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Information-Thermodynamic Consideration on Cancer Cells[1], Cancer and Structure

Bohdan Hejna*

Department of Mathematics, University of Chemistry and Technology, 166 28 Prague 6, Czech Republic

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***Corresponding author:** Bohdan Hejna, Department of Mathematics, University of Chemistry and Technology, 166 28 Prague 6, Czech Republic

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Introduction

In this paper we are thinking, in a freeway, about possible applications of the Information- Thermodynamics point of view in biology2. We can consider a principal 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 is generated and the 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.) This mechanism of aging by a 'tooth of time' can be described in a functional way by the 'carnotized' model of information transfer - direct Carnot Cycle viewed informationally. The growth of thermodynamic entropy in its environment is similar to the mentioned loss of the cell structure, measured, in biology, by the shortening the length of the cell telomere. The telomere is, approximately, a box of chromosomes and its length codes the age of the cell, or the number of the cell's predecessors in the normal situation. Also, the (sound) cells are aging any time, with or without telomeres.

Within the pathological proliferation of cells, the opposite situation arises. In this case the cells with a precise structure (but, of another type in comparison with the normal structure of its own original and 'normal' type) are generated. But this growth of structure in a certain locality in the whole organism is paid by a pumping oenergy from an environment of this locality (the rest of the whole body). This situation is describable again by our Information- Thermodynamic model, but by a reverse this time. The decreasing value of thermodynamic entropy or increasing value of information entropy is now evidenced locally with the higher growth of the thermodynamic entropy within the whole environment of the reverse cycle [1]. This process is similar to the growth of the cell structure in the pathological case which growth is now measured by the lengthening of the cell telomeres.

Basics of Information Thermodynamics

In the following text the symbols O and O' denote the Carnot Cycle, reversible and irreversible respectively, in the medium L. The symbols A and B denote heater with the temperature T_w and the cooler with the temperature T_0 respectively. The symbols $\Delta \mathrm{Q_W}$ and $\Delta \mathrm{Q_0}$ denote the relevant heats and $\Delta \mathrm{Q_{0x}}$ denotes the noise heat generated in the irreversible cycle O' (Figure 1).

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

$$\frac{\Delta \mathrm{Q_W}}{kT_w} input, \frac{\Delta \mathrm{A}^{[\prime]}}{kT_w} output(\triangleq \Delta \mathrm{I}^{[\prime]}), \frac{\Delta \mathrm{Q_0}}{kT_w} loss, \frac{\Delta \mathrm{Q_{0x}}}{kT_w} noise, \quad \text{eq 2.1}$$

where κ is Boltzman's constant. These changes are the absolute values of information entropies $H(\cdot)$, $H(\cdot|\cdot)$ contained in any message on inputs and outputs of a 'carnot ' (thermodynamically) described transfer channel κ [2]. For the simplicity further the reversible case O is used only.

[1]Supported by the grant of Ministry of Education of the Czech Republic MSM 6046137307.

[2]This paper is based on [5 6,7] improving them in language and formulations. The author emphasizes, strongly, that the ideas expressed here are his free hypotheses of analogy and functional type only. They are given as an inspiration.

