

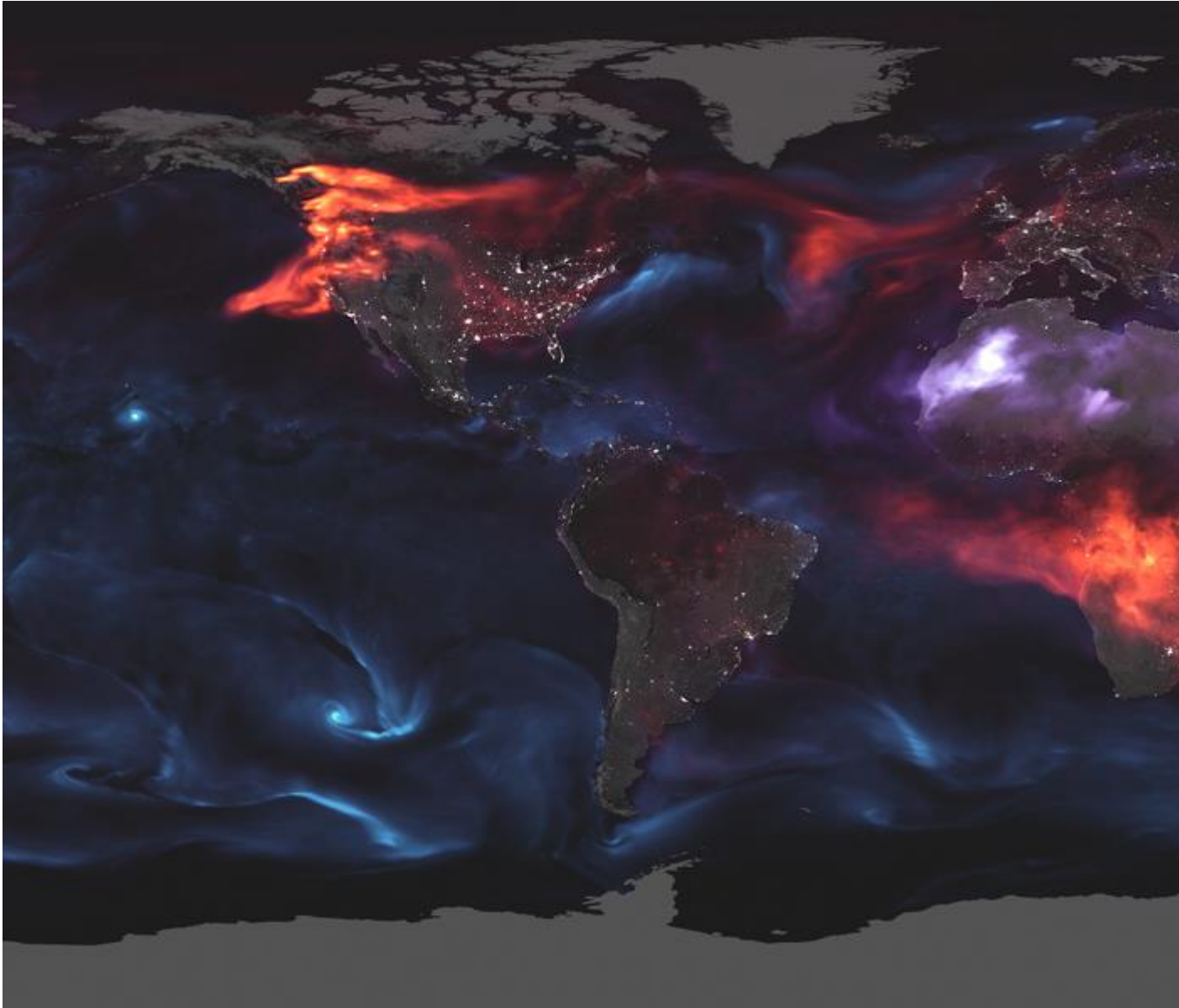
INJURED EARTH

Mirosław Kozłowski

Warsaw University, Warsaw, Poland

Abstract

In the first part of the INJURED EARTH (Mirosław Kozłowski, RG 2020) we presented experimental data (measurement) of the CoV-19 pandemic. The simultaneously in quite different part of the Earth atmosphere the blast of virus pandemic was noted, There is no possibility of the propagation of the pandemic through individual contacts. My fundamental thesis is that droplet of viruses pandemic are in the clouds(not weather clouds) but different aerosols clouds. On the figure beneath we see the different types of the aerosols clouds, especially over USA terrains The movement of clouds over terrains of different parts of Earth's atmosphere is the source of Pandemia./ The picture of pandemic propagation resembles the propagation of radioactive clouds from Chernobyl radioactive last For the moment we do not know the dimensions of Covid-19 clouds and its stability and lifetime. In my papers I presented the mathematical model for aerosols of COVID INTERACTIONS WITH OUR LUNGS. We are breathing the atmosphere air with Covid aerosols



Aerosol Earth

Model Visualization Credit: [NASA Earth Observatory](#), [GEOS FP](#), Joshua Stevens

Explanation: [For August 23, 2018](#), the identification and distribution of [aerosols](#) in the Earth's atmosphere is shown in this dramatic, planet-wide visualization.

Produced in real time, the Goddard Earth Observing System Forward Processing ([GEOS FP](#)) model relies on a combination of [Earth-observing](#) satellite and ground-based data to calculate the presence of types of aerosols, tiny solid particles and liquid droplets, as they circulate above the entire planet. This August 23rd model shows black carbon particles in red from combustion processes, like smoke from the fires in the United States and Canada, spreading across large stretches of North America and Africa. Sea salt aerosols are in blue, swirling above threatening typhoons near South Korea and Japan, and the hurricane looming near Hawaii. Dust shown in purple hues is blowing over African and Asian deserts. The location

of cities and towns can be found from the concentrations of lights based on satellite image data of the [Earth at night](#).

1 Mathematical model of CoV propagation

