The evolution seen from the angle of quantum physics

Dipl.-Phys. Dieter Drechsel

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1. Introduction

In previous publications [1,7] the author described the base rivalry in monotonous DNA sequences and their effect on the DNA repair mechanism.
According to this theory, many base building blocks compete for the occupancy of the newly released base site in the replication of monotonous DNA sequences in the elongation phase. This gives them more and more kinetic energy from replication position k to next position. Thus, there is a probability that a tautomeric base pair is formed behind the end of the monotonic sequence because of the tunneling effect. After its replication a different, irreparable base pair develops from the tautomeric base pair, when the rivalry - energy leads to a very strong hydrogen bond. This happens, however, by chance. In the following, we will describe the 3 phenomena: The tunnel probability (section 2), the probability for coming up of a high – energy – base building block (Elitist, section 3),and the combination of both phenomena (section 4). The result of these calculations is the equation (28). It is remarkable that follows from these calculations that the length of the monotonous sequences, and also the length of DNA increases itself in the course of evolution (section 5). (Read up all detailed computations in [7].)

2. Tunnel processes in biological hydrogen bonds

The rivalry energy in accord with the formula

$$T_k = k \times 0.975 \times 10^{-12} - 5.9086 \times 10^{-10}\ \eta \times 1/\varepsilon \quad \text{erg} \qquad \varepsilon = 1.11529 \qquad \eta = \text{cell viscosity} \tag{1}$$

arises [1],[7] While the energy in the quantum mechanic energy level n = 1 is -13,656 eV (ground state energy), the donor of the hydrogen bond receives because of base rivalry an energy which is not only over the energy level n = 2
(-3,414 eV), but extends into the potential field of hydrogen bond and thus provokes a tunnel passage.

2.1. The tunnel probability

How large is the probability of the tunnel passage of a proton through the potential wall? The calculation of the number of the protons passing through the potential wall in biological hydrogen bonds has been carried out for the first time by P. Ö. Löwdin [2], the calculation is carried out down to the last detail in [3]. The result is available for the areas I, II and III (before, within and behind the potential wall). The three wave equations of the proton are

$$\psi_I(x) = A_1 e^{ik_0 n_1 x} + B_1 e^{-ik_0 n_1 x}$$

$$\psi_{II}(x) = \frac{\alpha}{\sqrt{k_r(x)}} e^{+K} + \frac{\beta}{\sqrt{k_r(x)}} e^{-K}$$

$$\psi_{III}(x) = A_3 e^{ik_0 n_3 x}$$

with

$$k_0 n_1 = \frac{1}{\eta}\sqrt{2m(E - U_1)}$$

$$k_0 n_3 = \frac{1}{\eta}\sqrt{2m(E - U_3)}$$

$$\eta = \frac{h}{2\pi}$$

where h is Planck's constant, and m is the proton mass.

All constants in the equations $\psi_I, \psi_{II}, \psi_{III}$ can be calculated [7].

As a result, we need only the amplitude A_3 of the proton wave that comes through the wall and the amplitude A_1 of the proton wave approaching to the wall:

$$P = \frac{|A_3|^2}{|A_1|^2} = e^{-2K} = e^{-\frac{2}{\eta}\int_0^l \sqrt{2m(U-E)}dx} \qquad (2)$$

This is the probability of a single proton tunnelling through the wall. l is the width of the potential wall between the positions, where the tunnel energy E has its smallest level E_0, considering that the tunnelling takes place above of this level. When the temperature t is taken into account, in which the tunnel process is provoked, the "temperature-dependent tunnel-probability" is (3)

$$P_t = e^{-\frac{E}{k_B t}} \cdot P .$$

k_B is Boltzmann's constant. If the potential wall has the shape of a parabola, then

$$P_t = \exp\left[-\frac{U_2 - E}{k_B t t_0}(t - t_0) - \frac{U_2}{k_B t}\right] \qquad (4)$$

where U_2 = peak potential of the wall and the "characteristic temperature"

$$t_0 = \frac{h}{l \cdot \pi^2 k_B}\sqrt{\frac{U_0}{2m}} \qquad (5)$$

U_0 is the height of the wall; m is the proton mass. For the size of U_0, see [4], [5], [6].

$$E = -13.656\ eV + T_k$$

is the energy created by base rivalry up to the replication position k-1 when k means the mutation position.