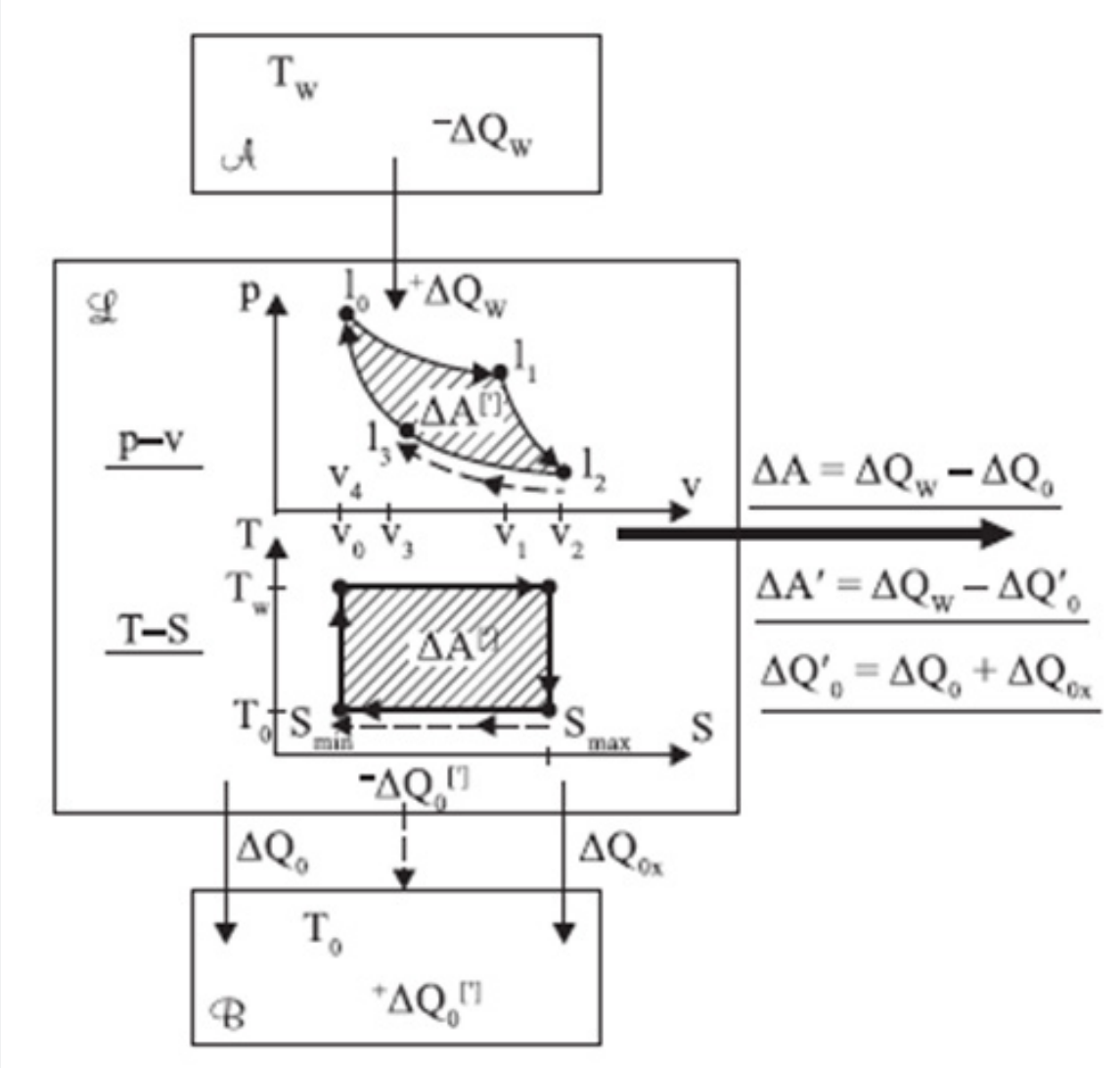

Figure 1: Carnot Cycle.

Reversible Carnot Cycle and Channel

(Figure 2) A reversible Carnot Cycle O running in 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 an information transfer channel K without noise. On the channel K the information entropies $H(X)$, $H(Y)$, $H(Y \mid X)$ are defined,

$$H(X) \overset{Def}{=} \frac{\Delta Q_W}{kT_W}\left[=\frac{\Delta Q_0}{kT_0}\right] \qquad \text{Eq 2.2}$$

$$H(Y) \overset{Def}{=} \frac{\Delta A}{kT_W} = \frac{\Delta Q_W - \Delta Q_0}{kT_W} = \frac{\Delta Q_W}{kT_W}\cdot\eta_{\max} = H(X)\cdot\eta_{\max} \triangleq \Delta I$$

$$H(Y \mid X) \overset{Def}{=} 0$$

(Figure 3)

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

$$H(X) - H(X \mid Y) = [T(X;Y) = T(Y;X)] = H(Y) - H(Y \mid X) \qquad \text{Eq 2.3}$$

[3]The sharp inequality represents Thomson-Planck's information formulation of the II. Principle of Thermodynamics. The equality in the relation (2.6) is valid for all reversible Carnot Cycles (with temperatures TW and T0) viewed informationally, and can be considered to be an information formulation of the first part of Carnot's theorem which states:

The efficiencies of all reversible Carnot Cycles with the working temperatures T_W and T_0 are equal.

[4]The loss $H(X \mid Y) \neq 0$ is inseparable from our type of transfer of $x \in X$; $x \sim \Delta Q_W$; let it be considered as a model for biological aging, see further.

