A heuristic point of view on the breathing in the corona virus environment – The "Naïve Theory"

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It is an attempt to analyze the breathing (respiration) of an already ill, by the corona virus disease [1-9], person in simple equational terms.

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Corona Virus Disease; Human Behavior;

Breathing (Respiration); Balance Equation;

Medical Treatment, Some Heuristic Connections

1. Introduction

Consider the total number ***N*** of the **CVM** (**C**orona **V**irus **M**olecules) that present in the lung (thorax) of an ill human. The balance of *N*, -- either its constancy, or its tendency up or dawn, -- can be decisive as regards life or death. The roles of **inhalations** and **exhalations** of the respiration, are very distinct in this regard, because the averaged spatial concentrations, i.e. densities, of the CVM -- those inside the thorax, n, and those in the surrounding space, n_o, -- strongly differ. Since the human is ill

$$n >> n_o, \qquad (1)$$

and when assuming that there is a good ventilation in the room we can even set, for simplicity, $n_o = 0$.

Taking the CVM out of the lung, the exhalations strongly influence *N*, much stronger than the breaths.

The Balance Equation for N is, in its main terms

$$\frac{dN}{dt} = -(the\ \ flow\ out) + (the\ source\ inside), \qquad (2)$$

see Fig.1

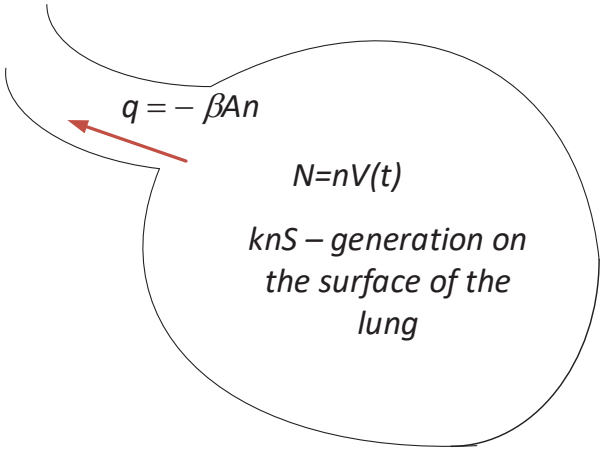

Fig 1. An illustration of the main terms of (2); "q" here is "q_{out}" in the main text. The "obvious" equality $N = nV$ appears to be a not simple, and very fruitful story.

Equation (2) readily obtains the form

$$\frac{dN}{dt} = -\beta An + kSn, \qquad (3)$$

where A is the *frequency of the breathing*, S is the area of the lung's surface, and β and k are some constants, such that their physical dimensions are $[\beta] = m^3$ and $[k] = \frac{m}{\sec}$.

That both terms in the right-hand side of (3) are directly proportional to n, starts a linear theory. Because of the relative smallness of the *outside* concentration n_o, the exhalation-flow *out*

$$q_{out} = -\beta An \qquad (4)$$

is much more significant than the breath-flow *inside*, $q_{in} = +\beta An_o$. That the velocity of each of the flows is directly proportional to A, is obvious.

The direct proportionality of the *source-term*

$$kSn \qquad (5)$$

with a constant k, to the lung's surface area S represents the assumption that the reproduction of the CVM – the essence of the illness -- takes place either on the whole surface, or on a certain known its part.

Writing (3) as

$$\frac{dN}{dt} = -Pn \ ,$$

we introduce the very important parameter

$$P(A) = \beta A - kS$$

for which we shall prove that $P \geq 0$, that is

$$A \geq \frac{kS}{\beta} \ , \qquad (6)$$

though we usually deal with the strict inequality $P > 0$, or

$$A > \frac{kS}{\beta} \ . \qquad (6a)$$

That is, there is a *threshold* for A, and if A is lower than the critical value

$$A_{cr} = \frac{kS}{\beta} \sim S ,$$

then one can die.

Considering A_{cr}, we observe in (6a) an important "competition" between A and S which are *mutually independent values*. This "competition" opens the way for many interesting conclusions. (Section 3)

We shall widely use the simple equality,

$$n = \frac{N}{V} \ , \qquad (7)$$

that seems to be undoubted, especially because we can simply measure V externally, via the respiration, that is, accept V as an empirically known, integral parameter. However, the question *what geometrically is "lung's volume"* V is very interesting and important. The

associated analysis that cannot be missed, reveals a reason for death that can be caused by CVM already in the thorax. The whole vision of our war against CV obtains the simple meaning of the competition of the oxygen molecules and those CVM -- namely **who will be the first coming to the free part of the lung's surface**.