2.2. The change in the tunnel probability due to temperature - and energy - change

We now consider two different tunnel processes. The first operation took place at a hydrogen bond where the potential wall peak value was U_{21}, and the second operation takes place at a hydrogen bond, where the potential wall peak value is U_{22}. In the first process, the energy E_1 operated on the donor at the temperature t_1. In the second process, the energy E_2 operates on the donor at temperature t_2.
For the operations 1 and 2 apply the equations for the tunnel probabilities:

Operation 1: $$P_1 = \exp\left[-\frac{U_{21}-E_1}{k_B t_1 t_{01}}(t_1 - t_{01}) - \frac{U_{21}}{k_B t_1}\right] \qquad (6)$$

and

Operation 2: $$P_2 = \exp\left[-\frac{U_{22}-E_2}{k_B t_2 t_{02}}(t_2 - t_{02}) - \frac{U_{22}}{k_B t_2}\right]. \qquad (7)$$

t_{01} = characteristic temperature of the hydrogen bond 1
t_{02} = characteristic temperature of the hydrogen bond 2
E_1 = total energy of a proton before tunnel process in the operation 1
E_2 = total energy of a proton before tunnel process in the operation 2

$$\frac{P_2}{P_1} = \exp\left\{+\frac{1}{k_B}\left[k'(t_{01}-t_{02}) - \frac{t_{02}E_1 - t_{01}(\Delta E_2 + E_1) - E_0(t_{02}-t_{01})}{t_{01}t_{02}} - \Delta_S\right]\right\}$$

With the abbreviations

$$k' = \frac{2m\pi^4 l^2 k_B^{\ 2}}{h^2} = 4.9738\cdot 10^{-6}\ eV\cdot grad^{-2}$$ *l*=width of the potential wall

$$k_B = 0.863\cdot 10^{-4}\ eV\cdot grad^{-1}$$ Boltzmann's constant

$$\Delta E_1 = E_1 - E_0 \qquad (8)$$

$$\Delta E_2 = E_2 - E_1$$

$$\Delta_C = \frac{E_2}{t_0} - \frac{E_1}{t_0} \qquad (9)$$

$$\Delta_s = \frac{E_2}{t_2} - \frac{E_1}{t_1} \qquad (9a)$$

$$\frac{P_2}{P_1} = \exp\left\{+\frac{1}{k_B}\left[k'(t_{01}-t_{02}) + \frac{\Delta E_2}{t_{02}} - \frac{\Delta E_1}{t_{01}}\frac{t_{02}-t_{01}}{t_{02}} - \Delta_S\right]\right\} \qquad (10)$$

Assuming that two consecutive tunnel operations always work on the *same type* of binding and therefore $t_{01}=t_{02}=t_0$, then

$$\left(\frac{P_2}{P_1}\right)_{t_{01}=t_{02}} = \exp\left\{\frac{1}{k_B}[\Delta_C - \Delta_S]\right\} \qquad (11)$$

This is the proportion of a second tunnel process probability to a first tunnel process probability where both processes take place at different energies and temperatures. In each case of tunnelling, a tautomeric base pair is created. After replication in each process a different base pair develops

from the tautomeric base pair which is inseparable, if a high rivalry energy led to an inseparable hydrogen bond. So the DNA repair mechanism is ineffective, and the base distribution changes irreparably in each of the two proceedings.
Let us now, for the present irrespective of energies and temperatures examine *statistically* the distribution changes in DNA- replicons. We shall come back to the equations (6) and (7) later in section 4.

3. The distribution of bases on the DNA during replication, and the chance of occurrence of high base rivalry energy

In this section, the distribution change is examined from the point of view of an observer which does not know the physical equations (6) and (7) but only knows that a monotonous sequence lengthening appears sometimes during replication. The observer calculates the prospects of a base component to reach that place where the lengthening occurs, provided that the ticket for that place during replication is decided by drawing lots.
During the replication of a certain DNA-segment, a distribution of all base components takes place which are produced in the cell onto the codogen matrix. This happens in accordance with the copy rule. In this distribution, some base components are exposed to the base rivalry (if they get to a monotonous sequence) but others not. Even fewer base components still have so much energy at the end of the base rivalry (that is, at the end of the monotonous sequence replication) that they provoke a tunnelling in the next replication position and can build an irreparable hydrogen bond because their donor energy is still over the quantum mechanical energy level n = 2.
It is assumed that *any given base component only accidentally* will possess the ability to reach and to maintain this high energy level. However, there will be one of all base components produced in the cell which best joins those qualities (to reach and to maintain the high energy) together in itself. We name this base component the *"elitist component". An elitist can arise when base building blocks clump together and thereby receive a large rotational energy, but in comparison, small translational energy.*
This section lists all the favourable and all possible distributions within a DNA-replicon. The favourable distributions are those in which the elitist component accidentally arises there where the base rivalry works. The proportion of the number of the favourable distributions to the number of all possible distributions is the appearance probability of the elitist component at this place where the base rivalry works during the DNA – replication.