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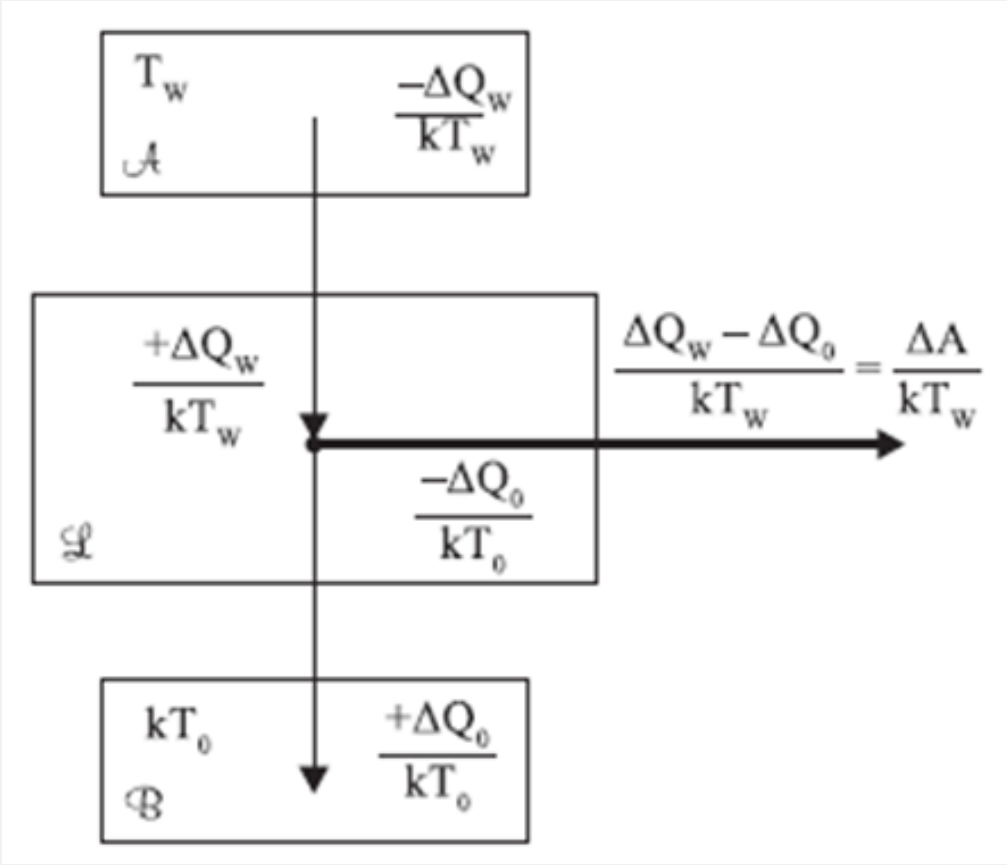

Figure 2: The schema of a reversible Carnot Cycle.

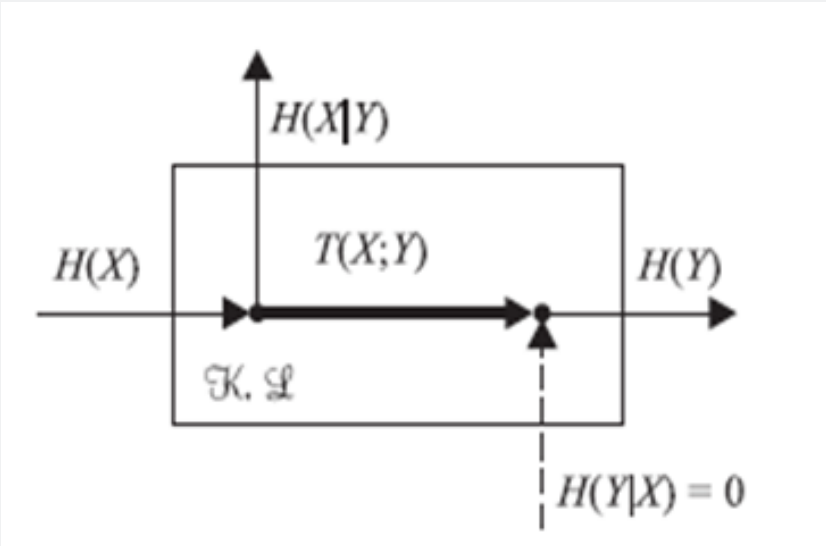

Figure 3: The information transfer channel modelling a reversible Carnot Cycle.