2. CV and oxygen molecules, and the processes in the lungs

Physics is always deeply associated with geometry, and here the geometry is so complicated that, *from the analytical side* our use of *V* is, in some sense, not so routine, as a "conceptual" one.

The form of the lung is so complex, as if one has to pass from "Jordan Curve" (closed, without self-crossings, but of however complicated form, line in the plane, in some sense realizing the map 1D → 2D), on to "Jordan Surface" in the 3D space!

Figuratively speaking, the lung fills the thorax *with its surface* (2D → 3D) which is finite and has no self-crossings.

Besides the problematic sense of V, a not quite trivial point is that despite the very simple mutual connection, the methodological roles of N and n are very distinct. While the balance equation must be written for N, n is a much more suitable parameter for the physical considerations, and any thinkable measurement of CVM here is directly in terms of n. It is like the fact that humidity of the breathing can be measured via the exhalation, not via the never known whole amount of the water in the thorax.

In this sense, n here is a more fundamental, initial parameter than N.

On the physical regard, the complicated structure of the lung requires the complete breath to involve some *diffusion* [10] of the air into the twists (convolutions, slots) of the lung. It would be good if the ability to propagate (i.e. the value of the diffusion coefficient) would be higher for oxygen than for CVM. All the molecules in focus are electrically neutral, but if it would be possible to induce onto the large-size CVM some electrical dipole moment, then, using that dielectric materials are attracted by electrical field, one could try to establish, by the applied electrical field, some control over the movement of CVM.

The CVM, supplied by the infected air, and absorbed by the lung's surface, occupy this surface, and thus interfaces with the oxygen molecules to be absorbed. If the *amount*

of the absorbed oxygen molecules is *insufficient*, then the death comes.

Thus, *a death-causing problem can appear already on the surface of the lungs, as an interruption of the sufficient oxygen supply*.

Perhaps, most simply, the competition between the oxygen and the CV molecules in their attempt to come first to the lung's surface, can be interpreted -- for the oxygen -- as shooting onto a screen whose area is changed. More precisely, the free part of the screen is contracted in a random manner. Even if the smaller-size oxygen molecules could easier come to the narrow slots of the surface, the CVM can prevent them to do this, especially if they can cause some inflammation.

At this point, we can see how effective is it to give (especially to a weak man – see the next section) to breath the air with the increased oxygen supply. This must lead to a very strong positive result, because both the larger part of the lung's surface will then be occupied be the oxygen molecules, and more of the oxygen will be absorbed via this part of the surface. Increasing twice the percentage of the oxygen in the air, we shall obtain the positive effect increased nonlinearly, i.e. more than twice.

2.1 Can the percolation theory help here?

The fact that the nature of the processes that interest us is **a threshold one,** such as the condition $A > A_{cr}$, suggests that the *percolation theory* [11] might be of some interest here. This theory considers the physics of the long paths of some particles, and reveals -- via interesting probability considerations [11] -- some threshold processes. The fact that the infected air brings both the needed oxygen and the CVM that can bother the oxygen to be absorbed – can be seen as some analogy to the situation with the conductive grid in the theory of percolation, where the total conductivity of the grid can be suddenly interrupted with the increase in the percentage of the cut branches.

In order to make this analogy more complete, we have to consider the trajectories of the molecules O_2 toward the lungs surface, and unite these trajectories in some way in clusters. We cannot continue with this topic here.

Let us return to the main line.

3. The role of the size

The inequality

$$A > \frac{kS}{\beta}$$

whose importance originates from the fact that *A* and *S* are defined independently, leads to some heuristically important conclusions; let us be confined by the following three:

1. Physical weakness, expressed in a noticeable decrease in *A*, compared to the usual frequency of breathing, is absolutely unacceptable for an ill man. It is thus suggested to critically consider the urgency in giving oxygen to an ill man, because just some shortage of oxygen could cause (force) the patient to intensively (quickly) breath – which should cause better cleaning of the lung from the CVM, according to (6). *A strong patient* may even be given an air supply with an intentionally reduced percentage of the oxygen (just add some N_2 to the air, while keeping its pressure) for the purpose of stimulation. Of course, this has to be

done under careful individual treatment and continuous observation, in order not to endanger the life, but we do have to distinguish between the necessity to urgently save one in a critical state – when giving the oxygen is absolutely necessary -- and the necessity to strengthen him -- for him not to soon return to the hospital with the same trouble.

