Information Thermodynamics and Cell Telomere*

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Abstract

We consider the principle loss of the cell structure (information) caused by the *normal cell duplication.* This loss, known as *aging by a 'tooth of time',* can be measured by information entropy. Functionally, it can be described by a *carnotized model of information transfer,* by a *direct heat cycle.* The growth of the thermodynamic entropy in its environment is similar to the loss of the cell structure, measured, in biology, by the *shortenning* the length of the cell *telomere* [24]. The telomere is, said approximatelly, a box of chromozomes and its length is coding the age of the cell, or in other words, is coding the number of the cell's predecessors in the normal situation.

During the *pathological cell proliferation* the opposite situation arises. Cells with a precise structure are generated, but of a type which is different from their normal type. This growth of cells' structure in a certain locality (within the whole organism) is 'paid for' by the draining off energy from its environment. Again, this situation is describable by the thermodynamic model of information transfer, but in a *reverse form* this time. The decreasing value of the thermodynamic entropy, or increasing value of the information entropy is now evidenced (locally), but 'paid for' by the environment. In terms of cells, the growth of the cells structure is now measured by the *lengthening* of their telomeres [24].

Keywords: Carnot Cycle, Heat entropy, Transfer channel, Information entropy, Transinformation, Telomere.

1. Introduction

In this article we are thinking, in a *free way,* about possible applications of the Information-Thermodynamics point of view in *biology.* 1 We can consider a principle loss of information (structure) within the process of generation of cells by their duplication (dividing). Within any duplication of a cell *(the predecessor)* its *follower*

lower are his free *hypotheses* of analogy and functional type only. They are given as an *inspiration.*

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is generated and the a distortion of the *duplicated* (copied) structure (information) of the parent cell in the follower arises. This loss is measurable by the quantity of (average) information amount. It is a loss of part of message being copied (transferred), it is a loss of information within this process of such an information transfer. (The whole *structure of the cell is a message,* also including 'a program' for its functionality in a texture.) Now we have reviewed a mechanism of aging by a 'tooth of time'. This mechanism can be described in a functional way by the *'camotized ' model of information transfer* - *direct Carnot Cycle viewed informationally.*

Within the pathological proliferation of cells the opposite situation arises. In this case the cells with a *precise structure* (but, of an *another type* in comparison with the normal structure of its own original and 'normal' type) are generated. But, this grow of structure in a certain locality in the whole organism is paid by a pumping off energy from an environment of this locality. This situation is describable again by our Information-Thermodynamic model, but by a *reverse* this time.

2. Basics of Information Thermodynamics

2.1 Reversible Carnot Cycle and Channel

Fig. 2.1. Carnot Cycle

We term the following ratios [expressed in *information* units *(Hartley, nat, bit)*] the *changes of the (thermodynamic) entropies of the system* $\mathcal L$ *in cycles* $\mathcal O$ *or* $\mathcal O'$ *:*

$$
\frac{\Delta Q_W}{kT_W} \text{ input}, \frac{\Delta A^{[l]}}{kT_W} \text{ output } (\triangleq \Delta I^{[l]}), \frac{\Delta Q_0}{kT_W} \text{ loss}, \frac{\Delta Q_{0x}}{kT_W} \text{ noise}, \tag{1}
$$

where k is Boltzman's constant. These changes are the absolute values of *information entropies H(·), H(·I·)* contained in any message on inputs and outputs of a *'carnot' (thermodynamically)* described transfer channel *K}*

Fig. 2.2. The schema of a reversible Carnot Cycle

A reversible Carnot Cycle $\mathcal O$ running in $\mathcal L$ (producing noise heat $\Delta Q_{0x} = 0$) can be considered to be a *thermodynamic, average-value* realization or, as such, as a model of an information transfer process running in a channel K *without noise.* On the channel K the information entropies $H(X)$, $H(Y)$, $H(Y|X)$ are defined,

$$
H(X) \stackrel{\text{Def}}{=} \frac{\Delta Q_W}{kT_W} \left[= \frac{\Delta Q_0}{kT_0} \right] \tag{2}
$$

$$
H(Y) \stackrel{\text{Def}}{=} \frac{\stackrel{\text{I}}{\Delta A}}{\stackrel{\text{I}}{\Delta T_W}} = \frac{\stackrel{\text{I}}{\Delta Q_W} - \stackrel{\text{I}}{\Delta Q_0}}{\stackrel{\text{I}}{\Delta T_W}} = \frac{\stackrel{\text{I}}{\Delta Q_W}}{\stackrel{\text{I}}{\Delta T_W}} \cdot \eta_{max} = H(X) \cdot \eta_{max} \stackrel{\triangle}{=} \Delta I
$$