is valid for the values of the quantities in (2.2). Following from $\frac{\Delta Q_W}{T_W} = \frac{\Delta Q_0}{T_0}$ and the definition (2.2) and the assumption (2.3) we have

$$\frac{\Delta Q_W}{kT_W} \cdot \eta_{\max} - 0 = \frac{\Delta Q_W}{kT_W} - H(X \mid Y) \qquad \text{Eq 2.4}$$

$$H(X \mid Y) = \frac{\Delta Q_W}{kT_W} \cdot (1 - \eta_{\max}) = \frac{\Delta Q_W}{kT_W} \cdot \beta = \frac{\Delta Q_0}{kT_W}, \beta = \frac{T_0}{T_W}$$

Thus, for the transinformations $T(X;Y)$ and $T(Y;X)$ defined in (2.3), and, using the definitions (2.2), we have

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$$T(X;Y) = H(X)\cdot(1-\beta) = H(X)\cdot\eta_{\max} \quad \text{Eq 2.5}$$

$$T(Y;X) = \frac{\Delta Q_W}{kT_W}\cdot\eta_{\max} = \frac{\Delta A}{kT_W} \quad \text{and thus}$$

$$T(X;Y) = \Delta I = H(Y) = T(Y;X)$$

As a consequence, we have[3,4]

$$H(Y) = \Delta I = T(X;Y) < H(X) \quad \text{Eq 2.6}$$

When the medium L has gone through the cycle O once [in agreement with $\oint_O \frac{\delta Q(T)}{T} = 0$ for a reversible cycle], [3] we have the following for the change ΔS_L of the heat entropy S_C of the whole reversible Carnot engine:

$$\Delta S_L = \oint \frac{\delta Q}{T} = \frac{\Delta Q_W}{T_W} - \frac{\Delta Q_0}{T_0} = 0 \quad \text{Eq 2.7}$$

As a consequence of the additivity of (substitute reversible [4]) changes of heat entropy, when the medium L has gone through the reversible cycle O once, we have the following for the change ΔS_{AB} of the heat entropy S_C in the system (AB) (the heater $A, T_W > 0$ and the cooler B; $T_0 > 0$, , $T_W > T_0$):

$$\Delta S_{AB} = -\frac{\Delta Q_0}{T_W} + \frac{\Delta Q_0}{T_0} = \frac{\Delta Q_0}{T_0}\cdot\eta_{\max} = \frac{\Delta Q_W}{T_W}\cdot\eta_{\max} = k\cdot T(X;Y) \quad \text{Eq 2.8}$$

Also, we have the following for the result change ΔS_C of the heat entropy S_C of the whole reversible Carnot engine:

$$\Delta S_C = \Delta S_L + \Delta S_{AB} = \frac{\Delta Q_W}{T_W}\cdot\eta_{\max} > 0 \quad \text{Eq 2.9}$$

The derivation (2.5) and the equation (2.9) together then imply that

$$\Delta S_C - k\cdot T(X;Y) = kH(X)\cdot(\eta_{\max} - \eta_{\max}) \quad \text{Eq 2.10}$$

$$\Delta S_C - k\Delta I = 0 \quad \text{Or also } \Delta(S_C - kI) = 0$$

On receiving the output information $\Delta I = H(Y)$ we see that:

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

The thermodynamic distinguishability of the system (AB), a thermodynamic structure of which we express by mutually different heats, decreases just by the value $\Delta S_C = \Delta S_{AB}$ (2.9); $\Delta S_C = k\Delta I > 0$.

The increasing ΔI of the structural distinguishability of the mechanical output of the cycle O 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 $K(\sim L)$. Now it is a message of the structure measure of the new gained and less structured resulting cell.

In other words, 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,K,Y) - the whole Carnot engine - complete system – the cell. Thus, it represents the relevant decrease of the structure (aging) of the whole body and, especially, of the aging cell [5].

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 $K \cong L$ 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 o 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 dene the values of changes of information entropies on the channel $K \cong L$ (with an information transfer process being realized by this cycle) by the changes of its physical (now thermodynamic) entropies, for instance, in this way:

$$H(X) \overset{Def}{=} \frac{\Delta A}{kT_W}, \text{ input entropy} \quad \text{Eq 2.11}$$

$$H(Y) \overset{Def}{=} \frac{\Delta Q_W}{kT_W} = \frac{\Delta Q_0 + \Delta A}{kT_W} \triangleq \Delta I, \text{ output entropy}$$

$$H(Y|X) \overset{Def}{=} \frac{\Delta Q_0}{kT_W} > 0, \text{ noise entropy}$$

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' (Figure 4).