2. The death of the huge-size animals, like dinosaurs, during the "Ice Age" is explained by the cold. We can assume, however, that the cold was not the only cause. The viruses in the air, and the insufficiently high A of the breathing of the giants (consider (6) for a very large S), which could not free the animals' lungs from the virus, could also cause the death. The nature of the environment should be carefully discussed. High humidity contributes to spreading and holding viruses in the air. The cold generally reduces the humidity, but not sufficiently everywhere, and there could be some, -- optimal for a pandemia -- conditions when both the cold and the humidity are significant, killing the animals together.

3. Generally, (6) shows the advantages of the small-size creatures, because small S makes it easy to realize the inequality. Thus, a (healthy) child, bird, or a mouse

have good chances not to become ill with the CV. As a matter of fact, already an average-size creature such as a dog demonstrates a relatively quick breathing and does not become ill with the corona virus. The tendency of some mothers to feed their children very well -- for them to become tall and wide -- which means a large S -- should *not* be encouraged. According to the discoveries of the archeologists, the armors of knights had shown that the knights were, as a rule, not higher than we are, about 173 cm height [12]. At the same time, sport and the associated development of the respiration are, of course, very useful. Similarly to the fact that our eye is most sensitive (adjusted) just to the green light that is in the maximum of the sunlight spectrum, our size and weight are also made optimal by Nature.

4. Further development of the "Naive Theory"

Despite the above reservations regarding the use of V, -- in terms of some averaged spatial distribution, we assume that the connection between the total number N of the CVM and their spatial density n can be taken as

$$n = \frac{N}{V} \ . \qquad \text{(7) repeated}$$

We then use that the volume oscillates because of the breathing as:

$$V(t) = V_o + \varepsilon(t) \qquad (8)$$

where V_o is some average value. Setting $\varepsilon(t) = \varepsilon_o \sin(2\pi A t)$, we realistically assume that the oscillations are significantly smaller than V_o, $\varepsilon_o << V_o$. For instance, $\varepsilon \approx V_o / 4$ seems reasonable. The frequency of the breathing A is the main (central) parameter of the whole theory.

By adding to (7) the balance equation for $N(t)$, we can find $N(t)$ and $n(t)$.

Using that, because of the smallness of $\varepsilon(t) / V_0$,

$$n = \frac{N}{V(t)} = \frac{N}{V_o + \varepsilon(t)} \approx \frac{N}{V_o}(1 - \frac{\varepsilon(t)}{V_o}) \qquad (9)$$

with the error of order

$$\left(\frac{\varepsilon(t)}{V_o}\right)^2,$$

and using (9) in (3), we obtain

$$\frac{dN}{dt} = -Pn = -P\frac{N(t)}{V(t)} \approx -\frac{P}{V_o}(1-\frac{\varepsilon(t)}{V_o})\,N(t). \qquad (10)$$

This linear time-variant equation is easily solved by separating the variables:

$$N(t) = K\exp\left\{-\frac{P}{V_o}\int_0^t [1-\frac{\varepsilon(\lambda)}{V_o}]d\lambda\right\}. \qquad (11)$$

with a constant K. Because of the smallness and the oscillatory nature of $\varepsilon(t)$, it appears from (11) that $N(t)$ is mainly proportional to the factor

$$e^{-\frac{P}{V_o}t}.$$

This factor obviously requires $P \geq 0$, that is,

$$A \geq \frac{kS}{\beta} . \qquad (12)$$

The opposite inequality would result in the tragedy of $N \to \infty$, as $t \to \infty$; thus (12) can be named *the survival condition*.

5. Do can we cause an antagonism between the CVM, causing them to destroy each other?

This is a topic for further development, mainly biological, for which the equational outlook should be somewhat continued. The initial idea assumed that since the CVM are "blind" they can attack each other, just as they can attack the lungs' cells. If this would be so, then it would be possible to insert to the thorax, just before the artificial ventilation of the lungs, many other CVM, causing all of the CVM present in the thorax – *because of their high concentration* -- *to collide,* attack and destroy each other. By controlling (measuring) $n(t)$ that should begin to reduce in time, the moment to start the ventilation should be determined.

This idea – that means, in fact, imagining CVM as billiard balls that can collide with each other just as they can collide with the wall, -- appears be too simplistic. A physician (see the Acknowledgements) explained to me that the lung's surface is covered by some protein layer (itself very complicated and many-functional) that catches (covers) the coming CVM whose subsequent self-reproduction is both due to the molecules themselves and the material of the layer. Thus, the collisions of CVM with the lung surface and their mutual collisions are *not* at all similar.