$$
H(Y|X) \stackrel{\text{Def}}{=} 0
$$

Fig. 2.3. The information transfer channel modelling a reversible Carnot Cycle

Thus we assume that the medium $\mathcal L$ going through a reversible Carnot Cycle $\mathcal O$ works as a thermodynamic, average-value model of an information channel \mathcal{K} ($\mathcal{K} \sim \mathcal{L}$) and that the *channel equation*

$$
H(X) - H(X|Y) = [T(X;Y) = T(Y;X)] = H(Y) - H(Y|X)
$$
\n(3)

²The symbols \mathcal{O}', A', I' and ΔQ_{0x} are for the irreversible case.

is valid for the values of the quantities in (2). Following from $\frac{\Delta Q_W}{T_W} = \frac{\Delta Q_0}{T_0}$ and the definition (2) and the assumption (3) we have

$$
\frac{\Delta Q_W}{kT_W} \cdot \eta_{max} - 0 = \frac{\Delta Q_W}{kT_W} - H(X|Y) \tag{4}
$$
\n
$$
\Rightarrow \frac{L(Y|Y)}{kT_W} \cdot \frac{\Delta Q_W}{kT_W} \cdot (1 - x) = \frac{\Delta Q_W}{kT_W} \cdot \frac{\Delta Q_W}{kT
$$

$$
H(X|Y) = \frac{\Delta \psi}{kT_W} \cdot (1 - \eta_{max}) = \frac{\Delta \psi}{kT_W} \cdot \beta = \frac{\Delta \psi}{kT_W}, \quad \beta = \frac{10}{T_W}
$$

Thus for the transinformations $T(X; Y)$ and $T(Y; X)$ defined in (3), and, using the definitions (2), we have

$$
T(X;Y) = H(X) \cdot (1 - \beta) = H(X) \cdot \eta_{max}
$$

\n
$$
T(Y;X) = \frac{\Delta Q_W}{kT_W} \cdot \eta_{max} = \frac{\Delta A}{kT_W}
$$
 and thus
\n
$$
T(X;Y) = \Delta I = H(Y) = T(Y;X)
$$
\n(5)

As a consequence we have ³⁴

$$
H(Y) = \Delta I = T(X;Y) < H(X) \tag{6}
$$

When the medium $\mathcal L$ has gone through the cycle $\mathcal O$ once [and in agreement with When the medium $\mathcal L$ has gone through the cycle $\mathcal O$ once [and in agreement with $\oint_{\mathcal O} \frac{\delta Q(T)}{T} = 0$], we have the following for the change $\Delta S_{\mathcal L}$ of the heat entropy $S_{\mathcal C}$ of the whole reversible Carnot

$$
\Delta S_{\mathcal{L}} = \oint_{\mathcal{O}} \frac{\delta Q}{T} = \frac{\Delta Q_W}{T_W} - \frac{\Delta Q_0}{T_0} = 0 \tag{7}
$$

As a consequence of the additivity of (substitute reversible [20]) changes of heat entropy, when the medium $\mathcal L$ has gone through the reversible cycle $\mathcal O$ once, we have the following for the change ΔS_{AB} of the heat entropy S_c in the system (AB) (the heater \mathcal{A} , $T_W > 0$ and the cooler \mathcal{B} , $T_0 > 0$, $T_W \geq T_0$:

$$
\Delta S_{\mathcal{A}\mathcal{B}} = -\frac{\Delta Q_0}{T_W} + \frac{\Delta Q_0}{T_0} = \frac{\Delta Q_0}{T_0} \cdot \eta_{max} = \frac{\Delta Q_W}{T_W} \cdot \eta_{max} = kT(X;Y) \tag{8}
$$

Also we have the following for the *result* change ΔS_c of the heat entropy S_c of the whole reversible Carnot engine:

$$
\Delta S_{\mathcal{C}} = \Delta S_{\mathcal{L}} + \Delta S_{\mathcal{A}\mathcal{B}} = \frac{\Delta Q_W}{T_W} \cdot \eta_{max} > 0
$$
\n(9)

The efficiences of all reversible Carnot Cycles with the working temperatures T_W *and* T_0 *are equal.* ⁴The loss $H(X|Y) \neq 0$ is inseparabile from our type of transfer of $x \in X$, $x \sim \Delta Q_W$; let it be considered as a model for bilogical aging, see further.

