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ORIGINAL ARTICLE

A Heuristic Point of View on the Breathing in the Corona Virus Environment: The "Naive Theory"

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Abstract

It is an attempt to analyze the breathing (respiration) of an already ill by the Corona Virus (CV) disease person in simple physical and equational terms, and to suggest (in these terms) a forcing of the CV Molecules (CVM) to destroy each other.

Key Words: *Corona virus disease; Dynamic Process; Energy flow; System structure; Molecular competition; Education*

A Brief Introduction – What for us is the Coronavirus Disease?

Very unfortunately, we live in wars and epidemies. In the wars, we are certainly guilty by our own, and the Epidemies sometimes move from the first plan to the background, so that it becomes less fashionable or interesting to speak about them. Then, even the semi-heuristic, semi-religion question arises of whether or not the Epidemies are some punishments for the wars, sent to us from the heaven, or something which we must have according to the English proverb saying that a good hunting dog must have some fleas.

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We collect some problems, partly belonging to physics, as suggestions to student projects.

The whole presented situation seems to be some interesting research field, which completes the purely biological studies that could not be touched here. It is not mandatory, but motivating reading, which teaches to see as simple what indeed can be simple.

The demographic problem

We do not have an infinite plane, to live on it as was once imagined, but the finite surface of the earth ball, and the resulted demographic (overpopulation) problem causing epidemies in space and also the problem in time – not enough time for the physician to understand the particular situation, and to decide what to do, e.g., to give or not to the patient an oxygen addition.

The Constructive Points

There are two basic threshold processes (problems) in focus, associated with breathing (respiration):

1. An insufficient oxygen flow because the CVM (Corona Virus Molecules) [1-9] cause inflammation of the lung cells and thus are bothering the oxygen molecules to penetrate the lungs (Figure 1).

The oxygen flow is important for many energetic processes in the body, even for the heart activity.

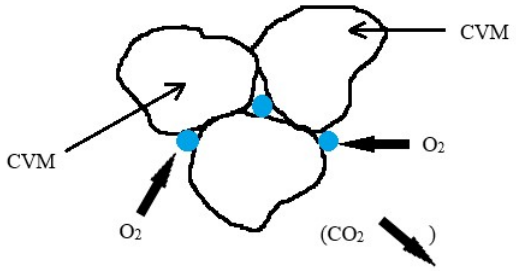

Figure 1) *By causing inflammation of lung cells, the CVM can close the diffuse entrances (between the cells) for O_2.*

If the flow of O_2 into the lung is lower than some needed level, the body dies. Notice – the death is a threshold phenomenon, depending on the ratio of the CVM and O_2 molecules in the taken air.

We can see a "competition" between CVM and O_2 – who comes first to the lung surface?

Question: Can we help O_2 in this competition by deflecting the massive CVM, assumed them having some dielectric properties (Figure 2), by an aside electrical field?

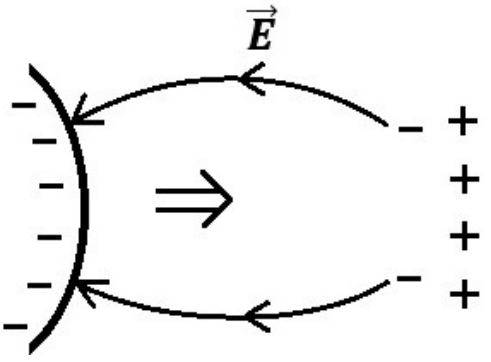

Figure 2) *The deflection by an aside electrical field of large molecules that can be polarized.*

The other (second) reason is our focus here:

2. Possibly insufficiently high frequency of breathing – see the following theory of Balance Equation.

According to both of the problems 1 and 2, the death can come already in the lungs, not just because of some biological action of the CVM penetrated into the depth of the body. It is a problem of breathing.

Balance equation

Consider the total number *N* of the CVM that is present in the lung (thorax) of an ill human. The balance of *N*, either its constancy or its tendency up or dawn, – is decisive as regards life or death. Obviously, we should not come to $N \rightarrow \infty$.

