are extensively studied as having a variety of sociological, economic, and logistical implications. These are *connected processes in which the probability of structure formation increases in time in proportion to the already reached level (preferential attachment process)* [27]. Each newly emerging random structure (graph vertex) starts from the  $M(x_0)$  experience. New reproducing vertices will enter such a structure at a rate proportional to the number of already existing vertices  $x_i$  corrected for a certain constant quantity  $\alpha$  (accelerating factor):  $\alpha > |\pm x_0|$ . The vertex distribution function  $P(x)$  in the limit is likewise a power-series distribution with a ‘tail’:

$$P(x) \propto \frac{1}{x^\gamma}. \quad (5)$$

The fraction  $P(x)$  of new vertices introduced into the growing structure is described by the expression [27]

$$P(x) = \frac{B(x + \alpha, \gamma)}{B(x_0 + \alpha, \gamma - 1)} \quad (6)$$

for  $x \geq x_0$ , where  $B(x, y)$  is the Euler beta function in the form

$$B(x, y) = \frac{\Gamma(x) \Gamma(y)}{\Gamma(x + y)}, \quad (7)$$

where  $\Gamma(x)$  is the standard gamma function.

Assuming the lifetime  $\tau$  of an individual as an argument  $x$  at the population level, the distribution  $P(\tau) = P(\tau_0) \exp(-c\tau)$  has a long tail of long-lived vertices (in the present example, individuals of a given species). Tail formation involves the accelerating factor of evolution providing advantages to long-living structures and therefore bringing forth more offspring. One example of hypothetical functions of structures’ life expectancy  $\tau$  and distribution  $P(x)$ , where  $x = \tau$ , is shown in Fig. 3.

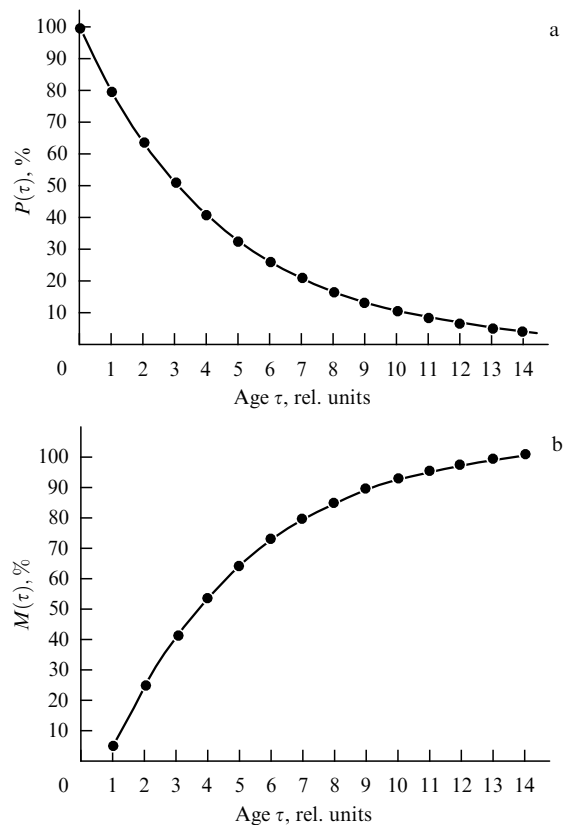
Maximum values of the distribution of contributions from each structure to the growth of offspring  $M(\tau)$  in the dependence of its life expectancy are shifted toward long-lived structures. The essence of the process lies in the fact that long-lived structures are rare phenomena fitting the population ‘tail’ (Fig. 3a). In the end, however, just these structures make the greatest contribution to the acceleration of evolution, because they live long and bring forth a large number of offspring.

The preferred input process accounts for the distribution of not only living species but also events occurring in social systems, such as the distribution of cities by population size, the wealth distribution in the society, the distributions of individual’s research output over the number of scientific publications and of the number of their citations, etc.