³The sharp enequality represents **Thomson-Planck's** information formulation of the II. Principle of Thermodynamics

The equality in the relation (6) is valid for all reversible Carnot Cycles (with temperatures T_W and *To)* viewed informationally, and can be considered to be an *information* formulation of the **first part** part of **Carnot's** theorem which states:

The derivation (5) and the equation (9) together then imply that

$$
\Delta S_{\mathcal{C}} - kT(X;Y) = kH(X) \cdot (\eta_{max} - \eta_{max})
$$

\n
$$
\Delta S_{\mathcal{C}} - k\Delta I = 0 \text{ or also } \Delta(S_{\mathcal{C}} - kI) = 0
$$
\n(10)

On receiving the output information $\Delta I = H(Y)$

• the *distinguishability* of the structure of the **mechanical output** of \mathcal{O} , being measured by $\Delta I = H(Y)$, **increases**; it is realized by the addition ΔA to the potential output energy of \mathcal{O} ,

• the *thermodynamic* **distinguishability** of the system *(AB) ,* a *thermodynamic structure* of which we express by mutually different heats, **decreases** just by the value $\Delta S_c = \Delta S_{AB} (9); \Delta S_c = k\Delta I.$

• The increasing ΔI of the *structural* distinguishability of the mechanical output models the receiver's average information increase by the addition ΔI contained in any message $y \sim \Delta A$, $y \in Y$, on the output of $\mathcal{K}(\sim \mathcal{L})$.

The value ΔS_C thus represents the **increase** in the whole *extensity* of the energy (the extensity is now the thermodynamic entropy) used for the coding the input message, and thus represents the **increase** of the **indistinguishability** of this energy within the whole isolated transfer chain (X, \mathcal{K}, Y) - the whole Carnot engine (thus, represnts the decrease of the structureness of the complete system)

2.2 Reverse Reversible Carnot Cycle and Channel

Reverse reversible Carnot Cycle works as a *heat pump.* In this cycle, comprehended as a thermodynamic, average-value realization, or model of the transfer process in a channel $K \cong \mathcal{L}$ which is transferring an (arbitrary) input message $x \in X$ containing the average information amount $H(X)$, we use these symbols and denotations:

 ΔQ_0 the heat drained off from the cooler B within the isothermal expansion at T_0 , ΔA the mechanical work delivered to the cycle by the compression phase at T_W , ΔQ_W the output heat delivered to the heater A by the isothermal phase at T_W .

Further, we define the values of changes of *information entropies* on the channel $\mathcal{K} \cong \mathcal{L}$ (with an information transfer process being realized by this cycle) by the changes of its *physical (now thermodynamic) entropies,* for instance, in this way:

$$
H(X) \stackrel{\text{Def}}{=} \frac{\Delta A}{kT_W}, \text{ input entropy} \tag{11}
$$
\n
$$
H(Y) \stackrel{\text{Def}}{=} \frac{\Delta Q_W}{kT_W} = \frac{\Delta Q_0 + \Delta A}{kT_W} \stackrel{\Delta}{=} \Delta I, \text{ output entropy}
$$
\n
$$
H(Y|X) \stackrel{\text{Def}}{=} \frac{\Delta Q_0}{kT_W} > 0, \text{ noise entropy}
$$

where $\Delta A \cong x \in X$ is the *input message*, $\Delta Q_W \cong y \in Y$ is the *output message* and ΔQ_0 is the *noise 'message'*.

Fig. 2.4. The additive information transfer model for a reversible Carnot Cycle without losses Now we consider a channel with the *additive* noise,

$$
H(Y|X) = \frac{\Delta Q_0}{kT_W} \cdot \frac{T_0}{T_0} = \frac{\Delta Q_0}{kT_0} \cdot \beta = \frac{\Delta Q_W}{kT_W} \cdot \beta = H(Y) \cdot \beta, \ \beta = \frac{T_0}{T_W} \tag{12}
$$

is to be valid. The noise with information entropy $H(Y|X)$ is the integral part of the definition of the transfer information process. It is not generated by a positive production of the noise heat $\Delta Q_{0x} > 0$ in the working medium \mathcal{L} ⁵.