The roles of inhalations and exhalations of the respiration are very distinct in this regard because the averaged spatial concentrations, i.e., the densities, of the CVM – those inside the thorax, *n*, and those in the surrounding space, n_0, strongly differ. Since the human is ill, in the steady state,

$$n \gg n_0 \qquad (1)$$

and when assuming that there is a good ventilation in the room, we can even set, for simplicity, $n_0 = 0$.

By taking the CVM out of the lung, the exhalations much stronger influence *N*, than the inhalations do, though an initial portion of *N* should come with an inhale; for this first moment we cannot set $n_0 = 0$.

All this and the following theory of the balance equation relates, of course, to the established steady-state.

The Balance Equation for *N* is, in its two main terms (Figure 3):

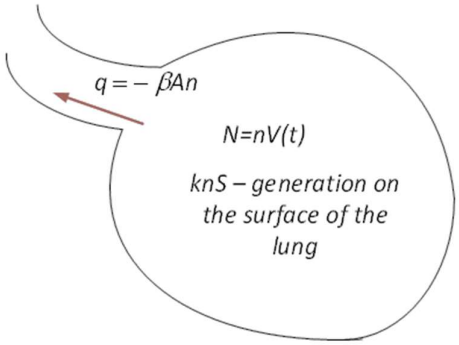

Figure 3) *The thorax, the two main terms of Equation 2; q here is q_{out} in the main text, the steady state.*

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

The "obvious" equality $N = nV$, defining some equivalent volume *V*, is not trivial, and must be considered. We just assume a priori that $N \rightarrow \infty$ and $n \rightarrow \infty$ should occur together.

The Balance Equation 2 readily obtains the form,

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

where A is the frequency of the breathing, measured in Hz. S is the area of the lung's surface, and β and k are some positive constants, whose physical dimensions are, as is easy to see, $[\beta] = m^3$ and $[k] = \frac{m}{sec}$.

That both terms on the right-hand side of Equation 3 are directly proportional to n, starts a linear theory. Such a theory often uses Laplace or Fourier transform, but we can manage with the ODE (Equation 3) after we write it either for $N(t)$, or $n(t)$.

Because of the relative smallness of the outside concentration n_0, in the steady-state, the exhalation-flow out

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

is much more significant for N than the inhalation-flow inside, $q_{in} = +\beta An_0$. That the velocity of each of the flows is directly proportional to A, is obvious.

The direct proportionality of the source-term

$$k\,S\,n \quad (5)$$

to the lung's surface area S represents the assumption that the reproduction of the CVM – the essence of the illness as we see it here – takes place either on the whole surface, or on a certain its part.

Writing Equation 3 as,

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

we introduce the important parameter,

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

regarding which we shall prove (in section, Basic Development of the "Naive Theory" – The Inequality for A) that $P>0$ necessarily, that is,

$$A > \frac{kS}{\beta} \quad (6)$$

Otherwise $N \to \infty$ (or $n \to \infty$).

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

$$A_{cr} = \frac{kS}{\beta} \sim S, \quad (6a)$$

then one can die.

Considering A_{cr}, we observe in Equation 6, an important "competition" between A and S which are mutually independent (independently measured) values. This independence is physically important, and this "competition" opens the way for some interesting conclusions (in section, Basic Development of the "Naive Theory" – The Inequality for A).

The Geometry

We widely use the simple equality,

$$n = \frac{N}{V} \tag{7}$$

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

Physics is always deeply associated with geometry, and here the geometry is so complicated that, from the analytical side, our use of V is more a "conceptual", than a routine one.

The form of the lung is so complex, that on the geometry regard it is most simply to see the complexity, by passing from "Jordan Curve" i.e., a line, closed in the plane, without any self-crossings, but of a however complicated form, in some sense realizing the map 1D→2D, on to "Jordan Surface" realizing the map 2D→3D, in the 3D space.