3.1. Enumeration of all possible distributions

The 4 bases A, C, G, T are represented by the terms E, *S*, *X*, *Y*. *E* is the **e**xchanged base, which in case of a mutation process will be replaced with an irreparable mutation by the **s**ubstituting base *S*. *X* and *Y* are any bases which do not change in the distribution change.

For the purpose of simplification, we look at only one base type e.g. the base type *S* in fig. 1. In the case of fig. 1 the copy – instruction requires that in the first monotonous sequence two identical bases *S*,*S*, in the second "monotonous" sequence one base S, and in the third monotonous sequence three identical bases *S*,*S*,*S* must exist.

Origin base sequence:

X	*S*	*S*	*E*	*X*	*Y*	*X*	*Y*	*Y*	*E*	*S*	*E*	*Y*	*E*	*X*	*X*	*S*	*S*	*S*	*Y*	*Y*	*X*

Split base sequence:

	S	*S*								*S*						*S*	*S*	*S*			
X				*X*		*X*								*X*	*X*						*X*
			E						*E*		*E*		*E*								
					Y		*Y*	*Y*				*Y*							*Y*	*Y*	
					Y		*Y*	*Y*				*Y*							*Y*	*Y*	

Figure 1: base sequence split into sequences of equal bases.

How large is the number of possibilities to distribute itself as in fig. 1 (agreeing with the copy – instruction)? Because all S-bases belong to the same base type, each base of the one monotonous sequence can accidentally appear in another monotonous sequence of the same base type. The enumeration of all possible cases to distribute itself in the base type *S* as in fig. 1 results in

$$\frac{6!}{2!1!3!} = 60$$

It is important to note that the replication is an establishment of an unchanged copy, only that the base components of a large stock are distributed randomly, but still according to the copy rule.

Designating the total number of the bases *S* as s, the total number of the bases E as e, the total number of the bases *X* and *Y* as x and y respectively, and further the number of bases which are located in the single monotonous sequences as

$$s_1, s_2, s_3, ..., e_1, e_2, e_3, ..., x_1, x_2, x_3, ..., y_1, y_2, y_3, ...$$

(in fig. 1 is $s_1 = 2, s_2 = 1, s_3 = 3$),

then the enumeration of all possible distributions agreeing to the copy –instruction in fig. 1 results in

$$r_1 = \frac{s!}{s_1!s_2!s_3!...} \cdot \frac{e!}{e_1!e_2!e_3!...} \cdot \frac{x!}{x_1!x_2!x_3!...} \cdot \frac{y!}{y_1!y_2!y_3!...} \qquad (12)$$

and the number of all possible distributions in a sequence that is different from the 1. sequence only in the fact that

in a box E the base number decreased by one, but in the box *S* the base number was increased by one, is

$$r_2 = \frac{(s+1)!}{(s_1+1)!s_2!s_3!...} \cdot \frac{(e-1)!}{(e_1-1)!e_2!e_3!...} \cdot \frac{x!}{x_1!x_2!x_3!...} \cdot \frac{y!}{y_1!y_2!y_3!...} \qquad (13)$$

With both equations, the number of all accidental possible distributions which agree with the copy – instruction is written down. All these distributions can appear during replication.

3.2. Enumeration of all favourable distributions, and the chance of occurrence of high base rivalry energy

Now we wish to know how often an elitist component appears in all these r distributions within a certain monotonous sequence.

We designate the number of all favourable distributions (that is the number of all cases in which the elitist accidentally appears within a monotonous sequence with the length s_1) as σ_{s1} and the number of cases in which the elitist accidentally appears within a monotonous sequence with the length s_1+1 as σ_{s1+1}.