We are supposing further that for changes of information at the values values $H(X)$, $H(Y|X)$, $H(Y)$, $H(X|Y)$ defined by (11) the relations (3) are valid and, then,

$$
\frac{\Delta A}{kT_W} - H(X|Y) = \frac{\Delta Q_0 + \Delta A}{kT_W} - \frac{\Delta Q_0}{kT_W}
$$
\n
$$
H(X|Y) \stackrel{\text{Def}}{=} 0.
$$
\n(13)

So, we have a channel *without losses.* For transinformations $T(X; Y)$, $T(Y; X)$, with respecting the definition (11) it is valid, within one run of the system $\mathcal{L} \cong \mathcal{K}$ through the reverse Carnot Cycle realizing a transfer process, that

$$
T(X;Y) = H(X) - H(X|Y) = \frac{\Delta A}{kT_W} - 0 = H(X)
$$
\n
$$
T(Y;X) = H(Y) - H(Y|X) = \frac{\Delta Q_0 + \Delta A}{kT_W} - \frac{\Delta Q_0}{kT_W} = \frac{\Delta A}{kT_W} = H(X).
$$
\n(14)

Consequently

$$
H(X) = H(Y) \cdot \eta_{\text{max}} \tag{15}
$$

 kT_W kT_W kT_W

where η_{max} is the efficiency of the relevant direct cycle. So it is valid, in accordance with relations (12) and (3), that

$$
H(X) = T(X;Y) = H(Y) \cdot \eta_{\text{max}} \tag{16}
$$

Now, let us notice the changes of thermodynamic entropy in an isolated system in which the described process is running:

$$
\Delta S_{\mathcal{A}\mathcal{B}} = \frac{-\Delta Q_0}{T_0} + \frac{\Delta Q_0}{T_W} = \frac{-\Delta Q_0}{T_0 T_W} \cdot (T_W - T_0) =
$$
\n
$$
= \frac{-\Delta Q_0}{T_0} \cdot \eta_{\text{max}} = -H(Y) \cdot \eta_{\text{max}} < 0
$$
\n
$$
(17)
$$

⁵From the relations for η and η_{max} follows that $\Delta Q_0 = f(T_0)$, where function $f(\cdot)$ is a notnegative function of the argument T_0 , $f(T_0) \ge 0$, for which $\lim_{T_0 \to 0} f(T_0) = 0$ is valid.

The thermodynamic (Clausius) entropy S_{AB} of the system (AB) is then lowering its value - the (termodynamic, heat) distinguishability of the systems **A** and **B** is growing up. Of course, it is at a detriment of the mechanical work ΔA delivered,

or, respectively, at a detriment of the entropy at the value $\frac{\Delta A}{T}$.

But, there is a need to gain this energy (entropy) and it is possible within such an isolated system by a not natural process of transformation heat to mechanical energy. But, this process is 'running ' at a background of a natural process of the heat transfer in accordance with the II. Principle of Thermodynamics.

2.2.1 Two connected Carnot Cycles

We are considering such a reversible process which is giving the mechanical work at a value $\Delta A^* \geq \Delta A$; we write it with regard to various directions of functioning of both these cycles $(T^*w \geq T_w$ and $T^*_{0} \leq T_0$;

$$
\frac{\Delta A^*}{kT_W^*} = H(X^*) \cdot \eta_{\text{max}}^* = H(X^*) \cdot \frac{T_W^* - T_0^*}{T_W^*}
$$
\n
$$
= \frac{1}{k} \Delta S_{A^*B^*}, \quad T_W^* \ge T_0^* > 0,
$$
\n
$$
\frac{\Delta A}{kT_W} = H(Y) \cdot \eta_{\text{max}} = H(Y) \cdot \frac{T_W - T_0}{T_W}
$$
\n
$$
= -\frac{1}{k} \Delta S_{AB}, \quad T_W \ge T_0 > 0
$$
\n(18)

For the whole change ΔS of the entropy *S* of the whole isolated system in which both these processes are running, following the II. Principle of Thermodynamics, is

$$
\Delta S = \Delta S_{\mathcal{A}^* \mathcal{B}^*} + \Delta S_{\mathcal{A} \mathcal{B}} \ge 0 \tag{19}
$$

But, for $\Delta S_{AB} \leq 0$, it must be valid that

$$
\Delta S_{\mathcal{A}^* \mathcal{B}^*} \ge |\Delta S_{\mathcal{A} \mathcal{B}}| \tag{20}
$$