The lung is finite and has no self-crossings, and, figuratively speaking, fills the thorax with its surface (2D→3D), similarly as "Jordan Curve" fills the 2D plane.

A more physical way for considering the complexity, is to apply the auto-similarity argument to the mutual analogy of the human lungs and the gills of a fish (In section, Some Research and Pedagogical Targets, or Possible Student Projects).

Besides the problematic sense of V, in the connection of N and n, a nontrivial point is that despite the formally very simple mutual analytical 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, which 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 molecules than for CVM. All of the molecules in focus are electrically neutral, but if it would be possible – as was already noted, 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.

Since a death-causing problem can appear already on the surface of the lungs, as an interruption of the sufficient oxygen supply, we can see how effective it can be to give (especially to a weak human) 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 by the oxygen molecules, and more of the oxygen will be absorbed via this part of the surface. By increasing the percentage of the oxygen in the air twice, we increase the positive effect more than twice.

On the Role of Inhales

Even though, for the balance equation the inhalations appear to be of minor importance, the general importance of the inhalation is clear:

1. It is absolutely necessary for taking the oxygen from outside, this to be deeply associated with the energetic processes in the body.

2. It is important for the internal processes in the thorax, because after being received, the oxygen cannot be immediately effectively absorbed by the lungs – some time is need for it to come to the whole (or most of the) lungs' surface. The inhales (breaths) provide some repeated pumping – that can be named "internal breathing" causing the oxygen molecule to penetrate deeper. However, the same occurs with the CVM, and the mentioned competition between the two kinds of molecules continues. This very complicated process must be considered together with the mentioned topic of diffusion of the molecules. It may be additionally noted regarding the inhalation that it has some automatic nature, because with the absorption of the oxygen, the pressure of the air in the thorax is reduced, which it is very difficult to tolerate, and taking more air becomes physiologically necessary. One notes that since CVM contribute, in their part, to the pressure of the infected air, they should similarly contribute to the effect of the automatism.

As for the exhalations, it is also desirable – as suggested by the teachers of sport – to perform them several times sequentially, with some pressure. However, it is not done automatically, but intentionally, aimed to free the thorax from the "used" air with CO_2 and with O_2 reduced from 21% to 16%. In

any case, the roles of the inhalation and exhalation are here very different.

Can the percolation theory [11] help here?

The fact that the nature of the processes that interest us is a threshold one, the condition $A > A_{cr}$, and the limitation on the oxygen absorption via the lung's surface, 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 of 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 randomly) cut branches. I imagine the ensemble of the CVM as a "flying" conductive grid that the flow of the oxygen has to finally pass through.

In order to make this intuitive 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 [11].

Basic Development of the "Naive Theory" – The Inequality for *A*

Despite the problematic sense of *V*, in terms of some averaged spatial distribution, we assume, for simplicity, that the connection between the total number *N* of the CVM and their spatial density *n* can be taken as Equation 7.

However, the volume oscillates because of the breathing as,

$$V(t) = V_0 + \varepsilon(t) \tag{8}$$

where V_0 is some average value. Setting $\varepsilon(t) = \varepsilon_0 \sin(2\pi A t)$, we realistically assume that the oscillations are significantly smaller than V_0, $\varepsilon_0 \ll V_0$. For instance, $\varepsilon_0 \approx V_0/4$ 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 $\varepsilon(t)/V_0$,

$$n = \frac{N}{V(t)} = \frac{N}{V_0 + \varepsilon(t)} \approx \frac{N}{V_0}\left(1 - \frac{\varepsilon(t)}{V_0}\right) \tag{9}$$

with the error of order $\left(\frac{\varepsilon(t)}{V_0}\right)^2$, and using Equation 9 and 3, we obtain,

$$\frac{dN}{dt} = -Pn = -P\frac{N(t)}{V(t)} \approx -\frac{P}{V_0}(1 - \frac{\varepsilon(t)}{V_0})N(t) \quad (10)$$

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

$$N(t) = K \exp\{-\frac{P}{V_0}\int_0^t [1 - \frac{\varepsilon(\lambda)}{V_0}]\, d\lambda\} \quad (11)$$

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 $e^{-\frac{P}{V_0}t}$. This factor obviously requires $P \geq 0$, that is,

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

(Equation 6 repeated)

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

The Role of the Size

The inequality $A > \frac{kS}{\beta}$ 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 Equation 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 must 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 so that he need not return to hospital with the same trouble.

