Evolution Physics (second revised edition)

Dipl.- Phys. Dieter Drechsel

1. Introduction

In a previous publication [1] the author described the base rivalry in monotonous DNA sequences and their effect on the DNA repair mechanism. As described in the article, during the monotonous sequence replication, energies appear theoretically to increase with a progressive replication fork up to the quantum mechanical energy level n=2 because of the base rivalry, and these rivalry energies affect the bond strength between the complementary bases. If there is a tautomeric base pair in the replication position where the rivalry energy is large enough, then in this position an irreparable mutation will occur, since the DNA repair mechanism cannot repair that error because too much binding energy.
Thus a mutation (caused by base rivalry) can occur only on condition that a transition of a base pair into its tautomeric form is happened. It is remarkable that this transition likewise can occur by the effect of base rivalry energy.
The base rivalry - energy which has an effect on a normal base pair provokes a tunnel process in its hydrogen bond, and produces the tautomeric form. After whose replication a different, irreparable base pair develops from the tautomeric base pair, when the rivalry - energy leads into a very strong hydrogen bond. This happens, however, by chance and in the following we will compute the probabilities of such accidental events. The result of these calculations is the equation (32) which could be useful for the theory of evolution and besides for clearing up of virus mutations. It is remarkable that follows from these calculations that the length of DNA increases itself in the course of evolution (section 7).

This second revised version differs from the previous version, because in section 7 the quantum number n = 2 is assumed for the calculation of the base rivalry energy instead of n = 1.

2. The problems

The replication of large DNAs takes place by simultaneous replications of so called replicons with a length of more than 10^4 bp. This happens continuously 5′ -> 3′ on the leading strand and discontinuously on the lagging strand in segments of 100 bp (eucariotic DNA) in the direction 3′ -> 5′. These segments (Okazaki-fragments) are replication units. When the replication of the whole replicon has finished, remains for the present on the lagging strand a small rest (s. fig. 1) which only after the last Okazaki - fragment is replicated. The length of this rest called “residual fragment” may be 0 … 100 bp (eucariotic DNA), and this is also a replication unit

because it has a replication beginning and a replication end. For the first understanding it is useful to explain the problems with such short residual fragment:

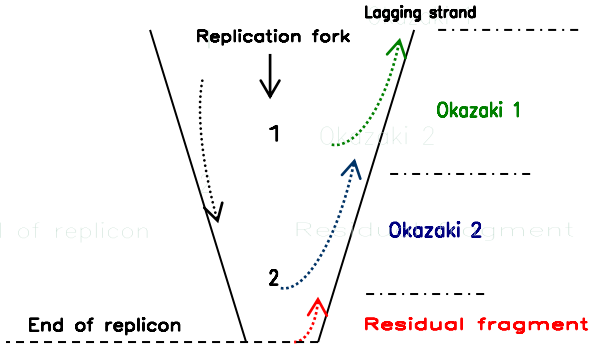

Fig. 1: Schematic specification of replication at end of replicon

We assume that in this residual fragment a long monotonous sequence GGGGGGATTA develops in 5'->3' on lagging strand during replication. So, high base rivalry energy comes into being.
To make this clear, let us look to fig 2 where only the small rest of replicon, the residual fragment is shown: One sees the base pair A - T at position 7 (fig. 2a). If the base rivalry energy is large enough (caused by replication of the long monotonous sequence GGG …) then it provokes a tunnel process transforming the base pair A – T into its tautomeric base pair A* - T* (fig. 2b).
(The transformation towards the tautomeric form means that a proton towards the other side of the hydrogen bond through this had tunnelled.)
Soon after that (almost at the same time), a base component dGTP, having the high base rivalry energy replaces the base A* so that the new base pair G – T* comes into being (fig.2c). This cannot be repaired because of the high binding energy which has arised between the two bases G and T*. During the replication after next, the base pair G-T* (which possesses an unusual geometry) will be transferred into the "perfect

wrong" base pair G-C, so that the irreparable mutation is perfect, and the monotonous sequence has lengthened itself. Thus an irreparable mutation arises.
It seems to be interesting, to combine both proceedings: There is the distribution process which generates the high base rivalry energy, and then also is the tunnel process which produces the tautomeric base pair. These problems will be examined in section 5.