[5]From the relations for η and $\eta_{\max}$ follows that $\Delta Q_0 = f(T_0)$, where function $f(\cdot)$ is a not-negative function of the argument $T_0 \cdot f(T_0) \geq 0$, for which $\lim_{T_0 \to 0} f(T_0) = 0$ is valid.

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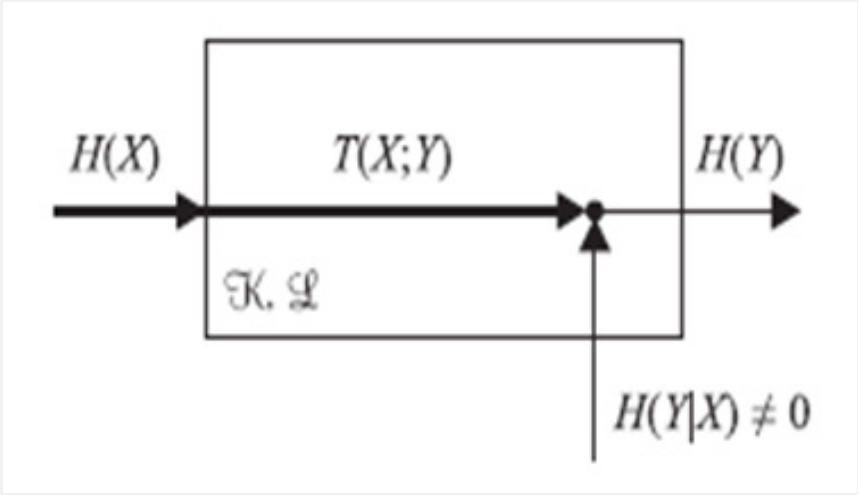

Figure 4: The additive information transfer model for a reversible Carnot Cycle without losses.

Now we consider a channel with the additive noise,

$$H(Y|X)=\frac{\Delta Q_0}{kT_W}\cdot\frac{T_0}{T_0}=\frac{\Delta Q_0}{kT_0}\cdot\beta=\frac{\Delta Q_W}{kT_W}\cdot\beta=H(Y)\cdot\beta,\beta=\frac{T_0}{T_w} \quad \text{Eq 2.12}$$

is to be valid. The noise with information entropy $H(Y|X)$ is an integral part of the definition of the transfer information process [6]. It is not generated by a positive production of the noise heat $\Delta Q_{0x}>0$ in the working medium L[5].

We are supposing further that for changes of information at the values $H(X), H(Y|X), H(Y), H(X|Y)$ defined by (2.11) the relations (2.3) are valid and then,

$$\frac{\Delta A}{kT_W}-H(X|Y)=\frac{\Delta Q_0+\Delta A}{kT_W}-\frac{\Delta Q_0}{kT_W} \quad \text{Eq 2.13}$$

$$H(X|Y)\overset{\Rightarrow}{\underset{=}{Def}}0.$$

So, we have a channel without losses. For transinformations $T(X;Y),T(Y;X)$, with respecting the definition (2.11) it is valid, within one run of the system $L\cong K$ through the reverse Carnot Cycle realizing a transfer process, that

$$T(X;Y)=H(X)-H(X|Y)=\frac{\Delta A}{kT_W}-0=H(X) \quad \text{Eq 2.14}$$

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

Consequently

$$H(X)=H(Y)\cdot\eta_{\max} \quad \text{Eq 2.15}$$

where $\eta_{\max}$ is the efficiency of the relevant direct cycle. So, it is valid, in accordance with relations (2.12) and (2.3), that

$$H(X)=T(X;Y)=H(Y)\cdot\eta_{\max} \quad \text{Eq 2.16}$$

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

$$\Delta S_{AB}=\frac{-\Delta Q_0}{T_0}+\frac{\Delta Q_0}{T_W}=\frac{-\Delta Q_0}{T_0T_W}\cdot(T_W-T_0)= \quad \text{Eq 2.17}$$

$$=\frac{-\Delta Q_0}{T_0}\cdot\eta_{\max}=-H(Y)\cdot\eta_{\max}<0$$

The thermodynamic (Clausius) entropy ΔS_{AB} of the system (AB) is then lowering its value - the (thermodynamic, heat) distinguishability of the systems A and B is growing up. Of course, it is at a detriment of the mechanical work _A delivered, or, respectively, at a detriment of the entropy at the value $\frac{\Delta A}{T_W}$.