The σ can be calculated as follows: From the above sequence one sees that in the total number of s=6 of replicating base components only s-1=5 are permutated, since the elitist should always remain in the monotonous sequence. The number of components in the three-digit (s_1=3) monotonous sequence are permuted, is s_1-1. The number of remaining base components which permute itself, and which are located somewhere in the replicating replicon, is s-s_1=6-3=3. Thus follows for the number of favorable distributions at the first operation:

$$\sigma_{s1} = \frac{(s-1)!}{(s_1-1)!(s-s_1)!} \qquad (14a)$$

and for the second operation, because there s has increased itself to s+1 and also s_1 has increased itself to s_1+1

$$\sigma_{s1+1} = \frac{s!}{s_1![s+1-(s_1+1)]!} = \frac{s!}{s_1!(s-s_1)!} \qquad (14b)$$

$$\sigma = \frac{\sigma_{s1+1}}{\sigma_{s1}} = \frac{s!}{s_1!(s-s_1)!} \cdot \frac{(s_1-1)!(s-s_1)!}{(s-1)!} = \frac{s}{s_1} \quad , \qquad (14c)$$

The *probability* that an elitist component appears in an s_1-digit monotonous sequence during replication (of whole repicon) is

$$W_{s1} = \frac{\sigma_{s1} \cdot r_e \cdot r_x \cdot r_y}{r_1} \qquad (15)$$

where

$$r_e = \frac{e!}{e_1! \cdot e_2! \cdot \ldots}$$

$$r_x = \frac{x!}{x_1! \cdot x_2! \cdot \ldots}$$

$$r_y = \frac{y!}{y_1! \cdot y_2! \cdot \ldots}$$

are the numbers of all possible e-, x-, y- distributions and r_1 is the number of all possible distributions. This is because that the convenient case (that means, +the elitist is within the s_1-digit monotonous sequence) also can appear in each of the e-, x-, y-distributions. The statistical propability that an elitist component appears in an (s_1+1)-digit monotonous sequence during replication is

$$W_{s1+1} = \frac{\sigma_{s1+1} \cdot r_{e-1} \cdot r_x \cdot r_y}{r_2} \qquad (16)$$

$$r_{e-1} = \frac{(e-1)!}{(e_1 - 1)! \cdot e_2! \cdot \ldots}$$

$$\frac{r_e}{r_{e-1}} = \frac{e}{e_1} \qquad (17)+$$

$$\frac{W_{s1+1}}{W_{s1}} = \frac{\sigma_{s1+1}}{\sigma_{s1}} \frac{r_{e-1} \cdot r_1}{r_e \cdot r_2} \qquad (18)$$

Defining the fraction $\frac{\sigma_{s1+1}}{\sigma_{s1}}$ as σ:

$$\frac{W_{s1+1}}{W_{s1}} = \sigma \frac{r_{e-1} \cdot r_1}{r_e \cdot r_2} \qquad (19)$$

Dividing equation (12) through equation (13) results in

$$\frac{r_1}{r_2} = \frac{e}{s+1} \cdot \frac{s_1 + 1}{e_1} \qquad (20)$$

By inserting (17) into (19):

$$\frac{W_{s1+1}}{W_{s1}} = \sigma \cdot \frac{s_1 + 1}{s+1} \qquad (21)$$

s is the total number of the substituting base type *S*. s_1 is the base number in the monotonous sequence which is lengthened in case of mutation. σ depends on the length of the replicon. Now we have calculated the probability for a distribution, in which the elitist appears there where the base-rivalry energy becomes highest (in the monotonous sequence sss . . .). On condition that at the end of this monotonous sequence exists a tautomeric base pair then an irreparable mutation develops itself.

4. The total probability of mutations caused by base rivalries in geological periods

Now it seems to be interesting to combine both tunnel probability and elitist probability.
Two possible processes can occur during replication of a monotonous sequence:

1. The elitist (for example, dGTP) comes (with the probability W_{s1}) into the monotonous sequence GGGGA.
It is now assumed that at cell temperature t_1 the cell viscosity is just so high that after s_1=4 replication positions the elitist dGTP reaches the base rivalry energy Ts_1 =3.818 eV.