It became clear that in order to allow effective antagonism between CVM, some of them (those that we specially insert into the thorax) have to receive – by some special treatment -- a covering that would allow the CVM already present in the thorax to become "glued" to the inserted CVM. A preliminary laboratory experiment in which this covering could be sprayed onto a dense ensemble of CVM, is required.

Another important requirement to the cover is that the interaction of the covered CVM with the lung's cells has to be much weaker than their interaction with the uncovered CVM, -- the method should *not* lead to taking by these CVM more of the free places on the lung's surface – the places needed for absorption of the oxygen molecules.

Development of the cover that would satisfy both of the requirements – that of a good contact between different CVM, and that of a bad contact of the CVM with the lungs cells – would be an achievement of modern biology.

Let us prepare the equational side for the suggested procedure.

5. The linear time-dependent equation

If we consider collisions of similar molecules belonging to an ensemble with the spatial concentration n, then the probability of the binary collisions is directly proportional to $n \cdot n$, and if these collisions result in a *decrease* of the concentration of CVM because of the "destroying" by the molecules each other, then a nonlinear term of the type

$$-\gamma n^2$$

with a positive constant γ, arises in the balance equation. However, if, by the reasons mentioned, we actually generate an air pulse with the specially covered molecules, then this pulse *is a known time function*. Denoting it as $n^*(t)$, we would have in the balance equation not the nonlinear term, but the *linear time-variant* term

$$-\gamma n^*(t)\cdot n. \qquad (13)$$

For this situation, the ballast equation becomes (n_o is still ignored, and P is as in the above):

$$\frac{dN}{dt} = -Pn - \gamma n^*(t)\cdot n\ , \qquad (14)$$

or

$$\frac{dN}{dt} = -\left[P + \gamma n^*(t)\right]\cdot n \qquad (15)$$

Using that $N(t) = nV(t) = (V_0 + \varepsilon(t))n$, we obtain from (15):

$$\frac{d\left[(V_o + \varepsilon(t))\cdot n\right]}{dt} = -[P + \gamma n^*(t)]\cdot n \qquad (16)$$

or

$$(V_o + \varepsilon(t))\frac{dn}{dt} + n\frac{d\varepsilon}{dt} = -[P + \gamma n^*(t)]\cdot n, \qquad (17)$$

which yields

$$\frac{dn}{n} = -\frac{\frac{d\varepsilon}{dt} + P + \gamma n^*(t)}{(V_o + \varepsilon(t))} dt, \qquad (18)$$

from which, approximately,

$$d(\ln n) \approx -\frac{1}{V_o}(1 - \frac{\varepsilon(t)}{V_o})(\frac{d\varepsilon}{dt} + P + \gamma n^*(t))\, dt. \qquad (19)$$

Finally,

$$n(t) \approx K \exp\{-\frac{1}{V_o}\int_0^t [\frac{d\varepsilon}{dt} - \frac{\varepsilon}{V_o}\frac{d\varepsilon}{dt} + P + \gamma n^*(t) - P\frac{\varepsilon(t)}{V_o} - \frac{\varepsilon(t)}{V_o}\gamma n^*(t)]dt\} \qquad (20)$$

The degree of the exponent includes the terms:

$$\frac{-P}{V_o}t,\ \frac{\varepsilon(t)-\varepsilon(0)}{V_o},\ \frac{P[\varepsilon(t)-\varepsilon(0)]}{{V_o}^2},\ \frac{\varepsilon^2(t)-\varepsilon^2(0)}{2V_o},\ -\frac{\gamma}{V_o}\int_0^t n^*(t)\,dt,\ \frac{\gamma}{{V_o}^2}\int_0^t \varepsilon(t) n^*(t)\,dt \qquad (21)$$

Since $n^*(t)$ is *compactly supported* (nonzero only in a finite interval), and $\varepsilon(t)$ is finite, if P is nonzero, then $\frac{-P}{V_o}t$ is the only expression here that infinitely increases in time, and all the others are not so important. Thus, as $t \to \infty$ the result of $n(t)$ and $N(t)$ being directly proportional to $e^{-\frac{P}{V_o}t}$ remains.

However, at the limiting (formally permitted) "balanced" case of $P = 0$, the value of

$$\frac{\gamma}{V_o^2}\int_0^t \varepsilon(t)n^*(t)dt \qquad (22)$$

and all the other nonzero terms become important as defining the stationary, almost constant $n(t)$ and $N(t)$.