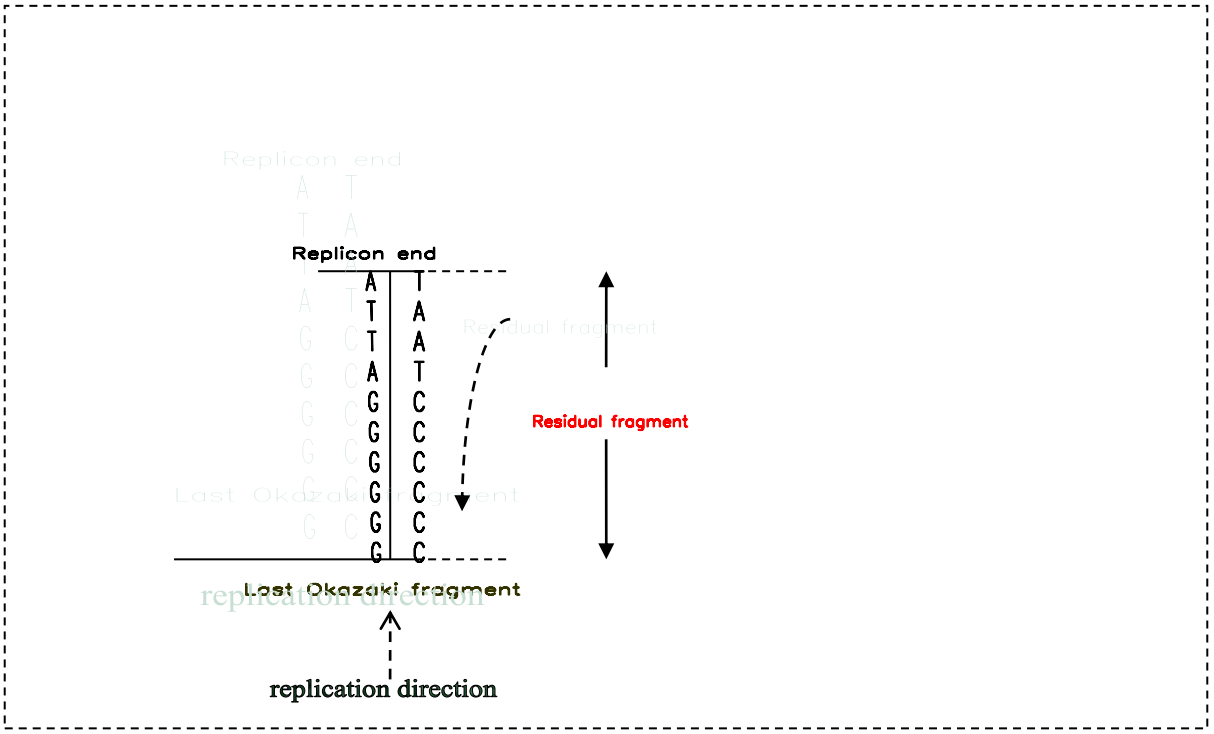

Fig. 2a: Replicating residual fragment before tunneling

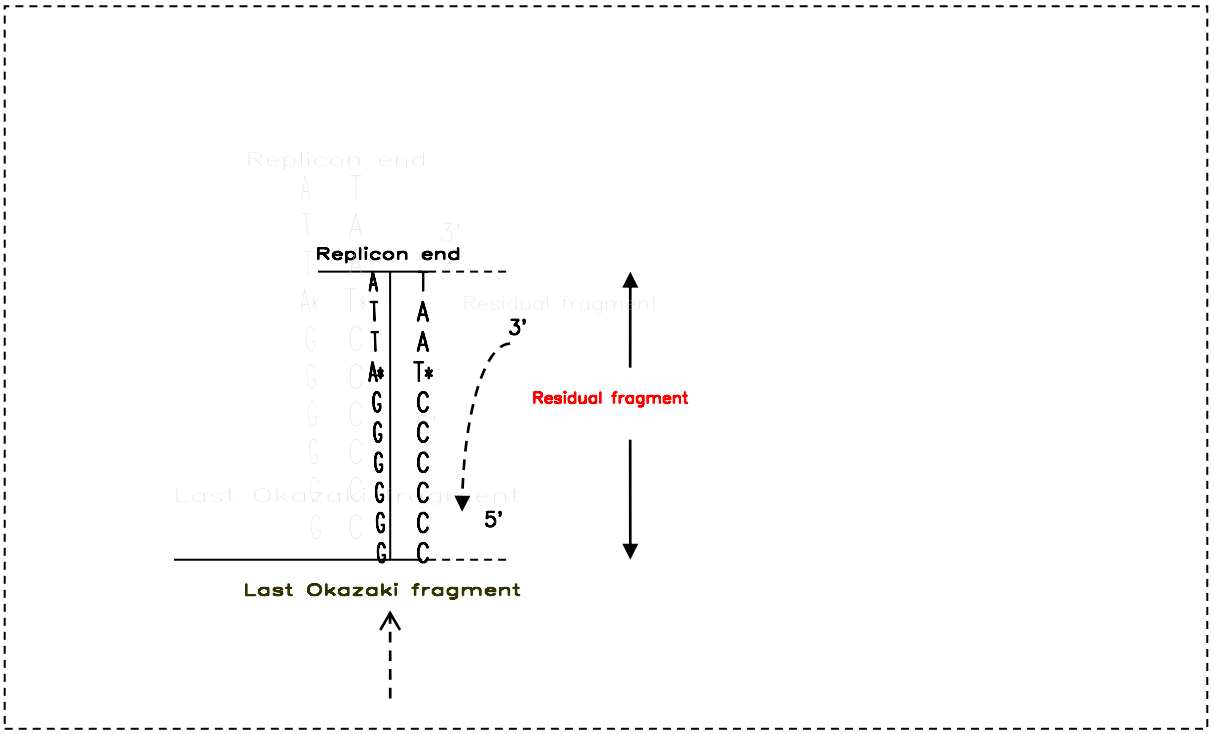

Fig 2b: Replicating residual fragment under tunnel influence

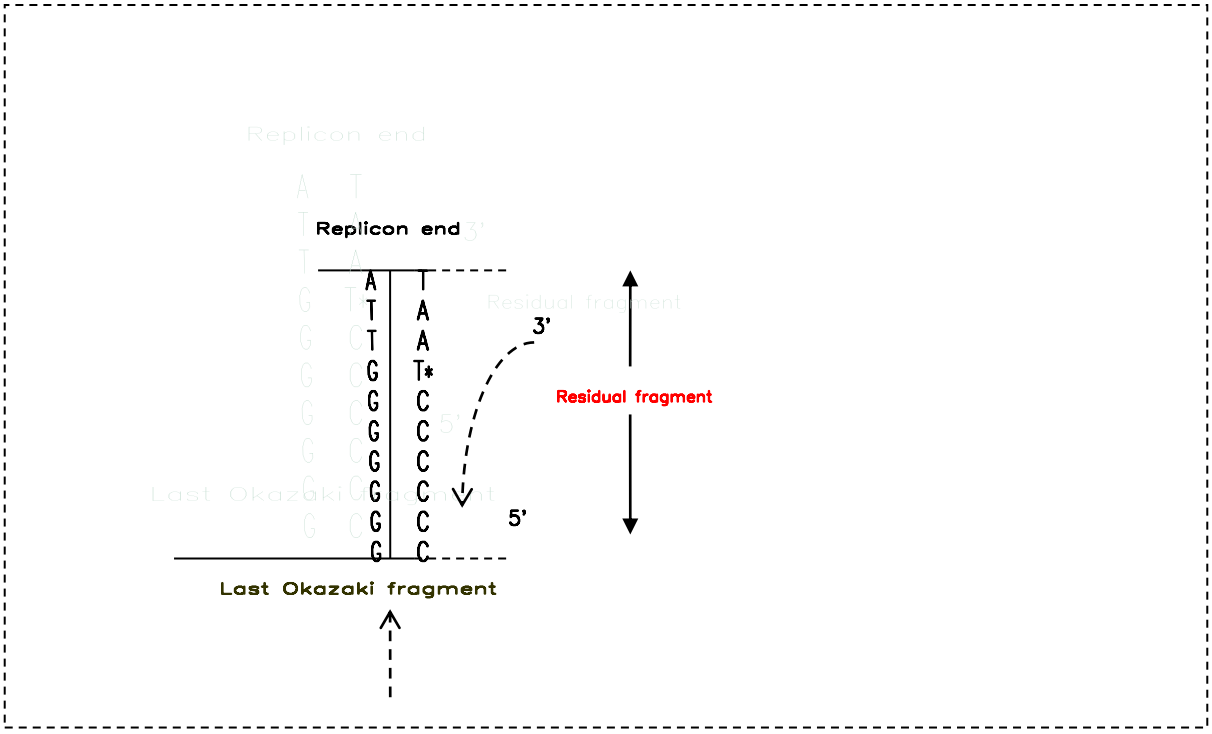

Fig. 2c: Replicating residual fragment after tunnelling; base A* is replaced by the high-energy-base G

In the following, we will describe the 3 phenomena: The tunnel probability (section 3), the distribution probability (section 4), and the combination of both phenomena (section 5).

3. Tunnel processes in biological hydrogen bonds

Figure 3 shows the participating energies, energy of the donor proton and potential energy of the potential wall in a hydrogen bond.

The energy E_{k+1}=(ground state energy)+T_k required for the provocation of the tunnel process is composed of the ground state energy -13.656 eV and the rivalry energy T_k which is created in fig. 2 up to the replication position 