This means that for the decrease of entropy about the value $|\Delta S_{AB}|$, the greater addition of $\Delta S_{A^*B^*}$ is to be generated, and, then, the whole entropy is growing up just about the value

$$
\Delta S = \Delta S_{\mathcal{A}^* \mathcal{B}^*} - |\Delta S_{\mathcal{A} \mathcal{B}}| \ge 0. \tag{21}
$$

The equality occures when $\eta_{\text{max}}^* = \eta_{\text{max}}$. In another case η_{max}^* > η_{max} which, e.g. for $T_0^* = T_0$, means that

$$
\Delta Q_W^* > \Delta Q_W \quad \text{and} \quad T_W^* > T_W \tag{22}
$$

*The environment (A*B*) of the entropy decrease area [which is the system (AB)] is being 'pumped off', as for its structure conserving energy,* *in a greater measure (or at least at the same one)* - *its undistinguishability, disorder (chaos) is growing up more (or at the same measure) than this decrease, the local growing up of the order or organization in* (AB) *is.*

3. Biology Analogy for Cell Generation

We can enviswge and suppose further, that the heat engines are, in a ceretain way, the real physical background (let us say a motor) for the cells' duplications.

3.1 Analogy for Normal Cell Proliferation

The result of the *normal* generation of cells is that: the continuously decreasing precise of the structure of the followers is generated in a sequence of copying, duplication the cells, and, the whole biological organism, the collection of textures of cells, is aging by "the tooth of time" - by the loss of their *structure,* the precision of the construction of the follower cells (for this, by the loss of both their inner and outer bounds). At the end, following a number of duplications, the incompatibility of the resulting structure of the last cell (the incompatibility of the information amount being represented by this cell) with such a certain minimal structure (information), which maintains its ability of both inner and outer communication, arises, that it is not recognizable as "a certain cell" of the certain cell type - so as being alive.

This mechanism can be described in a functional way by our *direct* 'carnotized' model of information transfer. Within any run of our model Carnot Cycle (modeling the cell duplication) the less output (average) information is gained in comparison with the input (average) information (2),

$$
H(Y) = \Delta I < H(X) \tag{23}
$$

If this resulting information is used again (in our model in the heat form with appropriate temperatures

$$
T_{W,i+1} < T_{W,i}, \ T_{0,i+1} = T_{0,i} = T_0, \ i \geq 1; \ T_{W,1} = T_W \tag{24}
$$

the less information is generated etc. But, contemporarily, any run of the cycle generates the positive addition of entropy (thermodynamic) of a wider isolated system in which this transformation (information transfer) runs (10),

$$
\Delta S_{\mathcal{C}} > 0 \tag{25}
$$

In this model it is the less and less distinguishability within the system **(AB)** [of parts $\mathcal A$ and $\mathcal B$ of the whole model heat engine [model for the body], as for their heat contents $[\Delta S_{AB} = \Delta S_c]$ - let it be, together with (23), our thermodynamic model of that normal aging of body by 'a tooth of time'; the system **(AB)** is the

model for the normal cell. So, the sought 'gen of aging' could be nothing else than a datum about the precision of that duplication; in our model it is the efficicency of the transformation of the input energy,

$$
\eta_{max} < 1 \tag{26}
$$

So it is clear that the loss of information transferred this way is a suitable functional model for the case of the duplication of cells.

As for cells, let us say that this principle reveals, signalizes itself by the phenomenon of the *shortening of telomeres* [24],

$$
l \cong f(S^+ - \Delta S_{AB}) < f(S^+), \quad \Delta S_{AB} > 0 \tag{27}
$$

 $f(\cdot)$ is a growing function and S^+ is a starting value for the given generation run.

3.2 Analogy for Pathologic Proliferation

Within the *pathological* generation, proliferation of cells the opposite situation than it is in a normal case arises. In the pathological case we have the cells [modeled by the subsystem $(\mathcal{A}\mathcal{B})$ within those two cycles with a more precise structure (but, of an another type in comparison with the normal structure of its own original and 'normal' type). But, this grow of structure in a certain locality in the whole organism is paid by a pumping off energy from an environment of this locality (from the rest sound parts of the organism-body).

This environment is then in a lack of energy for its normal functionality; this energy is consumpted in the favour of this local grow of structure signalized by that phenomenon that the followers of the predecessors have the telomeres lengthened, see [24],

$$
l \cong f(S^+ - \Delta S_{AB}) > f(S^+), \quad \Delta S_{AB} < 0 \tag{28}
$$

 $f(\cdot)$ is a growing function and S^+ is a starting value for the given generation run.