But there is a need to gain this energy (negative entropy _ow) 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.

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 $\left(T^*_W\geq T_W \text{ and } T^*_0\leq T_0\right)$;

$$\frac{\Delta A^*}{kT^*_W}=H(X^*)\cdot\eta_{\max}{}^*=H(X^*)\cdot\frac{(T^*_W-T^*_0)}{T^*_W} \quad \text{Eq 2.18}$$

$$=\frac{1}{k}\Delta S_{A^*B^*},T^*_W\geq T^*_0>0,$$

$$\frac{\Delta A}{kT_W}=H(Y)\cdot\eta_{\max}=H(Y)\cdot\frac{(T_W-T_0)}{T_W}$$

$$=-\frac{1}{k}\Delta S_{AB},T_W\geq T_0>0$$

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

$$\Delta S=\Delta S_{A^*B^*}+\Delta S_{AB}\geq 0 \quad \text{Eq 2.19}$$

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

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$$\Delta S_{A^*B^*} \geq \left|\Delta S_{AB}\right| \quad \text{Eq 2.20}$$

This means that for the decrease of entropy about the value $\left|\Delta S_{AB}\right|$, the greater addition of $\Delta S_{A^*B^*}$ is to be generated, and then, the whole [7] entropy is growing up just about the result value ΔS.

$$\Delta S = \Delta S_{A^*B^*} - \left|\Delta S_{AB}\right| \geq 0 \quad \text{Eq 2.21}$$

The equality occurs when $\eta_{\max}{}^{*} = \eta_{\max}$. In another case $\eta_{\max}{}^{*} > \eta_{\max}$ which, e.g. for $T_{0^*} = T_0$ means that

$$\Delta Q_W^* > \Delta Q_W \quad \text{and} \quad T_W{}^{*} > T_W \quad \text{Eq 2.22}$$

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, than the local growing up of the order or organization in (AB) is.

Biology Analogy for Cell Generation

We can envisage and suppose further that the heat engines are, in a certain way, the real physical background (let us say a motor) for the cells' duplications. Or, that the system (AB) models the cell and the temperature $T_W{}^{*}$, T_W and T_0 have, in this case, the biological meaning of the cells and the cells' environment properties affecting their generation or, in the pathological case, also the stability and development of their structure (and their metabolism).

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 or communication ability). 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 being driven by our metabolism) [8] the less output (average) information is gained in comparison with the input (average) information (2.2).

$$H(Y) = \Delta I < H(X) \quad \text{Eq 3.23}$$

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

$$T_{W,i+1} < T_{W,i}, T_{0,i+1} = T_{0_i} = T_0, i \geq 1; T_{W,i} \triangleq T_W \quad \text{Eq 3.24}$$

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 (2.10).

$$\Delta S_C > 0 \quad \text{Eq 3.25}$$

In this model it is the less and less distinguishability within the system (AB) [of parts A and 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 (3.23), 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 efficiency of the transformation of the input energy.

$$\eta_{\max} < 1 \quad \text{Eq 3.26}$$

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 [9] if the cells are equipped with them. "Candle of life is burning out", of course in any case.

$$l \cong f\left(S^+ - \Delta S_{AB}\right) < f\left(S^+\right), \underline{\Delta S_{AB} > 0} \quad \text{Eq 3.27}$$

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

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 (AB) within those two cycles] with a more precise structure (but, of another type in comparison with the normal structure of its own original and 'normal' type). But this growth of structure in a certain locality in the whole organism is paid by a pumping o_ energy from an environment of this locality (from the rest sound parts of the organism-body, from the environment of the 'youngering' locality).

This environment is then in a lack of energy for its normal functionality; this energy is consummated in the favor of this local grow of structure signalized by that phenomenon that the followers of the predecessors have the telomeres lengthened, see [9], if the cells are equipped with them. The cell itself is as young as for is real and the measure of its (but bad) structure.

$$l \cong f\left(S^+ - \Delta S_{AB}\right) < f\left(S^+\right), \underline{\Delta S_{AB} < 0} \quad \text{Eq 3.28}$$

where $f(\bullet)$ 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 (in the sense of the greater distinguishability of parts A and B is evidenced (2.17).

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$$\underline{\Delta S_{AB} = -kH(Y)\cdot\eta_{\max} < 0,\ H(Y) > 0} \quad \text{Eq 3.29}$$

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 such a locality, for the cancer cell.

