

The Corona Virus Problem *via* a Linear Time-Variant Equation

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Abstract

Besides the visual heuristic side, our research of the Corona Virus Problem includes the line of physics considerations and the equational line. The present work is merely devoted to the latter line. It appears that some proposed special covering of some of the Corona Virus molecules, which is purposed to cause an antagonism between these molecules and those uncovered, leads to turning a nonlinear balance equation into a linear time-variant one (which the physicists sometime call "parametric equation").

Keywords: Corona virus disease; Breathing (respiration); Balance equation; Medical treatment

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Introduction

We develop the proposition of [1], and especially of [2], where a specific possibility to fight the coronavirus (CV) disease is proposed. The point of [2] is to insert into the lungs of an already infected human before starting the ventilation of his lungs additional CV molecules $n^*(t)$, assuming that these molecules and those already present in the lungs, $n(t)$, can start to kill each other by their protein arrows. This requires some preliminary treatments namely special covering of the added molecules, for the molecules to be "glued" to each other in order to "compete". This is not a simple biology problem that cannot be solved here. However, focusing at the equational line of the whole research, we find this situation mathematically interesting. If the mentioned covering can be done, then we have because of the knowledge of the defined by us function $n^*(t)$, the term $n^*(t)N$ in the dynamic balance equation for the total number N of the CV molecules in the lung. This term makes this equation not a nonlinear, but a linear time variant one. This equation is easily solved, and studying the obtained $n(t)$ then appearing to be a decreasing time function we can predict the moment when artificial ventilation of the lung has to start.

The Balance Equation

For brevity of writing we denote "CV molecules" as CVM.

After ignoring the density of the CVM denoted in [2,3] as n_o outside the lung (since $n_o \ll n$), the complete balance equation for the total number $N(t)$ of the CVM in the lung becomes

$$\frac{dN}{dt} = -\beta An(t) - \gamma n^*(t)N(t) + kS(n(t) + n^*(t)) \quad (1)$$

Here $n^*(t)$ is a given function of the added specially treated CVM. Concentrated in time ("compactly supported") and artificially created, $n^*(t)$ has no connection to n_o . S is the area of the surface of the lung, A is the frequency of the breathing, and $-\beta An(t)$ are some positive constants of direct proportionality, having proper physical dimensions (**Figure 1**).

Consider each term of (1).

$-\beta An(t)$ is the flow of the CVM *out* of the lungs, i.e., the exhalation. Of course, the breath takes, from outside, some CVM *into* the lungs, but is not so important, because the spatial concentration of the CVM outside, n_o , is much lower than n inside. Thus, in our consideration of the balance, *exhalation is more important than breath*. Exception is when $n^*(t)$ (actually, a quick pulse that below we shall even model as $\delta(t)$) of the artificial addition is significant.

The sign "--" in the term $-\beta An(t)$ is because of the direction of this flow (the exhalation) out of the lungs. The situation is somewhat similar to that of charging of a capacitor, indeed: If the capacitor is charged by the current i , then for the accumulated on the capacitor's plate charge Q

$$i = -\frac{dQ}{dt} \quad (2)$$

and if it is discharged, then

$$i = \frac{dQ}{dt} \quad (2a).$$

Here Q is analogous to our N . Introducing the surface charge density of the plate

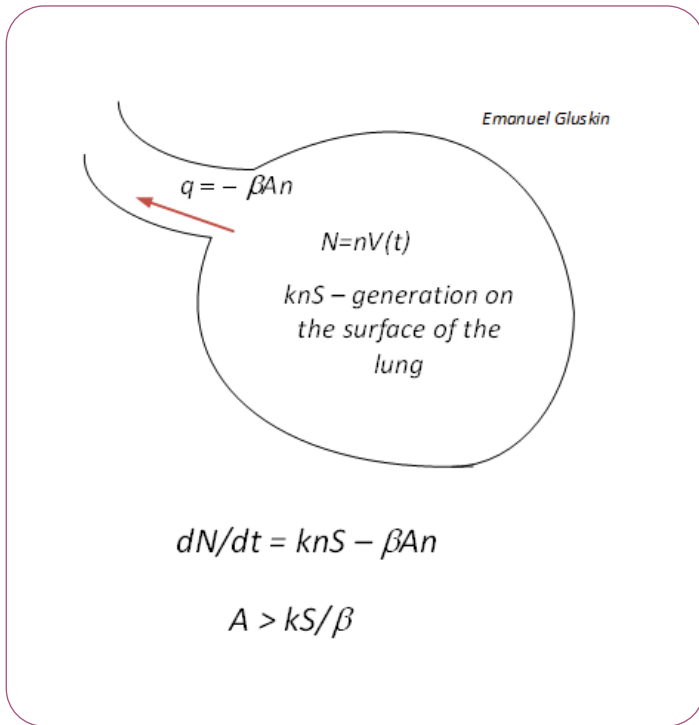


Figure 1: The schematic thorax, the main part of the balance equation, and the resulting inequality-the preceding material for [1].