In the context of biological evolution, each genus starts from a single species ( $x_0 = 1$ ) and new species evolve from praorganisms as a result of mutations in direct proportion to their already existing number at  $\alpha = 0$  and  $x_0 = m$ , and the duration of the reproductive period; therefore, one finds

$$P(x) = \frac{B(x, y)}{B(x_0, \gamma - 1)}, \quad (8)$$

where  $\gamma = 2 + 1/m$ . In the model developed by Barabási and Albert [26], an estimation of scientific paper citation is based on the same principles. The longer a scientific publication continues to attract interest (the longer it ‘lives’), the higher its



**Figure 3.** (a) Survival curve  $P(\tau)$  of ‘parents’ in a hypothetical population of reproducing, living structures of reproductive age, i.e., not subject to aging. (b) Curve of the integrated contribution  $M(\tau)$  of parents to the increasing number of offspring for each age group of the parents.

citation index. This case corresponds to

$$x_0 = 0, \quad \alpha = 1.$$

Sometimes, such a process is called *the Matthew effect (the rich will get richer, while the poor will get poorer)*<sup>3</sup> [28].

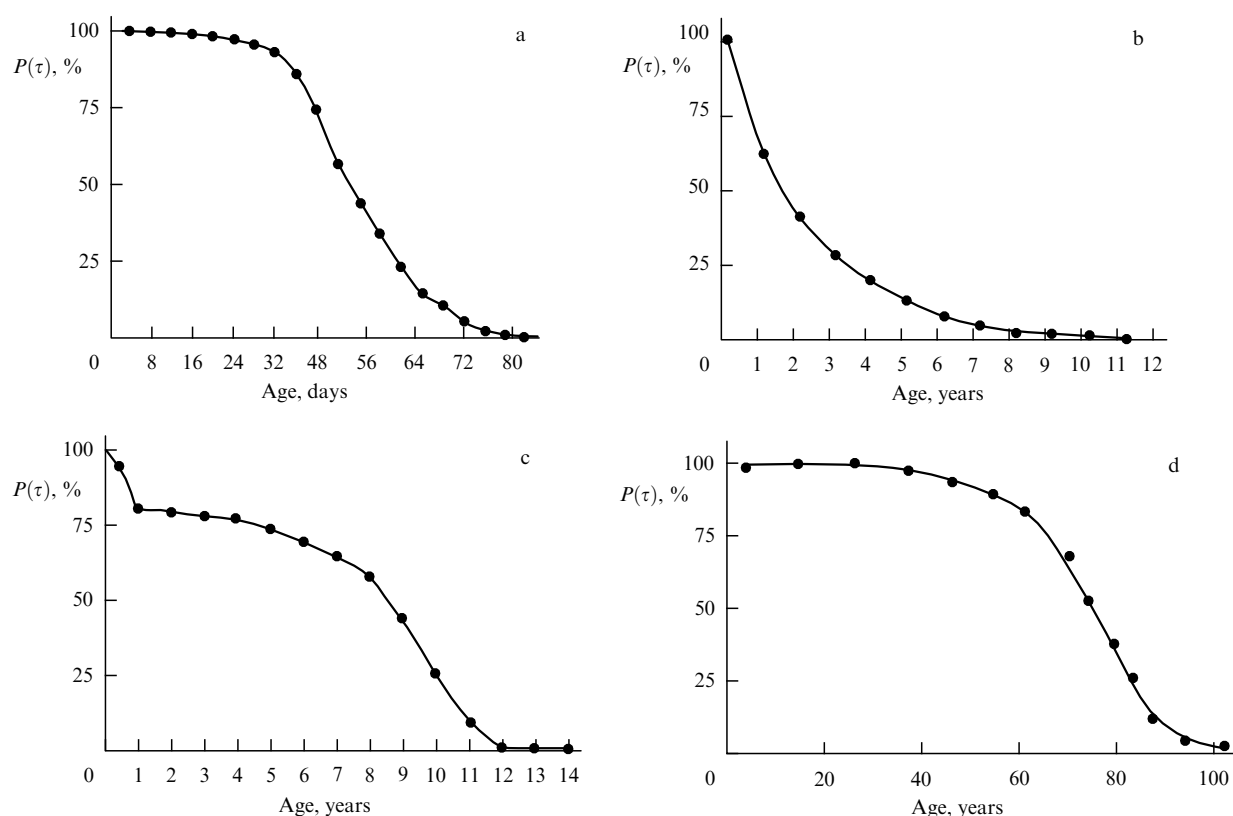
The processes in which the rate of structure formation is proportional to the previously reached level can be regarded as a variant of systems of random processes *with positive feedback and memory realized at the respective hierarchical level*. Memory accelerates biological evolution [7].