In this paper the mathematical model for CoV propagation, based on Boltzmann type equation is formulated. The CoV growth factor is defined . The host cells density is calculated. It is shown that the CoV evolution strongly depends on the growth factor k . For $k < 0.5$ CoV density oscillate and virus is in the “hesitate” state. For $k > 0.5$ CoV lost the oscillatory character and grows abruptly and emits to the host body. We argue that the oscillation of the density of CoV creates the CoV waves which can be coined as the CoV waves..

Since 2020, CoV has become the leading cause of death for humans between the ages of 40 and 74). But the overall effectiveness of CoV therapeutic treatments is only 50%. Understanding the CoV biology and developing a prognostic tool could therefore have immediate impact on the lives of millions of people diagnosed with CoV . There is growing recognition that achieving an integrative understanding of molecules, cells, tissues and organs is the next major frontier of biomedical science. Because of the inherent complexity of real biological systems, the development and analysis of computational models based directly on experimental data is necessary to achieve this understanding.

CoV development is very complex and dynamic. Primary malignant CoV patients arise from small nodes of cells that have lost, or ceased to respond to, normal growth regulatory mechanisms, through mutations and/or altered gene expression , This genetic instability causes continued malignant alterations, resulting in a biologically complex

Physicists have long been at the forefront of cancer diagnosis and treatment, having pioneered the use of X rays and radiation therapy. In the contemporary initiative, the US National Cancer Institute the conviction that physicists bring unique conceptual insights that could augment the more traditional approaches to cancer research is very appealing.

In this paper we present the first attempt to consider the CoV propagation as the physical medium with some sort of memory.

Let us consider the one-dimensional transport “particles”, .CoV. These viruses however may move only to the right or to the left on the rod. Moving CoVs may interact with the fixed host body cells the probabilities of such collisions and their expected results being specified. All particles will be of the same kind, with the same energy and other physical specifications distinguishable only by their direction.