2. We consider the sequence as an example (now are again the real bases Cytosin,…):+

$$\text{Re}\,pl.\,dir. \rightarrow \frac{C\,C\,C\,C\,T\,T}{G\,G\,G\,G\,A\,A}$$

The elitist is a base building block dGTP, which has obtained the energy Ts_1 = 3.818 eV by base rivalry up to the replication position s_1 = 4. Its total energy is

$$E = T_{s1} - \frac{13.656}{2^2} = 3.818 - 3.414 = 0.404\,eV$$

if the energy required for n = 2 is taken into account. This energy E is sufficient to provoke a tunneling process.
Because of its high rotation energy and because of its low translation energy, the elitist does not take the place of the 4. position but further is an independent base building block.[1] However in the next (5.) replication position, so in the elongation phase of the just replicating base pair T/A, the high elitist-energy will provoke a tunneling process and thus the formation of the tautomeric base pair T*/A* and, again because of its high energy, immediately there after, the base A* is replaced by the elitist base G so that the new base pair T*/G develops. The high energy of the elitist causes the binding proton of T*/G to reach the quantum mechanical energy level n = 2, so the lower curve in figure 2 applies. This strong bond can not be resolved because the elitist can retain his acquired base rivalry energy longer than the DNA repair mechanism works.

[1] The translation energy of all other dGTP is higher than the transl. energy of the elitist and therefore, they occupy all positions of the monot. sequence.

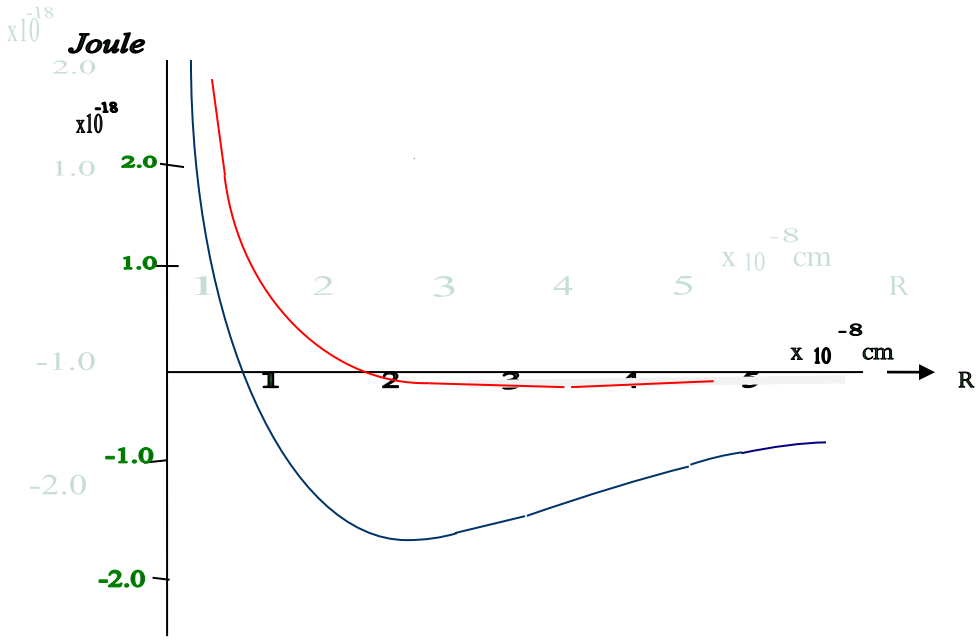

Fig 2: Hydrogen bond energy (between one base pair)
Upper graph: Both partners in ground state (n=1)
Under graph: Acceptor in ground state (n=1), donor (excited by base rivalry, n=2). Graphs calculated in [7].

The subsequent replication then arises

$$from\frac{T^*}{G}the"total\ false base\ pair"\frac{C}{G}$$

Thus, the irreparable mutation has taken place.
So is the total probability for an irreparable mutation (caused by base rivalry) at the end of an s_1-digit monotonous sequence the product of tunnel-probability and of the probability of the one arriving elitist:

$$\varpi = P_1 \cdot W_{s1} \qquad (22)$$

and then the monotonous sequence is extended to s_1+1.
In case of second mutation event (tunnel probability P_2, elitist probability W_{s1+1}) is the total probability

$$\varpi = P_2 \cdot W_{s1+1} \qquad (22a)$$

and then the monotonous sequence is extented to s_1+2.
Therefore, it is

$$\frac{P_2}{P_1} = \frac{W_{s1}}{W_{s1+1}} \qquad (23)$$

and from eq.(11) and from eq. (21) follows

$$\exp\left\{\frac{1}{k_B}\left[\Delta_C - \Delta_S\right]\right\} = \frac{1}{\sigma}\cdot\frac{s+1}{s_1+1} \qquad (24)$$