6. Therefore, the length of the new monotonous sequence is one position larger than the position k, where the rivalry energy in accord with the formula

$$T_k = k\left(\frac{e_p^2}{r_1} - \frac{e_p^2}{r_B}\right) - z\cdot\sqrt{\frac{e_p^2}{r_1} - \frac{e_p^2}{r_B}} \qquad (1)$$

arises (see [1]). (e_p = partialcharge, r_1 = distance between the related complementary bases, r_B = distance between successive bases, z is specific value based on the viscosity of the nucleoplasm.

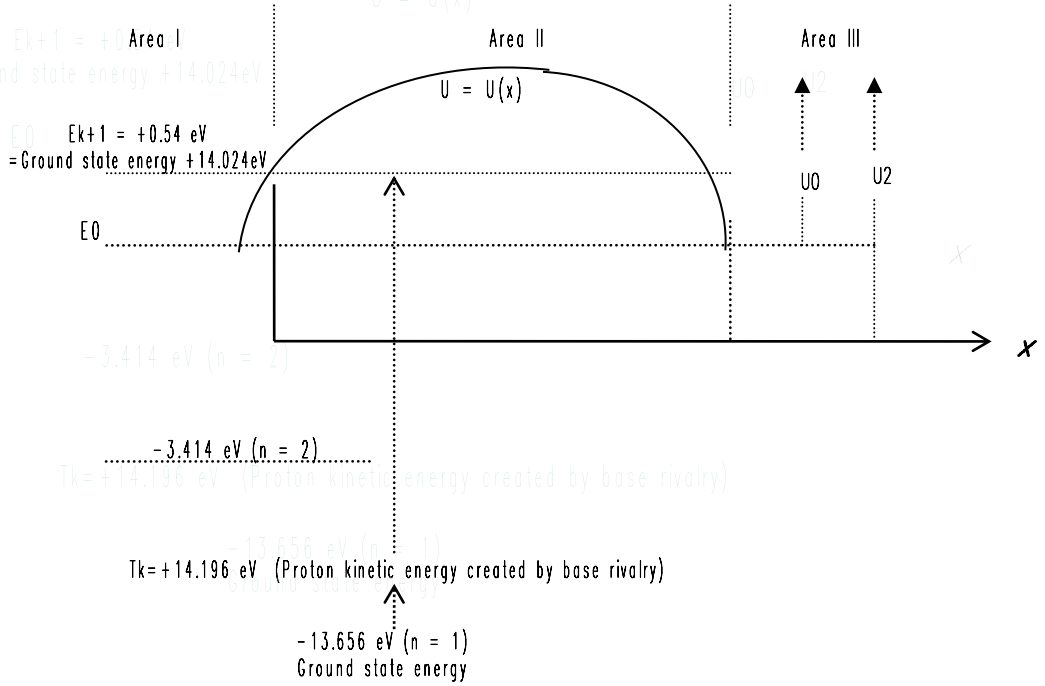

Figure 3 shows the energy levels, which the donor proton of a hydrogen bond passes because of the base rivalry, and the energies of the potential wall of the hydrogen bond:

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 the energy T_K, which is, for example +14,196 eV. This gives the donor proton the total energy -13,656 +14,196 = + 0,54 eV and reaches an energy that 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.