Let us define:

$u(z,t)$ = expected density of CoV at z and at time t moving to the right,

$v(z,t)$ = expected density of CoV at z and at time t moving to the left.

Furthermore, let

$\delta(z)$ = probability of collision occurring between a fixed scattering centrum and a cell moving between z and $z + \Delta$.

Suppose that a collision might result in the disappearance of the moving CoV without new items appearing. Such a phenomenon is called *absorption*. Or the moving particle may be reversed in direction or back-scattered. We shall agree that in each collision at z an expected total of $F(z)$ cells arises moving in the direction of the original cell, $B(z)$ arise going in the opposite direction.

The expected total number of right-moving CoV $z_1 \leq z \leq z_2$ at time t is

$$\int_{z_1}^{z_2} u(z,t) dz, \quad (2.6)$$

while the total number of CoV passing z to the right in the time interval $t_1 \leq t \leq t_2$ is

$$w \int_{t_1}^{t_2} u(z,t) dt, \quad (2.7)$$

where w is the CoV speed.

Consider the cell moving to the right and passing $z + \Delta$ in the time interval $t_1 + \frac{\Delta}{w} \leq t \leq t_2 + \frac{\Delta}{w}$:

$$w \int_{t_1 + \frac{\Delta}{w}}^{t_2 + \frac{\Delta}{w}} u(z + \Delta, t') dt' = w \int_{t_1}^{t_2} u\left(z + \Delta, t' + \frac{\Delta}{w}\right) dt'. \quad (2.8)$$

These can arise from cells which passed z in the time interval $t_1 \leq t \leq t_2$ and came through ($z, z + \Delta$) without collision

$$w \int_{t_1}^{t_2} (1 - \Delta \delta(z, t')) u(z, t') dt' \quad (2.9)$$

plus contributions from collisions in the interval $(z, z + \Delta)$. The right-moving cells interacting in $(z, z + \Delta)$ produce in the time t_1 to t_2 ,

$$w \int_{t_1}^{t_2} \Delta \delta(z, t') F(z, t') u(z, t') dt' \quad (2.10)$$

cells to the right, while the left moving ones give:

$$w \int_{t_1}^{t_2} \Delta \delta(z, t') B(z, t') v(z, t') dt'. \quad (2.11)$$

Thus

$$\begin{aligned} w \int_{t_1}^{t_2} u\left(z + \Delta, t' + \frac{\Delta}{w}\right) dt' &= w \int_{t_1}^{t_2} u(z, t') dt' + w \Delta \int_{t_1}^{t_2} \delta(z, t') (F(z, t') - 1) u(z, t') dt' \\ &\quad + w \Delta \int_{t_1}^{t_2} \delta(z, t') B(z, t') v(z, t') dt'. \end{aligned} \quad (2.12)$$

Now, we can write:

$$u\left(z + \Delta, t' + \frac{\Delta}{w}\right) = u(z, t') + \left(\frac{\partial u}{\partial z}(z, t') + \frac{1}{w} \frac{\partial u}{\partial t}(z, t') \right) \Delta \quad (2.13)$$

to get

$$\int_{t_1}^{t_2} \left(\frac{\partial u}{\partial z}(z, t') + \frac{1}{w} \frac{\partial u}{\partial t}(z, t') \right) dt' = \int_{t_1}^{t_2} \delta(z, t') ((F(z, t') - 1) u(z, t') + B(z, t') v(z, t')) dt'. \quad (2.14)$$

On letting $\Delta \rightarrow 0$ and differentiating with respect to t_2 we find

$$\frac{\partial u}{\partial z} + \frac{1}{w} \frac{\partial u}{\partial t} = \delta(z, t) (F(z, t) - 1) u(z, t) + \delta(z, t) B(z, t) v(z, t). \quad (2.15)$$

In a like manner

$$-\frac{\partial v}{\partial z} + \frac{1}{w} \frac{\partial v}{\partial t} = \delta(z, t) B(z, t) u(z, t) + \delta(z, t) (F(z, t) - 1) v(z, t). \quad (2.16)$$