Denoting the integral value of the pulse $n^*(t)$ as N^*, we have, for the time when the pulse is already finished

$$-\frac{\gamma}{V_o}\int_0^t n^*(t)dt \equiv -\frac{\gamma}{V_o}N^*.$$

The situation of (22) is also simple, since the inserted in the thorax pulse $n^*(t)$ should be naturally correlated with $\varepsilon(t)$ -- it is most simple to insert the covered molecules with the breath. Thus, we can take the compactly supported $n^*(t)$

directly proportional to $\varepsilon(t)$ at some half period of the $\varepsilon(t)$ -- this time-interval is physically most suitable.

Calculation of all of the terms in (21) is very easy. On the physical side, the *measurement* of the *established* value of n also should be relatively easy.

7. Some research and pedagogical targets, or possible student projects

1. To carefully consider the points of Section 3; in particular to learn for item 2 there about the situation with the elephants (huge animals with large S) in India, in the corona virus environment.
2. To develop methods for easy measurement of n (presumably, in the exhalated air).
3. To develop the cover material for the CVM, suggested for a use in Section 5.
4. To develop a method for creation ensembles of CVM with prescribed n for preliminary experiments with the cover.
5. To improve the "Naïve Theory" by taking one more term in the expansion of $1/V$, that is, using

$$\frac{1}{V_o + \varepsilon(t)} \approx \frac{1}{V_o}[1 - \frac{\varepsilon(t)}{V_o} + \frac{\varepsilon^2(t)}{V_o{}^2}] \quad . \qquad (23)$$

As is shown in the next section, this correction leads to a *small* addition to the degree in the main factor,

$$e^{-\frac{P}{V_o}t},$$

which shows a good precision of the theory.

6. To try to connect our results with the theory of diffusion regarding the processes in the thorax.
7. The same regarding the theory of percolation.
8. To consider in what degree *S* indeed is the *whole* area of the lung's surface.
9. To examine possibility to induce electrical dipole moment onto the CVM – a special point for the hope to influence the diffusion of the molecules, suggested in Section 2.
10. To invent a game of the billiard type where the walls (boards) of the table are covered (defended) by so soft a material that the collision of a ball with the wall would take some time, and thus to try to make the processes in the thorax "feasible". As well, to develop relevant computer games.

8. The use of (23)

Let us rewrite (19), using (23)

$$d(\ln n) \approx -\frac{1}{V_o}[1-\frac{\varepsilon(t)}{V_o}+\frac{\varepsilon^2(t)}{V_o{}^2}][\frac{d\varepsilon}{dt}+P+\gamma n^*(t)]dt \qquad (24)$$

The most essential term, appearing here after integration, still is $-\frac{P}{V_o}t$. However, there is now one more term that also, though slower, infinitely increases in time. Integrating the most relevant addition, appearing in the right-hand side of (24), namely

$$\int_0^t\left[-\frac{1}{V_o}P\frac{\varepsilon^2(t)}{V_o{}^2}\right]dt = -\frac{P}{V_o{}^3}\int_0^t\varepsilon^2(t)\,dt =$$

$$-\frac{P\varepsilon_o{}^2}{V_o{}^3}\int_0^t\sin^2(2\pi At)\,dt = -\frac{P\varepsilon_o{}^2}{2V_o{}^3}\int_0^t(1-\cos(4\pi At))\,dt$$

and using that the average of $\cos(4\pi At)$ is zero, we obtain the here important part of the integral as

$$-\frac{P\varepsilon_o^{\,2}}{2V_o^{\,3}}t\,.$$

Together with $-\frac{P}{V_o}t$ in the degree, the essential factor in $n(t)$ and $N(t)$ now is

$$e^{-\frac{P}{V_o}(1+\frac{\varepsilon_o^{\,2}}{2V_o^{\,2}})t}\,. \qquad (25)$$

Obviously, the condition $P(A)>0$ remains as the main one. Since the added degree may be just of several percentages, the good precision of the theory is confirmed.

9. Conclusions

The topic of breathing of a human having the corona virus disease is heuristically interesting, and our simple analytical tools and physical arguments are useful. Even though we have considered the process of the breathing very "macroscopically", it is argued that the cause of death can appear already on the lung's surface. As well, some unexpected connections (Section 3) are revealed on the way of the research. It is important that the macroscopic theory should be relevant for any mutation of the CVM. The frequency of breathing is a threshold parameter for survival. For large creatures, it must be correspondingly large, which hardly takes place in reality. The idea to cause an antagonism between the corona virus molecules for them to kill each other, presents, at this stage, just a line of thought, that I find motivating. Some topics for students' research projects are suggested. There is the hope that researchers, -- even professional biologists and physicians, -- will find some of the suggestions in the present discussion helpful for them.

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