3.1. 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. Considering the boundary conditions

$$\psi_I(0) = \psi_{II}(0)$$

$$\psi_I'(0) = \psi_{II}'(0)$$

$$\psi_{II}(l) = \psi_{III}(l)$$

$$\psi_{II}'(l) = \psi_{III}'(l)$$

all the constants in the equations $\psi_I, \psi_{II}, \psi_{III}$ can be calculated.

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

$$P_t = e^{-\frac{E}{k_B t}} \cdot P \; . \qquad (3)$$

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].

$$E = -13.656 \text{ eV} + T_{k-1}$$

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

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

E_0 = Smallest tunnel energy in the computation
U_{21} = vertex of the potential of the hydrogen bond 1
U_{22} = vertex of the potential of the hydrogen bond 2
t_{01} = characteristic temperature of the hydrogen bond 1
t_{02} = characteristic temperature of the hydrogen bond 2
t_1 = temperature during Operation 1
t_2 = temperature during operation 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

$$k' = \frac{2m\pi^4 l^2 k_B{}^2}{h^2} = 4.9738 \cdot 10^{-6}\ eV \cdot grad^{-2}$$

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

The ratio of the tunnel probabilities is

$$\frac{P_2}{P_1} = \exp\left\{ -\frac{U_{22} - E_2}{k_B t_2 t_{02}} (t_2 - t_{02}) - \frac{U_{22}}{k_B t_2} + \frac{U_{21} - E_1}{k_B t_1 t_{01}} (t_1 - t_{01}) + \frac{U_{21}}{k_B t_1} \right\}$$

$$= \exp\left\{ \frac{1}{k_B t_1} \left[(U_{21} - E_1) \frac{t_1 - t_{01}}{t_{01}} + U_{21} \right] - \frac{1}{k_B t_2} \left[(U_{22} - E_2) \frac{t_2 - t_{02}}{t_{02}} + U_{22} \right] \right\}$$

$$= \exp\left\{ \frac{1}{k_B t_1} \left[U_{21} \cdot \frac{t_1}{t_{01}} - E_1 \left(\frac{t_1}{t_{01}} - 1 \right) \right] - \frac{1}{k_B t_2} \left[U_{22} \frac{t_2}{t_{02}} - E_2 \left(\frac{t_2}{t_{02}} - 1 \right) \right] \right\}$$

$$= \exp\left\{ \frac{1}{k_B\, t_1}\left[U_{21} \cdot \frac{t_1}{t_{01}} - E_1 \frac{t_1}{t_{01}} + E_1 \right] - \frac{1}{k_B\, t_2}\left[U_{22} \frac{t_2}{t_{02}} - E_2 \frac{t_2}{t_{02}} + E_2 \right] \right\}$$

$$\frac{P_2}{P_1} = \exp\left\{ \frac{1}{k_B\, t_1}\left[U_{21} \cdot \frac{t_1}{t_{01}} - E_1 \frac{t_1}{t_{01}} \right] + \frac{E_1}{k_B\, t_1} - \frac{1}{k_B\, t_2}\left[U_{22} \frac{t_2}{t_{02}} - E_2 \frac{t_2}{t_{02}} \right] - \frac{E_2}{k_B\, t_2} \right\} \qquad (8)$$

Using the abbreviation

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

one gets

$$\frac{P_2}{P_1} = \exp\left\{ \frac{1}{k_B\, t_1}\left[U_{21} \cdot \frac{t_1}{t_{01}} - E_1 \frac{t_1}{t_{01}} \right] - \frac{1}{k_B\, t_2}\left[U_{22} \frac{t_2}{t_{02}} - E_2 \frac{t_2}{t_{02}} \right] - \frac{1}{k_B} \Delta_S \right\}$$

$$\frac{P_2}{P_1} = \exp\left\{ -\frac{1}{k_B\, t_{02}}\left[U_{22} - E_2 \right] + \frac{1}{k_B\, t_{01}}\left[U_{21} - E_1 \right] - \frac{1}{k_B} \Delta_S \right\} \qquad (10)$$

According to fig. 3:

$$U_{22} = U_{02} + E_0$$
$$U_{21} = U_{01} + E_0 \qquad (11)$$

$$\frac{P_2}{P_1} = \exp\left\{ -\frac{1}{k_B\, t_{02}}\left[U_{02} + E_0 - E_2 \right] + \frac{1}{k_B\, t_{01}}\left[U_{01} + E_0 - E_1 \right] - \frac{1}{k_B} \Delta_S \right\} \qquad (12)$$

From eq.(5):

$$U_{01,02} = k' t_{01,02}{}^2$$

$$\frac{P_2}{P_1} = \exp\left\{ -\frac{1}{k_B\, t_{02}}\left[k' t_{02}^2 + E_0 - E_2 \right] + \frac{1}{k_B\, t_{01}}\left[k' t_{01}^2 + E_0 - E_1 \right] - \frac{1}{k_B} \Delta_S \right\}$$