However, for this decrease of entropy (for this growth of structure) the energy is needed at the value (2.18) but, delivered from the environment (2.20) of this locality [the model for the exhausting the rest sound part of the body].

$$\underline{\Delta A^* > 0,}\ \ \Delta S_{A^*B^*} > 0 \quad \text{Eq 3.30}$$

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, see (2.19). In the case of cells (organism-body) We can see the wasting away of the whole organism. The final equilibrium is approaching again, and faster, just for the value $\Delta S_{A^*B^*}$ is valid that $\Delta S_{A^*B^*} > \Delta S_C > 0$ where $\Delta S_C = \Delta S_{AB} > 0$ in the normal case [10]. Proposingly it is, e.g., by a certain growing and positive function $f(\cdot)$ of $\Delta S^{\dagger}$ [exponential, power-polynomial function $f(\cdot)$ is await able].

$$\Delta S^{\dagger} \underset{=}{?} \Delta S_{A^*B^*} = \Delta S + \left|\Delta S_{AB}\right| > 0, \Delta S_{AB} < 0 \quad \text{Eq 3.31}$$

[where the 'formal' or mathematical grow of entropy of the whole (wider) system-organism is $\underline{\Delta S}$ from (2.19), (2.21)] or, possibly more realistic is,

$$\Delta S^{\dagger} \underset{=}{?} \Delta S_{A^*B^*} + \Delta S_{A'B'} > 0, \Delta S_{A'B'} > 0 \quad \text{Eq 3.32}$$

where $\Delta S_{A'B'}$ is the entropy growth adequate to the cell of the sound texture, Our reverse Information-Thermodynamic model, see (2.18)-(2.22), authorizes us to an awaiting of a stable (moderate) higher body temperature $T^*_{W_{body}}$ of a patient against the normal $T^*_{W_{normal}}$ or drifting the body temperature with a higher average value[6] indicated by our higher model temperature T^*_W and, also, within the model, by a less model temperature T_W of the problematic texture[7].

$$T^*_W > \mathrm{T}_W \quad \text{Eq 3.33}$$

Considering the body's or the tumor's environment temperature $T^*_{W_{body}}$ modeled by our T^*_W and with regards to the possible relation between the model and enzymatic 'driven' processes, see the Remark 7, should be $T^*_{W_{body}} < T^*_W$ in the body's reality. And now, within the model the T_W represents the measure of the structure of the tumor place growing in time.

$$\mathrm{T}_W = \mathrm{T}_{W,n} > \mathrm{T}_{W,i+1} > \mathrm{T}_{W,i} > \cdots > \mathrm{T}_{W,0},\ T^*_{W_{body,i}} > T^*_{tumor,i} \cong T_{W,i} \quad \text{Eq 3.34}$$

So, the temperatures $T_{W,i}$, $T_{W,i+1}$.... indicate the warming of A in our double-machine engine just as the model of growing structuring of the tumor place[8] within the body in times $i, 0 \le i \le n-2$, and being modeled just by the amount of heat concentrated in the A against the B of the driven machine.

Supposingly, a negative entropy ow across the problematic place is required from the beginning,

$$T^*_{W_{body,i}} > T^*_{W_{normal}} > T^*_{tumor,0} \cong T_{W,0} \quad \text{Eq 3.35}$$

Conclusion

For the pathology proliferating texture is consummating the energy from the rest of the organism it is logical, as for the therapy, to try to cut of this energy supply. Following the previous considerations, we can imagine the lowering of the tumor'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 limit[9] at least for a short time, the positive few of energy and heat (the heat at the temperature T^*_W in our model) from the sound texture, otherwise supporting the cell generation and metabolic processes in such problematic locality. The lowering of the body or the tumor's environment temperature also means the weakening the body metabolism nutricing the tumor (the lowering the temperature gradient from the sound to the ill texture by our model). [10]May be that the antipyretical effects of the Salicylic Acid (Aspirin) are the part of preventive effects having been evidenced.

[6]Similar with body's reactions to higher temperatures in case of more frequent viroses?

[7]These temperatures are those of our thermodynamic model, they are physical analogue for the cause of the same efficiency as it is in the real biological process. The values of the body's temperatures will be different (for both the normal and the pathological case) due to the influence of enzymes' functionality.

[8]Also mixed with the temperature from its own metabolism (?) and with consideration of Remark 7.