2. The death of the huge-size animals, like dinosaurs, during the "Ice Age" is traditionally 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 Equation 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 the viruses in the air. The cold generally reduces the humidity, but not sufficiently everywhere, and there could be some, optimal for the pandemia, conditions when both the cold and the humidity are significant, together killing the animals.

3. Generally, Equation 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. The found by the archeologists armors of knights had shown that the knights were, in average, about 173 cm height [12], not really higher (taller) than we are. 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.

Can We Cause an Antagonism Between the Different CVM, Causing Them to Destroy Each Other?

This is a topic for a further development, mainly a biological one, 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 then present in the thorax – because of their high concentration to collide, attack and destroy each other. By controlling (measuring) $n(t)$ that should then 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 (Acknowledgement) 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 collisions with each other 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 this cover is that the interaction of the covered CVM with the lung's cells must 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.

Let me assume that 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 a serious achievement of modern biology.

Let us prepare the equational side (still in the borders of the "naïve theory") for the suggested procedure.

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,

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

For this situation, the ballast equation becomes (n_0 is still ignored, and P is as in the above):

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

or

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

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

$$\frac{d[(V_0+\varepsilon(t)).n]}{dt} = -[P + \gamma n^*(t)].n \qquad (16)$$

or

$$\left(V_0 + \varepsilon(t)\right)\frac{dn}{dt} + n\frac{d\varepsilon}{dt} = -[P + \gamma n^*(t)].n \qquad (17)$$

which yields,

$$\frac{dn}{n} = -\frac{\frac{d\varepsilon}{dt}+P+\gamma n^*(t)}{(V_0+\varepsilon(t))}\,dt \qquad (18)$$

from which, approximately,

$$d(\ln n) \approx -\frac{1}{V_0}(1-\frac{\varepsilon(t)}{V_0})(\frac{d\varepsilon}{dt} + P + \gamma n^*(t))dt \qquad (19)$$

Finally,

$$n(t) \approx Kexp\{-\frac{1}{V_0}\int_0^t[\frac{d\varepsilon}{dt} - \frac{\varepsilon}{V_0}\frac{d\varepsilon}{dt} + P + \gamma n^*(t) - P\frac{\varepsilon(t)}{V_0} - \frac{\varepsilon(t)}{V_0}\gamma n^*(t)]dt\} \qquad (20)$$

The degree of the exponent includes the terms,

$$\frac{-P}{V_0}t, \frac{\varepsilon(t)-\varepsilon(0)}{V_0}, \frac{P[\varepsilon(t)-\varepsilon(0)]}{V_0^2}, \frac{\varepsilon^2(t)-\varepsilon^2(0)}{2V_0}, -\frac{\gamma}{V_0}\int_0^t n^*(t)dt, \frac{\gamma}{V_0^2}\int_0^t \varepsilon(t)n^*(t)dt \qquad (21)$$

Since $n^*(t)$ is compactly supported (nonzero only in a finite time interval), and $\varepsilon(t)$ is finite, if P is nonzero, then $\frac{-P}{V_0}t$ 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 $N(t)$ be directly proportional to $e^{-\frac{P}{V_0}t}$ remains.

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

$$\frac{\gamma}{V_0^2}\int_0^t \varepsilon(t)n^*(t)dt \qquad (22)$$

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

$$-\frac{\gamma}{V_0}\int_0^t n^*(t)dt \equiv -\frac{\gamma}{V_0}N^*$$

The situation of Equation 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 Equation 21 is very easy. On the physical side, the measurement of the established value of n also should be relatively easy.