The system of partial differential equations of hyperbolic type (2.15, 2.16) is the Boltzmann equation for one dimensional transport phenomena (Kozłowski, Marciak-Kozłowska, 2009)

Let us define the total density for CoV, $\rho(z, t)$

$$\rho(z, t) = u(z, t) + v(z, t) \quad (2.17)$$

and density of cells current

$$j(z,t) = w(u(z,t) - v(z,t)). \quad (2.18)$$

Considering equations (2.15 – 2.18) one obtains

$$\frac{\partial \rho}{\partial z} + \frac{1}{w^2} \frac{\partial j}{\partial t} = \delta(z,t)u(z,t)(F(z,t) - B(z,t) - 1) + \delta(z,t)v(z,t)(B(z,t) - F(z,t) + 1). \quad (2.19)$$

Equation (2.19) can be written as

$$\frac{\partial \rho}{\partial z} + \frac{1}{w^2} \frac{\partial j}{\partial t} = \frac{\delta(z,t)(F(z,t) - B(z,t) - 1)j}{w} \quad (2.20)$$

or

$$j = \frac{w}{\delta(z,t)(F(z,t) - B(z,t) - 1)} \frac{\partial \rho}{\partial z} + \frac{1}{w\delta(z,t)(F(z,t) - B(z,t) - 1)} \frac{\partial j}{\partial t}. \quad (2.21)$$

Denoting, D , diffusion coefficient

$$D = -\frac{w}{\delta(z,t)(F(z,t) - B(z,t) - 1)}$$

and τ , relaxation time

$$\tau = \frac{1}{w\delta(z,t)(1 - F(z,t) - B(z,t))} \quad (2.22)$$

equation (2.21) takes the form

$$j = -D \frac{\partial \rho}{\partial z} - \tau \frac{\partial j}{\partial t}. \quad (2.23)$$

Equation (2.23) is the Cattaneo's type equation and is the generalization of the Fourier equation (Kozłowski, Marciak-Kozłowska, 2009). Now in a like manner we obtain from equation (2.15 – 2.18)

$$\frac{1}{w} \frac{\partial j}{\partial z} + \frac{1}{w} \frac{\partial \rho}{\partial t} = \delta(z,t)u(z,t)(F(z,t) - 1 + B(z,t)) + \delta(z,t)v(z,t)(B(z,t) + F(z,t) - 1) \quad (2.24)$$

or

$$\frac{\partial j}{\partial z} + \frac{\partial \rho}{\partial t} = 0. \quad (2.25)$$

Equation (2.25) describes the conservation of cells in the transport processes.

Considering equations (2.23) and (2.25) for the constant D and τ the hyperbolic Heaviside equation is obtained:

$$\tau \frac{\partial^2 \rho}{\partial t^2} + \frac{\partial \rho}{\partial t} = D \frac{\partial^2 \rho}{\partial z^2}. \quad (2.26)$$

where τ is the relaxation time

In the stationary state transport phenomena $dF(z,t)/dt = dB(z,t)dt = 0$ and $d\delta(z,t)/dt = 0$. In that case we denote $F(z,t) = F(z) = B(z,t) = B(z) = k(z)$ and equation (2.10) and (2.11) can be written as

$$\begin{aligned}\frac{du}{dz} &= \delta(z)(k-1)u(z) + \delta(z)kv(z), \\ -\frac{dv}{dz} &= \delta(z)k(z)u(z) + \delta(z)(k(z)-1)v(z)\end{aligned}\tag{2.27}$$

with diffusion coefficient

$$D = \frac{w}{\delta(z)}\tag{2.28}$$

and relaxation time

$$\tau(z) = \frac{1}{w\delta(z)(1-2k(z))}.\tag{2.29}$$

The system of equations (2.27) can be written as

$$\frac{d^2u}{dz^2} - \frac{d}{dz} \left(\frac{\delta k}{\delta k} \right) \frac{du}{dz} + u \left[\delta^2(2k-1) + \frac{d\delta}{dz}(1-k) + \frac{\delta(k-1)}{\delta k} \frac{d(\delta k)}{dz} \right] = 0,\tag{2.30}$$