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

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

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

With the abbreviations

$$\Delta E_1 = E_1 - E_0$$
$$\Delta E_2 = E_2 - E_1 \qquad (13)$$

$$\Delta_C = \frac{E_2}{t_0} - \frac{E_1}{t_0} \qquad (14)$$

resulting in

$$\frac{P_2}{P_1} = \exp\left\{ + \frac{1}{k_B}\left[k'\left(t_{01} - t_{02} \right) - E_1 \frac{t_{02} - t_{01}}{t_{01}\, t_{02}} + \frac{\Delta E_2}{t_{02}} + E_0 \frac{t_{02} - t_{01}}{t_{01}\, t_{02}} - \Delta_S \right] \right\} \qquad (15)$$

$$\frac{P_2}{P_1} = \exp\left\{+\frac{1}{k_B}\left[k'(t_{01}-t_{02})+\frac{\Delta E_2}{t_{02}}-\frac{\Delta E_1}{t_{01}}\frac{t_{02}-t_{01}}{t_{02}}-\Delta_S\right]\right\} \qquad (16)$$

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

$$\left(\frac{P_2}{P_1}\right)_{t_{01}=t_{02}} = \exp\left\{\frac{1}{k_B}\left[\Delta_C-\Delta_S\right]\right\} \qquad (17)$$

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-fragments. We shall come back to the equations (16) and (17) later in section 5.

4. 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. Those produced base components which are exposed to the base rivalry respond in very different ways: Most of them lose their obtained energy owing to deviation, friction or owing to short fading times. Only a few are scarcely deflected, or have long fading times. 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 a high energy level because exact properties of a base component produced in the cell cannot be identified. However, there will be one of all base components produced in the cell which best joins those qualities (to reach and to maintain a high energy) together in itself. We name this base component the *"elitist component".*
This section lists all the favourable and all possible distributions within the certain DNA-segment. 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.

4.1. Enumeration of all possible distributions
The 4 bases A, C, G, T are represented by the terms *C, S, X, Y. C* is the **c**oncerned 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. 4. In the case of fig. 4 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*	*C*	*X*	*Y*	*X*	*Y*	*Y*	*C*	*S*	*C*	*Y*	*C*	*X*	*X*	*S*	*S*	*S*	*Y*	*Y*	*X*

Split base sequence:

	S	*S*								*S*						*S*	*S*	*S*			
X				*X*		*X*								*X*	*X*						*X*
			C						*C*		*C*		*C*								
					Y		*Y*	*Y*				*Y*							*Y*	*Y*	

Figure 4: base sequence split into sequences of equal bases.

How large is the number of possibilities to distribute itself as in fig 4 (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. 4 results in

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

This is the same as in a classroom with 10 students, where by the teacher's direction the students distribute themselves on 2 benches containing four students each and 1 bench containing only two students. How the students arrange themselves is irrelevant to the teacher. There are $\frac{10!}{4!\cdot 4!\cdot 2!}=3150$ various outcomes for the way the teacher has directed the distribution.

In a group of 7 students which are to distribute to 1 bench with 4 students and 1 bench with 3 students, there are 7!/4!/3! = 35 possibilities.

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 *C* as c, 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

$s_1, s_2, s_3, ..., c_1, c_2, c_3, ..., x_1, x_2, x_3, ..., y_1, y_2, y_3, ...$

(in fig. 4 is $s_1=2, s_2=1, s_3=3$),

then the enumeration of all possible distributions agreeing to the copy –instruction in fig. 4 results in

$$r_1=\frac{s!}{s_1!s_2!s_3!...}\cdot\frac{c!}{c_1!c_2!c_3!...}\cdot\frac{x!}{x_1!x_2!x_3!...}\cdot\frac{y!}{y_1!y_2!y_3!...} \qquad (18)$$

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

$$r_2=\frac{(s+1)!}{(s_1+1)!s_2!s_3!...}\cdot\frac{(c-1)!}{(c_1-1)!c_2!c_3!...}\cdot\frac{x!}{x_1!x_2!x_3!...}\cdot\frac{y!}{y_1!y_2!y_3!...} \qquad (19)$$

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.

4.2. 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.

To make this clear we take once again the second example of the students: We ask how often one of the 7 students (the "elitist component") in 35 distributions will sit in the bench