This is my principal conjecture, at variance with V I Klyatskin’s.

One of the first such models in biology was the taxon formation model for biotic organisms, proposed in 1925 by Yule [29].

Unfortunately, nobody knows what biological structures were on Earth at the dawn of evolution, but there are hundreds of hypotheses addressing this issue. Most researchers believe, however, that structures capable of replication (reproduction) were in all probability polymers composed of parallel RNA and DNA type chains present in the currently existing living cells [30]. Having survived and reproduced organisms did not always meet *the preferred input criterion for random networks with a simple power-law growth dependence*

<sup>3</sup> The term was coined from the parable in *The Gospel of Matthew*, Ch. 13: “For whosoever hath, to him shall be given, and he shall have more abundance: but whosoever hath not, from him shall be taken away even that he hath.”



**Figure 4.** Dependences of survival  $P(\tau)$  (percent of survived structures) for real populations of reproducing, living individuals of different species: survival distribution (a) for insects (male *Drosophila melanogaster*); (b) for birds (Lapwings *Vanellus vanellus*); (c) for mammals (Dall's sheep), and (d) for humans (men in England in 1960–1962) [31].

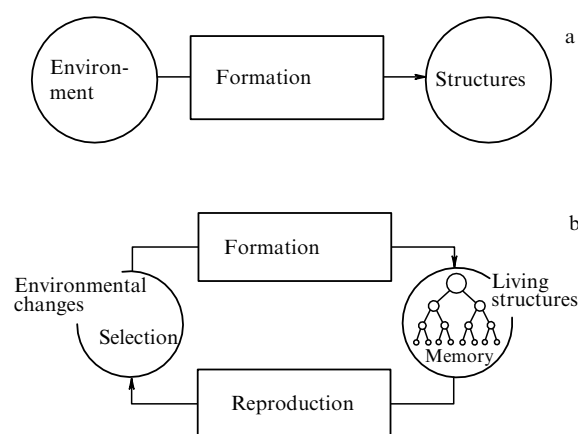
$\exp(-a\tau)$ . Figure 4 illustrating this situation depicts dependences of survival  $P(\tau)$  for real populations of living, reproducing individuals of different species [31].

As species become increasingly more complicated, their lifetime surpasses the length of their reproductive period, and a new mechanism for learning offspring develops. Learning in primitive structures and unicellular organisms has only two outcomes: *either adaptation to the environmental conditions enabling the species to reproduce, or the inability to adapt and death*. In multicellular organisms, especially mammals, learning provided an additional advantage: *knowledge from the experience of elders*. The character of reproduction changed accordingly.

It appears clear that random emergence of structures in chaotic media that gave rise to living matter and its evolution from protozoans to mammals, including humans, is a necessary but not sufficient condition. Figure 5 illustrates the discrepancy between V I Klyatskin's and my views.

### Biological evidences

Unusual insertion sequences (ISs) were discovered in bacteria in the 1960s. Hopping and insertion genetic texts were documented in fruit flies (*Drosophilae*) and plants (maize) It becomes evident that mixing of genetic texts involves not only 'letters' or 'syllables', i.e., micrographs (IS elements), but also 'words' (transposons) and 'sentences' (episomes or complex plasmids), i.e., oligographs, as well as whole 'texts' (macrographs) (crossing-over in sexual mating). New genetic texts considerably affected regulation at all hierarchical levels of living structures. A popular hypothesis maintains that mitochondria are prokaryotic (bacterial) intracellular inclu-



**Figure 5.** Schematic of a structure formation proposed by V I Klyatskin (a), and my scheme of the appearance and evolution of living structures (b). The living structure in scheme (b) is a hierarchical structure that remembered beneficial variants of its changes meeting environmental conditions in the process of reproduction (and thereafter learning).

sions in karyotes, i.e., symbiosis leading to integration into a single organism. It is equally well known that the mixing of genofunds at the bacterial level occurred via gene transfer by viruses. A book by R B Khesin [32] dealing with gene transfer and genome integration was published in the mid-1980s. The first part of my paper [2] gives examples of horizontal genomic links in organisms. It can be argued that something like genetic engineering appeared at the initial stages of biological evolution under natural conditions.