$$\frac{du}{dz} = \delta(k-1)u + \delta kv(z).\tag{2.31}$$

Equation (2.30) after differentiation has the form

$$\frac{d^2u}{dz^2} + f(z) \frac{du}{dz} + g(z)u(z) = 0,\tag{2.32}$$

where

$$f(z) = -\frac{1}{\delta} \left(\frac{\delta}{k} \frac{dk}{dz} + \frac{d\delta}{dz} \right),\tag{2.33}$$

$$g(z) = \delta^2(z)(2k-1) - \frac{\delta}{k} \frac{dk}{dz}.$$

For the constant absorption rate we put

$$k(z) = k = \text{constant} \neq \frac{1}{2}.$$

In that case

$$f(z) = -\frac{1}{\delta} \frac{d\delta}{dz},\tag{2.34}$$

$$g(z) = \delta^2(z)(zk-1).$$

With functions $f(z)$ and $g(z)$ the general solution of the equation (2.30) has the form

$$u(z) = C_1 e^{(1-2k)^{1/2} \int \delta dz} + C_2 e^{-(1-2k)^{1/2} \int \delta dz}. \quad (2.35)$$

In the subsequent we will consider the solution of the equation (2.32) with $f(z)$ and $g(z)$ described by (2.34) for Cauchy condition:

$$u(0) = q, \quad v(a) = 0. \quad (2.36)$$

Boundary condition (2.36) describes the generation of the heat carriers (by illuminating the left end of the strand with laser pulses) with velocity q heat carrier per second.

The solution has the form:

$$u(z) = \frac{2qe^{[f(0)-f(a)]}}{1 + \beta e^{2[f(0)-f(a)]}} \left[\frac{(1-2k)^{\frac{1}{2}}}{(1-2k)^{\frac{1}{2}} - (k-1)} \cosh[f(x) - f(a)] \right. \\ \left. + \frac{k-1}{(1-2k)^{\frac{1}{2}} - (k-1)} \sinh[f(x) - f(a)] \right], \quad (2.37)$$

$$u(z) = \frac{2qe^{(f(0)-f(a))}}{1 + \beta e^{2[f(0)-f(a)]}} \left[\frac{(1-2k)^{\frac{1}{2}} + (k-1)}{k} \sinh[f(x) - f(a)] \right],$$

where

$$f(z) = (1-2k)^{\frac{1}{2}} \int \delta dz, \\ f(0) = (1-2k)^{\frac{1}{2}} \left[\int \delta dz \right]_0, \\ f(a) = (1-2k)^{\frac{1}{2}} \left[\int \delta dz \right]_a, \quad (2.38) \\ \beta = \frac{(1-2k)^{\frac{1}{2}} + (k-1)}{(1-2k)^{\frac{1}{2}} - (k-1)}.$$

Considering formulae (2.17), (2.18) and (2.37) we obtain for the density, $\rho(z)$ and current density $j(z)$.

$$j(z) = \frac{2qwe^{[f(0)-f(a)]}}{1 + \beta e^{2[f(0)-f(a)]}} \left[\frac{(1-2k)^{\frac{1}{2}}}{(1-2k)^{\frac{1}{2}} - (k-1)} \cosh[f(z) - f(a)] \right. \\ \left. - \frac{1-2k}{(1-2k)^{\frac{1}{2}} - (k-1)} \sinh[f(z) - f(a)] \right] \quad (2.39)$$

and

$$q = \frac{2qe^{[f(0)-f(a)]}}{1 + \beta e^{2[f(0)-f(a)]}} \left[\begin{array}{l} \frac{(1-2k)^{\frac{1}{2}}}{(1-2k)^{\frac{1}{2}} - (k-1)} \cosh[f(z) - f(a)] \\ - \frac{1}{(1-2k)^{\frac{1}{2}} - (k-1)} \sinh[f(z) - f(a)] \end{array} \right]. \quad (2.40)$$

Equations (2.39) and (2.40) fulfill the generalized Fourier relation

$$j = -\frac{w}{\delta(z)} \frac{\partial \rho}{\partial z}, \quad D = \frac{W}{\delta(z)}, \quad (2.41)$$

where D denotes the diffusion coefficient.