(with $\Delta_C = \frac{E_2}{t_0} - \frac{E_1}{t_0}$, $\Delta_S = \frac{E_2}{t_2} - \frac{E_1}{t_1}$)

$$\left[\Delta_S - \Delta_C\right] + k_B \ln\left[\frac{1}{\sigma}\cdot\frac{s+1}{s_1+1}\right] = 0 \qquad (25)$$

Equation (25) describes the change of temperature and energy necessary to provoke the lengthening of a monotonous sequence for two positions to s_1+1+1.

Considering equation (14c) then

$$[\Delta_c - \Delta s] - k_B \ln\left[\frac{s_1}{s} \cdot \frac{s+1}{s_1+1}\right] = 0 \qquad (26)$$

In the equation's left side there is an entropy change

$$\Delta_C = \frac{E_2}{t_0} - \frac{E_1}{t_0}$$

which relates to the characteristic temperature t_0, and an entropy change

$$+\Delta_S = \frac{E_2}{t_2} - \frac{E_1}{t_1}$$

E_1 and E_2 are the energies relating to the temperatures in each case provoking the tunnel process 1 or 2, respectively.

s_1 is the previous length from the monotonous sequence which is extended by two positions. s is the previous total number of substituting bases. Equation (26) can be written as

$$[\Delta_S - \Delta_C] + k_B \ln\left[\frac{s_1}{s} \cdot \frac{s+1}{s_1+1}\right] = 0 \qquad (27)$$

If there are two tunnel proceedings, where $E_1 = E_2 = E$ then

$$E\left(\frac{1}{t_2} - \frac{1}{t_1}\right) + k_B \ln\left[\frac{s_1}{s} \cdot \frac{s+1}{s_1+1}\right] = 0 \qquad (27a)$$

5. Evolution physics

It seems to be justified, these calculations to transfer to the theory of evolution. In the history of the earth are happened many temperature changes. Warm and cold periods alternated. Especially the transition from a warm to a cold period has created higher forms of species. Assuming the temperature of a first period is t_1, and in a following period the temperature is t_2. $t_2 < t_1$. $\tau = t_1 - t_2$ is the temperature difference between the two periods which has caused the cell viscosity to increase up to $\Delta\eta = 0.18404 \times 10^{-3}$ Pa s and so always provokes the lengthening of a monotonous sequence for one position in the case of base rivalry [7].

These mutations will always lead to a change of distribution of DNA. So it is conceivable that in early warm periods only plain forms of DNA have existed with very short (monotonous) sequences. In some individuals these short (monotonous) sequences must have lengthened themselves through temperature decrease considering of equation (27a) because the cytoplasm viscosity has enlarged itself. Thus, with change from warmer down to always colder periods

(caused by slowly cooling of earth) the distribution within DNAs has changed itself, so that always more longer monotonous sequences have developed themselves.

We want to examine this problem in more detail:

The equation

$$\frac{E \cdot \tau}{t_1^2 - \tau \cdot t_1} + k_B \ln\left[\frac{s_1}{s} \cdot \frac{s+1}{s_1+1}\right] = 0 \qquad (28)$$

(calculated from eq.(27a)was derived on condition that every temperature reduction τ enlarges the viscosity of the cell plasma by $\Delta\eta = 0.18404 \text{ x } 10^{-3}$ Pa s and too, that each such temperature reduction provokes an extension of an s_1-digit monotonous sequence exactly by 1 position during replication. The base rivalry energy T_k thus remains constantly during every extension of the s_1-digit monotonous sequence.

From the eq. (28) the s can be calculated:

$$s = \frac{\beta}{\frac{s_1+1}{s_1} - \beta} \qquad (29)$$

with $\beta = e^{\frac{1}{k_B} \cdot \frac{E \cdot \tau}{t_1^2 - t_1 \cdot \tau}}$

with k_B = Boltzmann constant = 0.8631 x 10^{-4} eV/grad (The temperature t_1 is entered here in Kelvin).

It can be seen that for every t_1 and τ a certain s belongs. In particular, it can be seen that with decreasing cell temperature t_1, the base number s becomes ever larger.