This situation is describable again by our Information-Thermodynamic model, but by a *reverse* this time. In this model a *local* decreasing of entropy is evidenced (17),

$$
\Delta S_{\mathcal{AB}} = -kH(Y) \cdot \eta_{\text{max}} < 0, \ H(Y) > 0 \tag{29}
$$

as for the heat contents of the system (AB) , and, then, the grow of structure of such a locality (within a wider isolated system) appears; the system (AB) is the model for the cancer cell.

However, for this decrease of entropy (for this grow of structure) the energy is needed at the value (18) but, delivered from the environment (20) of this locality [the model for the exhausting the rest sound part of the body],

$$
\Delta A^* > 0, \quad \Delta S_{A*B*} > 0 \tag{30}
$$

This is the price for the greater and greater structure of this its (open) part, the problematic locality which leads to the grow of entropy of this environment and, consequently, to the grow of entropy of the whole isolated wider system (organismbody), see (19).

In the case of cells we can see the wasting away of the whole organism. The final equilibrium is approaching again and faster, e.g.:

$$
(\Delta S^{\dagger} \stackrel{?}{=} \Delta S_{\mathcal{A}^* \mathcal{B}^*} - \Delta S_{\mathcal{A} \mathcal{B}} = \Delta S_{\mathcal{A}^* \mathcal{B}^*} + |\Delta S_{\mathcal{A} \mathcal{B}}| > \Delta S > 0
$$
\n(31)

while the grow of entropy of the whole (wider) system-organism is ΔS , (19)].

Our reverse Information-Thermodynamic model authorizes us to an awaiting of a stable (moderate) higher body temperature of a patient (the medicin fact) and, also, a less temperature of the problematic texture, see $(18)^6$,

$$
T_W^* > T_W \tag{32}
$$

The equations (27) and (31) should be valid for the cells' structure changes whether or not the cells are equipped with telomeras.

4. Conclusion

For the pathology proliferating texture is consumpting the energy from the rest of the organism it is logical, as for the therapy, to try to cut off this energy supply. Following the previous considerations, we can imagine the lowering of the tumour's consumption of energy from the organism, body as a whole e.g. by the body's cooling (therapy by intensive freeze, swimming in a cold bath; especially in a not-operable cases of such disease). This could be the way to $\lim_{t \to \infty}$ at least for a short time, the positive flow of heat supporting the cell metabolic processes **in** such problematic localities. This means the lowering the temperature gradient between the sound and the ill textures.⁸May be that the **antipyretical effects of the Salicylic Acid**

⁸Even in the case of the sacrum where the 'impuls' of higher temperature of this locality is used, the inevitable successive process of natural cooling is imaginable, creating the appropriate negative temperature gradient.

⁶These temperatures are those of our thermodynamic model. The values of the body's temperatures will be different (for both the normal and the pathological case) due to the influence of *enzyms.* Nevertheless the inequality sign will remain the same due to the general physical base.

⁷Nowadays it seems to be proved that the cancer tumour is modifying its environment **in** such a way that this environment is pressed to deliver to it certain proteins for its growing up. The question is how to cut or to limit this nutricing connection. One of the method is cryotherapy used for the breast cancer: **within the environment of the tumour the freezing gas is injected with the result of the tumour's diminishing.**

From the point of view of the energy consumption also the real surgery method of *devitalization* which is narrowing the cnonection between the pathology tumour and its environment *[MUDr. Karel Fortyn, CSc. , Ustav iivocisne fyziologie a genetiky AV CR v Libechove, 1gs1, 1971-2001]* seems to be a right way.

(Aspirin) are the part of preventive effects having been registered these days.

We can imagine this situation: Supposingly, the occurance of the pathological cell is indicated as the consequnce of a virosis [whether or not such cell is a result of the (natural) degeneration or is (really) caused by a virus] - the result (as for the pathological change of the cell structure) is the same; **the organism reacts by its higher temperature.** But, this defence of the body is not functioning at this case, but, on the contrary, it **creates just what the pathological cell requires** - **just the higher temperature of the body** - let us say that it is a thermodynamic explanation why the **imunology of the body fails.**

It seems to be right to await that our Information-Thermodynamic approach, now expressing the biological terms as growing and lowering of a structure of cells in terms growing and lowering of a measure of a certain physical structure {the author used the information entropy defined on the thermodynamic structure}, is capable, in the quantitative-functional-analogy point of view, describe the very general properties of very various phenomena.

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