Analogously we define the generalized diffusion velocity $v_D(z)$

$$v_D(z) = \frac{j(z)}{n(z)} = \frac{w(1-2k)^{\frac{1}{2}} \left[\cosh[f(z) - f(a)] - (1-2k)^{\frac{1}{2}} \sinh[f(x) - f(a)] \right]}{(1-2k)^{\frac{1}{2}} \cosh[f(x) - f(a)] - \sinh[f(x) - f(a)]}. \quad (2.42)$$

Assuming constant cross section for CoV scattering $\delta(z) = \delta_o$ we obtain from formula (2.38)

$$\begin{aligned} f(z) &= (1-2k)^{\frac{1}{2}} z, \\ f(0) &= 0, \\ f(a) &= (1-2k)^{\frac{1}{2}} a \end{aligned} \quad (2.43)$$

and for density $\rho(z)$ and current density $j(z)$

$$j(z) = \frac{2qwe^{-(1-2k)^{\frac{1}{2}} a \delta}}{1 + \beta e^{-(1-2k)^{\frac{1}{2}} a \delta}} \left[\begin{array}{l} \frac{(1-2k)^{\frac{1}{2}}}{(1-2k)^{\frac{1}{2}} - (k-1)} \cosh \left[(2k-1)^{\frac{1}{2}} (x-a) \delta \right] \\ - \frac{(1-2k)}{(1-2k)^{\frac{1}{2}} - (k-1)} \sinh \left[(2k-1)^{\frac{1}{2}} (x-a) \delta \right] \end{array} \right], \quad (2.44)$$

$$\rho(z) = \frac{2qe^{-(1-2k)^{\frac{1}{2}} a \delta}}{1 + \beta e^{-(1-2k)^{\frac{1}{2}} a \delta}} \left[\begin{array}{l} \frac{(1-2k)^{\frac{1}{2}}}{(1-2k)^{\frac{1}{2}} - (k-1)} \cosh \left[(2k-1)^{\frac{1}{2}} (x-a) \delta \right] \\ - \frac{1}{(1-2k)^{\frac{1}{2}} - (k-1)} \sinh \left[(2k-1)^{\frac{1}{2}} (x-a) \delta \right] \end{array} \right]. \quad (2.45)$$

Formulae (2.44) and (2.45) describe the kinetic of the growth of the CoV aggregation. The development of the CoV strongly depends on the coefficient k . In the following we will call k -the growth coefficient. For $k < 0.5$ the density of the cell oscillate, Fig.1 a, 2 a. On the other hand for $k > 0.5$ the cell density grows exponentially, Fig. 2 a, 2 b.

For $k < 0.5$ the CoV aggregation emits the wave with length $\lambda =$ size of the tumor. For $k = 0.5$ the cancer development has a cusp. Fig1 a. For $k = 1.91$ density of the tumor cells has a singularity. For $k < 0.5$ the density of the cell oscillate, Fig.2 a, a. On the other hand for $k > 0.5$ the cell density grows exponentially, Fig. 2 b.

The first stage $k < 0.5$ we will call the “hesitation” period in which tumor send the “information” waves to the host body. The response of the host depends on the willing to cooperate with CoV . For $k < 0.5$ the response of the host is *negative* and CoV density is stable. For $k > 0.5$ the *angiogenesis* starts – the host cooperates with CoV and density grows abruptly

It seems that the first “hesitation” stage is the exchange the information tumor \rightarrow host \rightarrow tumor and *vice versa*. Next , through the singularity point $k = 1.9$, for $x = 3 \text{ um}$ the cancer obtain the information, *go* and *metastasis* process starts.