Some Research and Pedagogical Targets, or Possible Student Projects

1. To carefully consider the second point of the section, The Role of the Size, regarding the modern situation with the huge animals with large S in the corona virus environment. Such situation in India is (Acknowledgement) as follows: some 8 lions gave positive tests, but no elephants and rhinos were detected ill. The latter may be, however, because of the immunity to this virus, developed in the animals over numerous centuries.

2. To create an auto-model theory allowing us to transfer from the breathing in the air to the breathing (by the fish) in the water, and to make some constructive conclusions from such a theory, as re the structure of the human lungs.

3. Using the fact that fishes absorb by their gills the oxygen from the water, to add to the water some other molecules that can be absorbed by the gills, and to study the possibility for the living creatures to survive, via such biological modelling.

4. To develop methods for an easy measurement of *n* (presumably, in the exhalated air).

5. To develop the cover material for the CVM, suggested for a use in section, Can We Cause an Antagonism Between the Different CVM, Causing Them to Destroy Each Other?

6. To develop a method for creation ensembles of CVM with prescribed *n* for preliminary experiments with the cover.

7. To improve the "Naive Theory" by taking one more term in the expansion of 1/V, that is, using

$$\frac{1}{V_0+\varepsilon(t)} \approx \frac{1}{V_0}\left[1-\frac{\varepsilon(t)}{V_0}+\frac{\varepsilon^2(t)}{V_0^2}\right] \quad (23)$$

As is shown in the next section, this correction leads to a small addition to the degree in the main factor, $e^{-\frac{P}{V_0}t}$, which shows a good precision of the theory.

8. To try to connect our results with the theory of diffusion regarding the processes in the thorax.

9. The same regarding the theory of percolation.

10. To consider in what degree *S* indeed is the whole area of the lung's surface.

11. To examine possibility to induce electrical dipole moment onto the CVM – a special point for the hope to influence the diffusion (propagation) of the molecules, suggested in section, The Geometry.

12. 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".

13. To develop relevant computer games.

14. To consider in what degree can the reproduced (new) CVM belong to the gas phase (or all of them have to immediately enter the body via the lung's cells).

The Use of Equation 23 (A Way to Check the Precision of The Theory)

Let us rewrite Equation 19, using Equation 23.

$$d(\ln n) \approx -\frac{1}{V_0}[1 - \frac{\varepsilon(t)}{V_0} + \frac{\varepsilon^2(t)}{V_0^2}][\frac{d\varepsilon}{dt} + P + \gamma n^*(t)]dt \quad (24)$$

The most essential term, appearing here after integration, still is $-\frac{P}{V_0}t$. 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 Equation 24, namely,

$$\int_0^t \left[-\frac{1}{V_0}P\frac{\varepsilon^2(t)}{V_0^2}\right]dt = -\frac{P}{V_0^3}\int_0^t \varepsilon^2(t)dt$$
$$= -\frac{P\varepsilon_0^2}{V_0^3}\int_0^t sin^2(2\pi At)dt$$
$$= -\frac{P\varepsilon_0^2}{2V_0^3}\int_0^t (1 - \cos(4\pi At))dt$$

and using that the average of $\cos(4\pi At)$ is zero, we obtain the here important part of the integral as $-\frac{P\varepsilon_0^2}{2V_0^3}t$ .Together with $-\frac{P}{V_0}t$ in the degree, the essential factor in *n(t)* and *N(t)* now is,

$$e^{-\frac{P}{V_0}(1+\frac{\varepsilon_0^2}{2V_0^2})t} \quad (25)$$

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.

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 for death can appear already on the lung's surface. As well, some unexpected heuristic connections, associated with the role of S (section, Basic Development of the "Naive Theory" – The Inequality for A) are revealed on the way of the research. 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 irus 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. We should be altogether, absolutely humanistic and friendly in this noble struggle.

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