Definitive evidence of the benefits of genomic network growth for the expansion of long-living progeny was obtained as early as the 1950s by mating insects, e.g., *Drosophila* [33].

*Climatic catastrophes* temporarily impoverished species diversity, on the one hand, and *repleted the genofund of our planet at the level of microorganisms*, on the other hand; thereby, the post-catastrophic stages of evolution were promoted. The evolution peculiarity lay in the fact that gene transfer accelerated evolution, but randomness sometimes made it wasteful [7]. Living matter possessed of memory gradually turned into an extended network of genetic texts, i.e., the planet's biosphere.

Gene graphs migrated over various biocenoses, besides undergoing rare and random mutations. Genetic exchange occurred even between totally unrelated organisms, such as bacteria, higher plants, and animals. De Vries wrote about mutation periods ('jumps in mutagenesis') as early as 1900 [34].

### Is it possible to prove which biological evolution model is valid?

The main property of any model pretending to be a scientific theory is its *internal consistency*. Abstracting from practical application of a theory, the choice of one model or another from a set of possible ones is arbitrary. But the arbitrariness in choosing models or hypotheses is limited insofar as we rely on experimental findings. The limitations are imposed by *maximum simplicity of the model*, on the one hand, and the *necessity of its experimental verification*. The *freedom of choice* lies within the bounds of these constraints.

The search for an answer to the question of life's origin on our planet is an incorrect inverse physical problem. Unfortunately, we can thus far observe the sole realization of the advent of life, i.e., life on Earth. The emergence of living matter being an accomplished fact, any historical reasoning is of a hypothetical character. One feature of incorrect problems is that all currently available 'evidence' has to be used to reconstruct how the process developed in time in the past. Such problems are highly susceptible to initial conditions that we do not know. Attempts to transform this inverse problem into a direct one have so far failed, and it continues to defy unambiguous solution.

A I Oparin [35], followed by S L Miller and H Urey [36, 37], tried to experimentally reconstruct the appearance of living matter on Earth by simulating its early environmental conditions. No unambiguous result was obtained. Nevertheless, if a successful result were even achieved, it could not serve in full measure as evidence of the fact that life on Earth was forming according to a scenario chosen by the authors of the experiment. It should be proved that the authors' scenario is the only possible, or at least most probable.

### Conclusion

As follows from the foregoing, further discussion concerning the mechanisms of the origin of life on Earth, while interesting, has no sense in the context of the present-day advancement of cosmophysics and biophysics. Nevertheless, transition from the model proposed by V I Klyatskin (Fig. 5a) to my model (Fig. 5b) is needed at least for explaining the available biological facts.

I am grateful to V I Klyatskin for interest in my work and to the reviewer for the proposal to expound the idea of biological evolution in terms of random graphs.