3. Conclusions

In this paper we argue that the CoV aggregates in host evolution can be described as the process which strongly depends on the growth factor k , defined in the paper. For $k < 0.5$ agregatesr is stable with oscillatory behavior of the CoV density. For $k > 0.5$ the density grows exponentially. For the moment the CoVr wave emission was not observed. It seems that the observation of the emitted waves can be important therapeutic tool for the description of the CoVstatus. The stop of the emission of the waves is the signature of the invasive evolution of the CoV. In that case we can anticipate the correlation of the tumor growth and psychic of the host. It is interesting

to note that in paper by Erica K. Sloan and others (Sloan, 2010)] the role of the *neuroendocrine* activation in cancer propagation is described and investigated

References

- Jemal, A.:The Journal of the American Medical Association, 2005 ; 294 :1255–1259.
- Marusic, M., Bajzer Z, Freyer J.P., and Vuk-Pavlovic S: Analysis of growth of multicellular tumour spheroid by mathematical models. Cell Prolif. 1994; 27;73-78
- Kozlowski M . Marciak-Kozłowska J.. From femto- to attoscience and beyond, NOVA, USA, 2009
- Sloan Erica, K, et. al., Cancer Res, 2010; 70 (18) 7042-7052
- Sutherland, R.M. Cell and environment interactions in tumor microregions: the multicell spheroid model. Science, 1988; 240 :177–184.

Figure captions

Fig. 1 a Cells (CoV) density, formula (2.45) as the function of the growth factor k , for $x=3$ μm , $a=1$ μm . Fig.1 b , the same as in Fig 1 a but for $k>0.5$

Fig .2 a Cells (CoV) density , formula (2.45) as the function of x and growth factor k , for $k<0.5$, $a=10$ μm . Fig. 2 b the same as in Fig 2a but for $k>0.5$,