with four seats. This student may have the number 1. There are the following versions:

```
1 2 3 4              1 4 5 6
1 2 3 5              1 4 5 7
1 2 3 6              1 4 6 7
1 2 3 7
1 2 4 5              1 5 6 7
1 2 4 6
1 2 4 7
1 2 5 6
1 2 5 7
1 2 6 7

1 3 4 5
1 3 4 6
1 3 4 7
1 3 5 6
1 3 5 7
1 3 6 7
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The student with the number 1 (the elitist component) can sit

$$1+\sum_{1}^{2}+\sum_{1}^{3}+\sum_{1}^{4}=20$$

times within 35 accidental distributions on the four seat bench, giving the probability 20/35 = 57%. One can take any one number from 1 to 7; the number 20 of all favourable distributions remains the same. 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=7 of replicating base components (above referred to as "students") only s-1=6 are permutated. The number of components in the four-digit ($s_1=4$) monotonous sequence are permuted, is s_1-1 since the elitist No.1 should always remain in the monotonous sequence. The number of remaining base components which permute itself, and which are located somewhere in the replicating fragment, is $s-s_1=7-4 = 3$. Thus follows for the number of favorable distributions at the first operation:

$$\sigma_{s1}=\frac{(s-1)!}{(s_1-1)!(s-s_1)!} \qquad (19a)$$

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

$$\sigma_{s1+1}=\frac{s!}{s_1!\,[s+1-(s_1+1)]!}=\frac{s!}{s_1!(s-s_1)!} \qquad (19b)$$

$$\sigma=\frac{\sigma_{s1+1}}{\sigma_{s1}}=\frac{s!}{s_1!(s-s_1)!}\cdot\frac{(s_1-1)!(s-s_1)!}{(s-1)!}=\frac{s}{s_1}\ , \qquad (19c)$$

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

$$W_{s1} = \frac{\sigma_{s1} \cdot r_c \cdot r_x \cdot r_y}{r_1} \qquad (20)$$

where

$$r_c = \frac{c!}{c_1! \cdot c_2! \cdot \ldots}$$

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

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

are the numbers of all possible c-, x-, y- distributions and $r_{1(2)}$ 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 c-, x-, y-distributions. The statistical propability that an elitist component appears in an (s_1+1)-digit monotonous sequence during replication is

$$W_{s1+1} = \frac{\sigma_{s1+1} \cdot r_{c-1} \cdot r_x \cdot r_y}{r_2} \qquad (21)$$

$$r_{c-1} = \frac{(c-1)!}{(c_1-1)! \cdot c_2! \cdot \ldots}$$

$$\frac{r_c}{r_{c-1}} = \frac{c}{c_1} \qquad (22)$$

$$\frac{W_{s1+1}}{W_{s1}} = \frac{\sigma_{s1+1}}{\sigma_{s1}} \frac{r_{c-1} \cdot r_1}{r_c \cdot r_2} \qquad (23)$$

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

$$\frac{W_{s1+1}}{W_{s1}} = \sigma \frac{r_{c-1} \cdot r_1}{r_c \cdot r_2} \qquad (24)$$

Dividing equation (18) through equation (19) results in

$$\frac{r_1}{r_2} = \frac{c}{s+1} \cdot \frac{s_1+1}{c_1} \qquad (25)$$

By inserting (22) and (25) into (24):

$$\frac{W_{s1+1}}{W_{s1}} = \sigma \cdot \frac{s_1+1}{s+1} \qquad (26)$$

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 DNA – fragment.

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.

5. The total probability of mutation which is caused by base rivalry.

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 GGGGGA. A tunnel process (probability P_1) creates a tautomeric base pair (A*-T*) at the end of the monotonous sequence because of base rivalry energy of the elitist.
2. Then the elitist replaces *necessarily* the base A* by the base G , and the binding energy between the new complementary bases G and T* increases itself because of high energy of the elitist and so causes an irreparable extension of the monotonous sequence.
(s. fig. 2c).

So is the total probability for an irreparable mutation (caused by base rivalry) at the end of an s1-digit monotonous sequence the product of tunnel-probability and of the probability of the one arriving elitists:

$$\varpi = P_1 \cdot W_{s1} \qquad (27)$$

and then the monotonous sequence is extended to s_1+1.

In case of second mutation event (tunnel probability P_2, elitist probability W_{s+1}) is the total probability

$$\varpi = P_2 \cdot W_{s1+1} \qquad (27a)$$

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

Therefore, it is

$$\frac{P_2}{P_1} = \frac{W_{s1}}{W_{s1+1}} \qquad (28)$$

and from eq.(17) and from eq. (26) follows

$$\exp\left\{\frac{1}{k_B}\left[\Delta_C - \Delta_S\right]\right\} = \frac{1}{\sigma}\cdot\frac{s+1}{s_1+1} \qquad (29)$$

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

$$\left[\Delta_S - \Delta_C\right] + k_B \ln\left[\frac{1}{\sigma}\cdot\frac{s+1}{s_1+1}\right] = 0 \qquad (30)$$

Equation (30) describes the change of temperature and energy necessary to provoke the lengthening of a monotonous sequence for two positions to s_1+1+1.

The first mutation event which generates the length s_1+1 can be a hydrogen bond with the characteristic temperature t_{01}, and the second mutation event which generates the length s_1+2 can be a hydrogen bond with the characteristic temperature t_{02}. However, in the lengthening of a monotonous sequence the same base type is always attached to the end. Therefore, the characteristic temperature remains the same at each lengthening of one and the same monotonous sequence.

Therefore it is easier to calculate only these types of changes which always relate to the same monotonous sequences. If so $t_{01} = t_{02} = t_0$, and considering equation (19c) then

$$\left[\Delta_c - \Delta s\right] - k_B \ln\left[\frac{s_1}{s}\cdot\frac{s+1}{s_1+1}\right] = 0 \qquad (31)$$

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

$$\Delta_C = \frac{E_2}{t_0} - \frac{E_1}{t_0} \qquad (14)$$

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

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

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 (31) can be written as

$$\left[\Delta_S - \Delta_C\right] + k_B \ln\left[\frac{s_1}{s}\cdot\frac{s+1}{s_1+1}\right] = 0 \qquad (32)$$