### References

1. Klyatskin V I *Usp. Fiz. Nauk* **182** 1235 (2012) [*Phys. Usp.* **55** 1152 (2012)]
2. Ivanitskii G R *Usp. Fiz. Nauk* **180** 337 (2010) [*Phys. Usp.* **53** 327 (2010)]
3. Zel'dovich Ya B et al. *Usp. Fiz. Nauk* **152** 3 (1987) [*Sov. Phys. Usp.* **30** 353 (1987)]
4. Davis P, Kenyon D H *Of Pandas and People: The Central Question of Biological Origins* 2nd ed. (Dallas, TX: Haughton Publ. Co., 1993)
5. Hoyle F, Wickramasinghe N C *Evolution from Space* (London: Dent, 1981)
6. Ivanitskii G R et al. *Biofizika* **30** 418 (1986)
7. Ivanitskii G R *Virazhi Zakonomernostei. Pravilo BIO — Sterzhen' Nauki* (Overbanks of Regularities. The BHS Rule — the Pivot of Science) (Moscow: Nauka, 2011)
8. Borisyuk G N, Borisyuk R M, Kazanovich Ya B, Ivanitskii G R *Usp. Fiz. Nauk* **172** 1189 (2002) [*Phys. Usp.* **45** 1073 (2002)]
9. Darwin Ch *On the Origin of Species by Means of Natural Selection, or the Preservation of Favoured Races in the Struggle for Life* 1st ed. (London: J. Murray, 1859)
10. Alon N, Spencer J H *The Probabilistic Method* (New York: Wiley, 2000) [Translated into Russian (Moscow: Binom. Laboratoriya Znaniy, 2007)]
11. Dorogovtsev S N, Mendes J F F *Adv. Phys.* **51** 1079 (2002)
12. Kolchin V F *Sluchainye Grafy* (Random Graphs) (Moscow: Fizmatlit, 2004)
13. Callaway D S et al. *Phys. Rev. Lett.* **85** 5468 (2000)
14. Bollobás B et al. *Random Struct. Algorithms* **18** 279 (2001)
15. Erdős P, Gallai T *Matematikai Lapok* **11** 264 (1960)
16. Pennock D M et al. *Proc. Natl. Acad. Sci. USA* **99** 5207 (2002)
17. Kumar R et al. "Extracting large-scale knowledge bases from the web", in *VLDB'99, Proc. of 25th Intern. Conf. on Very Large Data Bases, September 7–10, 1999, Edinburgh, Scotland, UK*; <http://www.vldb.org/conf/1999/P60.pdf>
18. Dangelchev Ch *Physica A* **338** 659 (2004)
19. Caldarelli G et al. *Phys. Rev. Lett.* **89** 258702 (2002)
20. Finean J B *Biological Ultrastructure* (New York: Academic Press, 1967) [Translated into Russian (Moscow: Mir, 1970)]
21. Bollobás B *Random Graphs* 2nd ed. (Cambridge: Cambridge Univ. Press, 2001)
22. Cohen R, Havlin S *Phys. Rev. Lett.* **90** 058701 (2003)
23. Dorogovtsev S N, Mendes J F F, Samukhin A N *Phys. Rev. Lett.* **85** 4633 (2000)
24. Gilbert E N *Ann. Math. Stat.* **30** 1141 (1959)
25. Erdős P, Rényi A *Publ. Math. Debrecen* **6** 290 (1959)
26. Barabási A-L, Albert R *Science* **286** 509 (1999)
27. Newman M E J *Contemp. Phys.* **46** (5) 323 (2005)
28. Merton R K *Science* **159** 56–63 (1968)
29. Yule G U *Phil. Trans. R. Soc. Lond. B* **213** 21–87 (1925)
30. Spirin A S *Paleontol. Zh.* (5) 11 (2007) [*Paleontological J.* **41** 481 (2007)]
31. Lamb M J *Biology of Ageing* (Glasgow: Blackie, 1977) [Translated into Russian (Moscow: Mir, 1980)]
32. Khesin R B *Nepostoyanstvo Genoma* (Genome Instability) (Moscow: Nauka, 1984)
33. Clarke J M, Smith J M J. *Genet.* **53** 172–180 (1955)
34. de Vries H *Die Mutationen und die Mutationsperioden bei der Entstehung der Arten* (Leipzig: Veit, 1901) [Translated into Russian: *Izbrannye Proizvedeniya* (Selected Works) (Moscow: Medgiz, 1932) p. 55–76]
35. Oparin A I *Vozniknovenie Zhizni na Zemle* (The Origin of Life) (Moscow–Leningrad: Biomedgiz, 1936) [Translated into English (New York: Dover Publ., 1953)]
36. Miller S L *Science* **117** 528 (1953); <http://www.issol.org/miller/miller1953.pdf>
37. Miller S L, Urey H C *Science* **130** 245 (1959)