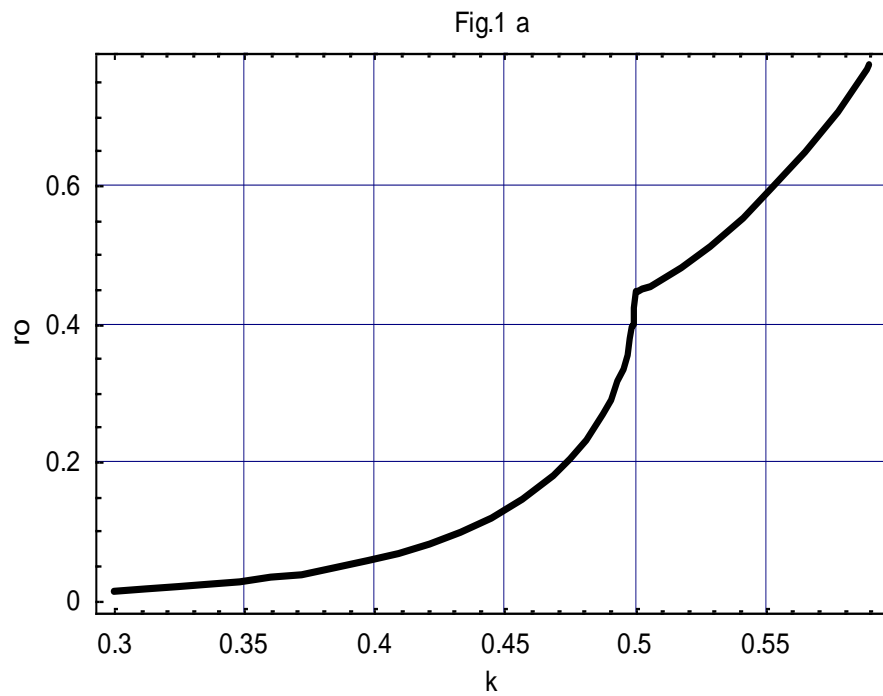


Fig 1b

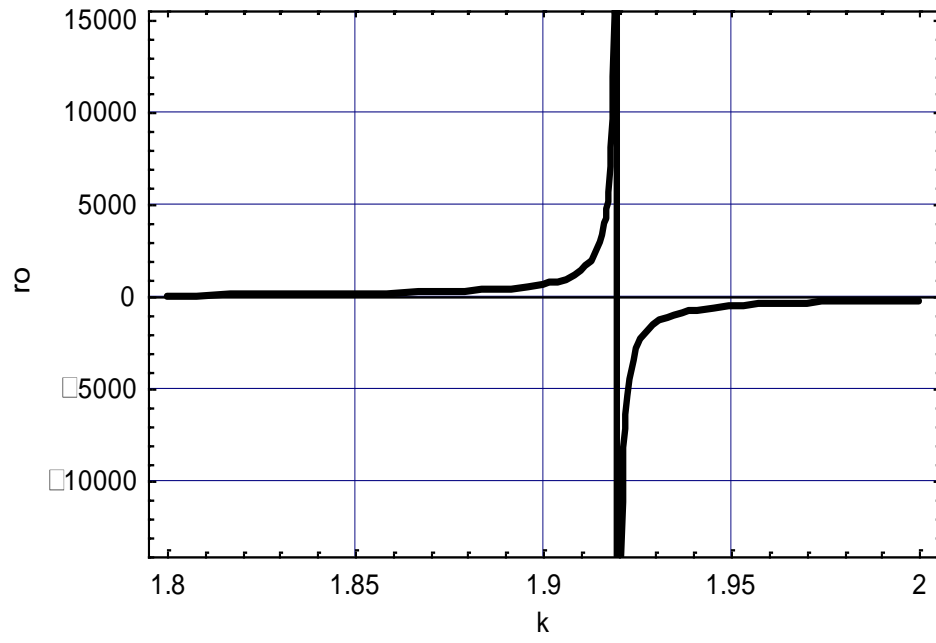


Fig.2a , $k < 0.5$, $\text{del} = 1/\mu\text{m}$

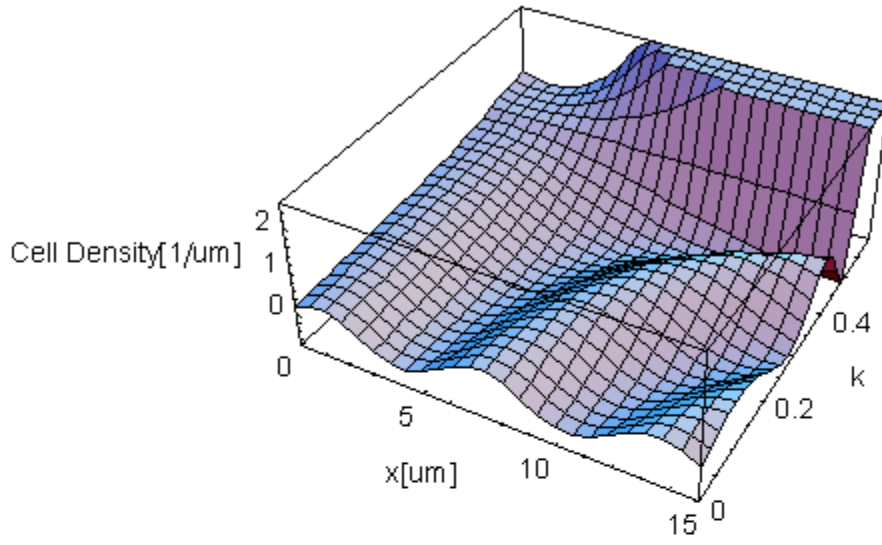


Fig2 b, $k > 0.5$, $\text{del} = 1/\mu\text{m}$