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

$$\Delta_S + k_B \ln\left[\frac{s_1}{s}\cdot\frac{s+1}{s_1+1}\right] = 0 \qquad (32a)$$

6. Interpretation of the equation (32)

From the equation (32) one finds that the left bracket represents the entropy change between two mutations whereas the right bracket is a measurement of the DNA – distribution change. The influence of the energies E_1 and E_2 is determinate by the base rivalry.
If occurs a temperature decrease from t_1 to t_2, then any time occurs an irreparable mutation of an (s_1+1)-digit up to an (s_1+1+1)-digit monotonous sequence. One must take into account that this can occur only in tunnel effects (caused by base-rivalry) within long spaces of time or in a large number of DNA – fragments since the tunnel process is rare.

At a monotonous sequence replication, the energy needed for an irreparable hydrogen bond creation *and* the energy needed for a tunnel process are only then reached if the monotonous sequence is long enough to accelerate the competing base components to a high energy level. The necessary length of the monotonous sequence depends on the viscosity of the braking nucleoplasm. The viscosity depends on the temperature. The viscosity is small at high temperatures; thereby, the competing base components obtain high energy after only a few replication steps. The viscosity becomes larger at low temperatures; thereby the required number of replication steps needed to provoke an irreparable lengthening of the monotonous sequence increases. Therefore, the original length s_1, where the monotonous sequence lengthening begins, depends on temperature. At higher temperatures, s_1 is small; at lower temperatures, s_1 is large. This is a condition for the equation (32). We shall see that this means a very important consequence for the number s (section 7).

7. 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. Normally, we explain this phenomenon with the help of theory of Darwinism. With the help of equation (32) we also receive another explanation: When after a warm period follows a cold period then (in a large DNA – lot) will occur irreparable mutations because of base rivalry. 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 (with a length 3).These short (monotonous) sequences must have lengthened themselves through temperature decrease considering of equation (32) 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 of 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

$$\frac{E\cdot\tau}{t_1^2-\tau\cdot t_1}+k_B\ln\left[\frac{s_1}{s}\cdot\frac{s+1}{s_1+1}\right]=0 \qquad (33)$$

(calculated in the publication „Die Physik irreparabler Mutationen" [7]) was derived on condition that every temperature change $t_1-\tau$ reduces the viscosity of the cell plasma by $\Delta\eta=0.324\cdot10^{-3}\,Pa\cdot s$ and too, that each such temperature change provokes an extension of an s_1-digit monotonous sequence exactly by 1 position during replication. The base rivaly energy T_k thus remains constant during every extension of the s_1-digit monotonous sequence.

We can thus calculate equation (33) not only for one base rivaly event, but also for several processes at different temperatures t_1 and different τ, where the high temperatures correspond to very early earth-ages and the small monotonic sequence-lengths s_1 correspond to the simplest living beings. We proceed in such a way that we assign certain s_1 to certain cell temperatures t_1 and determine the s using the equation (33):

τ^0	s_1	s_1+1	s	$t_1{}^0C$	$\eta[Pa\times s]$	E_k [eV]	n
1.85	6	7	12	68,1			
1.74	8	9	21	64.3	0.52×10^{-3}	0.411	2
1.7	10	11	41	60.49	1.168×10^{-3}	0.4089	2
1.7	12	13	136	57.03	1.816×10^{-3}	0.4067	2
1.5	14	15	310	52.87	2.464×10^{-3}	0.404	2
1.3	16	17	380	49.98	3.112×10^{-3}	0.406	2
1.15	18	19	510	47.4	3.76×10^{-3}	0.405	2
1.03	20	21	690	45.07	4.408×10^{-3}	0.397	2
0.93	22	23	860	43.02	5.056×10^{-3}	0.4026	2
0.85	24	25	1251	41.17	5.704×10^{-3}	0.4016	2
0.77	26	27	1440	37.77	6.352×10^{-3}	0.4007	2
0.71	28	29	1673	36.23	7×10^{-3}	0.3991	2

The energy $E_k = T_k - \frac{Rhc}{n^2} = T_k - \frac{13.656}{n^2}$ is always $E \approx +0.4eV$, as can be seen by the substitution of the different cell viscosities z into the basic competition equation

$$E_k = k\left(\frac{e_p^2}{r_1} - \frac{e_p^2}{r_B}\right) - z\cdot\sqrt{\frac{e_p^2}{r_1} - \frac{e_p^2}{r_B}} - 3.414eV \qquad (\mathrm{n} = 2)$$

The energy level n = 2 is because the equation (33) 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.
The cause for this increase is the equation (33): This equation must be recalculated for every t_1, τ, s_1 (ie, for each new evolution period). So statistically an one-position-lengthening of the monotonous sequence happens at the cell temperatures (t_1 and $t_1-\tau$), if are located s S-base components in the cell (within a mixture of any base components).

Many organisms in which such irreparabel mutations occurred, possess an advantage in the selection of species, so that they reach a higher stage in their hierarchy. Each new irrreparabel mutation goes along with an intense rise of the number s. Therefore the higher the stage the longer the DNA. The gradual reductions of t_1 and τ provoke the intense increase of DNA-length of the higher organisms in each new epoch. This process is always going along with lengthening of monotonous sequence for one position.

Just the increase of the number s at every (by base rivalry caused) mutation event offers the chance for all other bases, to produce the triplets in an always larger variety between the S-bases, which are present in always larger numbers.