As long as the cell temperature t_1 does not decrease, the s (relevant for the size of the replication unit) remains the same. If the cell temperature t_1 decreases, then the equation (29) requires a larger value for s, and this means, if is expected a new irreparable extension of the monotonic sequence, a larger distribution quantity s, i.e. a larger replication unit is necessary. It follows that if the t_1 is reduced (long enough), i.e. in a subsequent epoch, the replication unit must increase in order to provoke a new irreparable extension of the monotone sequence. Thus equation (29) can also be calculated for several processes at different temperatures t_1,where the high temperatures of very early geothermal ages and the small monotonous sequence lengths s_1 correspond to the simplest organisms. We

proceed by assigning certain s_1 to specific cell temperatures t_1 and using eq.(29) to determine the s. The calculated numbers s of the substituting bases prove that there are organisms that have responded to temperature reductions as described in our calculations.

In the following table are calculated [from equation (29)]the temperature – dependent base lengths and the monotonous sequence lengths, as they arise in the different evolution epochs.

τ^0	s_1	s_1+1	s	$t_1{}^0C$	$\eta[Pa\times s]$	E [eV]	n
1.85	6	7	12	66			
1.74	8	9	21	62.3	0.319×10^{-3}	0.405	2
1.7	10	11	42	58.82	0.687×10^{-3}	0.405	2
1.7	12	13	151	55.42	1.055×10^{-3}	0.405	2
1.5	14	15	346	52.02	1.423×10^{-3}	0.405	2
1.3	16	17	436	49.02	1.792×10^{-3}	0.402	2
1.15	18	19	608	46.42	2.16×10^{-3}	0.405	2
1.025	20	21	713	44.12	2.528×10^{-3}	0.404	2
0.93	22	23	1101	42.07	2.896×10^{-3}	0.404	2
0.85	24	25	1794	40.21	3.264×10^{-3}	0.403	2
0.78	26	27	2532	38.51	3.632×10^{-3}	0.404	2
0.72	28	29	3552	36.97	4×10^{-3}	0.404	2

$$E = T_{s1} - 13.656/n^2$$

The first part of the equation is the base rivalry energy normalized to eV, the second term $\frac{13.656}{2^2}$ is the energy which after base rivalry up to position s_1 excites the binding proton in the hydrogen bond to the n = 2 level. The difference is the tunnel energy, which at the position s_1 + 1 provokes a tunneling process and thus a new distribution s_1 + 1, s + 1.
The quantum number n must be n = 2 for each mutation process. This is achieved because of base rivalry at s_1 and t_1.
s is the number of substituting bases necessary for the elitist occurrence probability during an irreparable process in a replication unit

(= replicon); the total size is four times because of the four base types. By constantly enlarging a replication unit, it divides into two parts several times. If 18 doubles occur in the course of time, this results in the hom. sap. 2^{18} x 3552 x 4 = 3.72 x 10^9 bp.

It is always $E \approx +0.4eV$, as can be seen by the substitution of the different cell viscosities z into the base rivalry equation (30).

The energy level n = 2 is because the equation (28) was derived on the assumption that at the position where this equation holds, the energy level n = 2 has been reached [7].

We find out *that s increases quite strongly* when s_1 is lengthened for only two positions.

So statistically a two-position-lengthening of the monotonous sequence happens at the cell temperatures t_1 and t_1-τ, if are located s S-base components in the distribution unit.

Such distributions, which have a smaller s_1 and higher cell temperatures, belong to more primitive organisms which have smaller replication units and smaller lengthes of DNA.

Since the table corresponds to the successive lowering of the ambient temperature on the earth, it follows from this the *successive extension of monotonous seqences and replication units and the DNA in the course of earth development and cooling.*

6. Conclusion

In this work, the author tried to find a mathematical connection between the cytoplasm temperature reduction and the DNA distribution change within long evolution periods.

From the equation (28) it follows that *in the course of the evolution,* the gradual reduction of the cytoplasm temperature in the cases of base rivalry resulted in a strong lengthening of monotonic sequences and in a strong increase of the total number of bases. Therefore has led to a big lengthening of the DNA.

Thus, the calculations show that the very long DNAs of the highly organized organisms did not originate from Darwinian constraints,[2] but only to satisfy equation (23), since this equation is the basis of the above calculations. So you just have to assume the validity of this equation however, it can easily be justified (see [8] page 10).

[2] In any case, only 1% of all base pairs are used for protein encoding

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