[9]Nowadays it seems to be proved that the cancer tumor 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 methods is cryotherapy used for the breast cancer: within the environment of the tumor the freezing gas is injected with the result of the tumor's diminishing. From the point of view of the energy consumption also the real surgery method of devitalization which is narrowing the connection between the pathology tumor and its environment [MUDr. Karel Fortýn, CSc., Ústav zivocisne fyziologie a genetiky AV CR v Libechove, 1957, 1971-2001] seems to be a right way.

[10]Even in the case of the cure where the 'impuls' of higher temperature to this locality is used (e.g., the socrum case), the inevitable successive process of natural cooling is imaginable, creating the appropriate negative temperature gradient from the tumor to its environment - the plain physical result should be the same as it is for cooling its environment.

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We can imagine this situation: Supposingly, the occurrence of the pathological cell is indicated as the consequence 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 $T^*_{W_{body}}$ expressed by our model temperature T^*_W . 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 supposingly supporting the energy delivery to the problematic place - let us say that it is a thermodynamic explanation why the immunology of the body fails.

Our Information-Thermodynamic approach seems to be near to the reality. Our model equations (3.27), (3.28) and (3.31)-(3.34) should be general description for the cells' structure changes whether or not the cells are equipped with telomeres.

Let us repeat, in free words, the main idea and motivation of this paper. The cancer cell reveals itself as the younger one, more (but badly) structured, than the sound cell of the adequate age of the body could be. The older and sound cells are less (but naturally) structured. In special case of the cells their actual age is measured by the length of their telomeres. Nevertheless, the cancer cells are as the younger ones independently of their equipment by telomeres.

In general, the younger age of the normal cells is describable by their higher structuring organization, Their older age reveals them as more disorganized [11]. When the process of the cell's youngering is started it means the local lowering of the natural degeneration and growing its inner organization-structure. It is the local degression of its entropy in the sense of lowering the degree of its disorganization - in the direction toward the greater degree of its structural (even bad) ordering. This decrease of entropy (as the measure of disorder) or, the building a structure (in fact it does not matter if not desirable, generally said) needs the delivery of energy from the environment of this phenomenon (like mafia organization in the case of cancer). Model of expressing this my thinking is based on information (message/structure) transfer expressed by the thermodynamics language.

For the nutrition of the problematic place-cell a certain gradient of energy toward this place is needed. In my double-machine engine model it is he temperature gradient. This gradient should be, I believe, in the very base of all processes of structure's building, including the biological ones, ensuring this energy transfer and driving it.

My proposition following from this point of view is to cut for this process needed logistics, its motor and fuel. So, let us try cooling the environment of the cancer cell. I suppose that the body reacts just by the higher temperature when the cancer cells are occurring. And which higher temperature is that fuel and motor for the higher energy delivery or nutrition of the cell. The higher body temperature is common body's weapon initializing the immune system, but now it is possible that this higher temperature is just that what the cancer structure requires. It is positive back-_re and cycle. So let us cool the environment of the cancer place (or the whole body?).

The experiment should be this:

We take samples of the ill place but with the environmental texture which is sound (and using nutrition from an adequate lotion). We can have three samples. One sample will be maintained in the normal temperature of body as for the sound environmental texture. The second sample will be used with lightly higher temperature of this environmental texture. The third and the most important sample will have lightly lower temperature of the environmental texture (as under cold body as maximum). For the needed temperatures should be used a tube ring or spiral laid under the environmental texture only and with the cancer place in the center of this ring or spiral.

When I am right, we could see the disintegration of the cancer cells in the cooled sample, the others are the control or check ones _ nothing could happen or the growing could happen. Or nothing could happen in all three cases. I do not know. I want the cancer place to be more and more hungry just by it being less and less nutrited. I suppose the nutrition gradient is dependent on the temperature gradient streaming originally to the cancer place and now this gradient is to be or should be reverted by the environmental cooling. It means the cancer's

place devitalization.

The experiment is based on my thermodynamic consideration or _philosophy_ which in fact is neglecting the complexity of the whole cancer phenomenon. But all the complicated chemical and biological processes are, in their very base, driven or influenced just by temperature and the temperature of the body is now its weapon but now used by its inner enemy growing. This experiment is, as it is proposed, the global one with the possible global results. May be that after its possible results the detailed study of the very complicated structure and functionality of the parts of the cancer cell could be possible with so far missing view will have been gained

this way? Let us believe.

The results of experiment has been drafted above could give some initial data to be used in FVSOOM and CHOOM [12] procedures and help for chemotherapy considerations, e.g. [13]. May be?

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