Here is for example, the last row (table above), where base rivalry generates the 29-digit monotonous sequence from a 28-digit monotonous sequence at the cell temperature 36.23 grad C. This requires absolutely s = 1673 S - bases (same bases as in the monotonous sequence). Surely, there are all the other base- types in this fragment, so that 4 x 1673 = 6692 is the number of bases, and this is (approximately) the length of an Okazaki-fragment which is a replication unit, with a distribution start and a distribution end. The Okazaki – fragment is the copy

of the distribution on the leading strand, where the base rivalry is held.
Such pairs in the table above, which show a smaller s_1 and higher temperatures, belong to more primitive organisms which have smaller replication units and smaller lengthes of DNA.
To sum it up it can be said, that the gradual reductions of t_1 and τ provoke the increase of the length of DNA, and so the great variety of the coded triplets also increases itself.

The DNA – length has increased itself in the course of evolution.

8. Mutation physics

A similar effect of the equation (32) is when viruses will be brought in a large amount from an overheated (318.3 K) area into a cooler area (316.8 K) for a certain period. Look at an animal body in which each cell is infected by the same virus. The virus replication happens synchronously with the host replication. The residual fragment of the virus is a replication unit. The fragment

GGGGGAAGGACGGTTTTCCCCTG [according to eq.(32a) with E=0.5eV, t_1=316.8 K, t_2=315.3 K, s=10, s_1=5]

should be found once in the virus – genom. If there are 10^{12} cells in the animal body, then 10^{12} of such fragments are to find in the infected animal body.

These fragments are replicated within 36000 seconds. Considering eq. (27), we expect an irreparable mutation with the total probability

$$\varpi = P \cdot W$$

in each of the 10^{12} virus - fragments every 36000 seconds. Let be $P = 36 \times 10^{-12}$,
we get W = 0.1. Then will be expected

$$36 \times 10^{-12} \times 0.1$$

mutations in each of the 10^{12} virus–residual fragments every 36000 seconds:

$$\frac{36\cdot 10^{-12}\cdot 0.1}{36000\,s}=\frac{36\cdot 10^{-12}\cdot 0.1\ irrep.vir.mut.}{36\cdot 10^{3}\,s}=\frac{0.1}{10^{15}}=\frac{100}{10^{18}\,s}$$

Therefore, in the entire animal-body will occur

$$\frac{100\cdot 10^{12}\ irrep.vir.mut.}{10^{18}\,s}=\frac{100\cdot 10^{12}\ irrep.vir.mut}{10^{12}\cdot 10^{6}\,s}=\frac{1\ irr.vir.mut}{10^{4}\,s}=\frac{1}{2.78\,h}$$

This means that one irreparable virus mutation in the animal is expected after every 2.78 hours.

When so animals which are infected by a virus in an overheated area and then exposed to a lower temperature, then this can cause suddenly occurring dangerous virus mutations in birds or in other animals, and where the transmission to human beings cannot be excluded. A lengthening (caused by base rivalry) of a monotonous sequence can only happen, when the viscosity of the nucleoplasm increases itself. Increased viscosity causes a longer path of the competing base components until they have reached the necessary energy. A larger nucleoplasm viscosity is only possible in a long period of temperature decrease or by a cold influence on a large number of virus fragments (as in the calculated example: Viruses of a host remain in a warm area within a long period, and then in a cold area within a long period).

9. Summary

In small DNA-fragments, in the case of monotonous sequence replication there are two processes which can occur:

1. The change of thermodynamic entropy
2. The change of DNA-distribution

In this treatise, the attempt was made to find a connection between both processes: At an irreparable mutation (caused by base rivalry) there is a mathematical connection between the temperature change and the change of the DNA distribution: Within a long time period or in a large number of DNA-fragments there exists the equation

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

where the left bracket is the change of thermodynamic entropy, and the right bracket is a measurement of

DNA-distribution (monotonous sequence length s_1 enlarged up to s_1+2).
This equation could be useful for the theory of evolution and besides for clearing up of virus mutation. From the equation (33) it follows that in the course of the evolution, the gradual reduction of the temperature in the cases of base rivalry has led to an *incessant extension of monotonic sequences and to a strong increase in the total number of bases in the DNA, and so, has led to an incessant lengthening of the DNA.*

References

[1] W. D. Drechsel, Tumour physics. Mathematical Biosciences 213 (2008) 135-140
[2] P. Ö. Löwdin, Quantum genetics and the aperiodic solid. Some aspects on the biological problems of heredity, mutations, aging, and tumors in view of the quantum theory of the DNA molecule. Uppsala, Sweden, Uppsala Univ., Quantum Chemistry Group for Research in Atomic, Molecular and Solid-state Theory, 1963
[3] W. D. Drechsel, Berechnungen der Tunnel – Wahrscheinlichkeit in biologischen Wasserstoffbrückenbindungen
Available at: http://www.basenkon.com/also-tunnel-8.pdf
[4] Jan Florian and Jerzy Leszczynski, Spontaneous DNA Mutations Induced by Proton Transfer in the Guanine-Cytosine Base Pairs: An Energetic Perspective. J. Am. Chem. (1996)
3010-3017
[5] Yongho Kim, Sangbae Lim, Yangsoo Kim, The Role of a Short and Strong Hydrogen Bond on the Double Proton Transfer in the Formamidines Formic Acid Complex: Theoretical Studies in the gas phase and in solution. J. Phys. Chem A (1999) 6632-6637.
[6] Henryk Chojnacki, Jozef Lipinski, and W. Andrzej Sokalski, Potential Energy Curves in Complementary Base Pairs and in Model Hydrogen Bonded Systems. International Journal of Quant Chemistry, Vol XIX (1989) 339-346
[7] W. D. Drechsel, Die Physik irreparabler Mutationen, www.basenkon.com/kalkulation.pdf