$$\sigma = \frac{Q}{S}$$

Where, S is the area of the plate, we obtain from (2a)

$$\frac{d\sigma}{dt} = -\frac{i}{S}$$

The important distinction to our case, is that while in $\sigma = \frac{Q}{S}$, S is really constant, in $n = \frac{N}{V}$, V is not constant, as we are breathing, causing the thorax and V to oscillate. This, however, does not represent any serious difficulty. That our focus is on n, and not on N, is because in the respiration we can measure (by any means – electrical, optical, or chemical) n, but not N. Just compare this with measurement of humidity of the air in lungs. Obviously, we are able to directly measure the humidity in the respiration air, but not directly the total amount of the water in the lungs.

The other most important term in (1) is

$$kS(n(t) + n^*(t))$$

which can be written (before the special treatment associated with $kSn(t)$ starts i.e. when $n^*(t)$ is still zero) as

$$kSn(t)$$

Here S is the area of the surface of the lung. This term presents a source of the CVM, because the existing CVM reproduce (multiply) themselves on the cells of the lungs – the essence of the disease. Contrary to the term $-\beta An(t)$ that reflects decrease in N, $kSn(t)$ means increase; thus in a very rough approximation in

the stationary balance state, it has to be $kSn(t) + kSn(t) = 0$, that is $A \geq \frac{kS}{\beta}$. In fact, a more detailed analysis (see below and [1-3]) requires the inequality

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

even more surely

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

The term $-\gamma n^*(t)n(t, x, y, z)$ of (1), associated with our belief that the mutual killing of the CVM is possible, also decreases N(t). This term is obtained from

$$-\gamma n^*(t)n(t, x, y, z)$$

in which we need not assume to $n^*(t)$ be spatially dependent, while what is the coordinate dependence of n is not important. Indeed,

$$\iiint_{x y z} -\gamma n^*(t)n(t, x, y, z) dx dy dz =$$

$$-\gamma n^*(t) \iiint_{x y z} n(t, x, y, z) dx dy dz =$$

$$-\gamma n^*(t)N(t)$$

On the Determination of N(t) and n(t)

For the spatial density of the CVM in the lung, i.e. n(t), we have

$$n(t) = \frac{N}{V(t)} = \frac{N}{V_0 + \varepsilon(t)} \approx \frac{N}{V_0} \left(1 - \frac{\varepsilon(t)}{V_0}\right) \quad (3)$$

Where, in the volume of the lung $V(t) = V_0 + \varepsilon(t)$, V_0 is the average value, and $\varepsilon(t)$ is the relatively small oscillatory part due to the respiration.

Substitution of (3) into (1) yields

$$\frac{dN}{dt} \approx \left[-(\beta A - kS) \frac{1 - \frac{\varepsilon(t)}{V_0}}{V_0} - \gamma n^*(t) \right] N(t) + n^*(t)kS \quad (4)$$

from which

$$N(t) = kS \int_0^t n^*(t) dt + Ke^{-\frac{P}{V_0} \int_0^t [1 - \frac{\varepsilon(t)}{V_0}] dt - \int_0^t \gamma n^*(t) dt} \quad (5)$$

Where, $P = \beta A - kS$.

Observe from (5) that

$$K = N(0)$$

and that immediately after $n^*(t)$ is finished

$$N = kSN^* + N(0)e^{-\gamma N^*} \quad (6)$$

Where,

$$N^* = \int_{-\infty}^{+\infty} n^*(t) dt \quad (7)$$

Taking $n^*(t)$ as Dirac's function $\delta(t)$ (meaning here a relatively quick realistic pulse), we have $N^* = 1$. Also taking

$$\varepsilon(t) = \varepsilon_0 \sin 2\pi At,$$

we obtain from (5):

$$N(t) = kS + Ke^{-\frac{P}{V_0} [t + \frac{\varepsilon_0 \cos 2\pi At}{2\pi A V_0}] - \gamma} \quad (8)$$

Observe that $N(t)$ is maximal when $\cos 2\pi At = -1$, and minimal when $\cos 2\pi At = 1$. The relative difference between the maximal and the minimal values of N appears to be

$$\frac{N_{\max} - N_{\min}}{N_{\min}} = e^{\frac{\varepsilon_0 P}{\pi A V_0^2}} = e^{\frac{\varepsilon_0 (\beta - \frac{kS}{A})}{\pi V_0^2}} \quad (9)$$

By decreases in both S and V_0 , the relative difference is increased, which obviously means that the respiration of a small living creature is more effective.

Among the two factors of the last term in (8), i.e.,

$$e^{-\frac{P}{V_0} t} \text{ and } e^{-\frac{P}{V_0^2} \frac{\varepsilon_0 \cos 2\pi At}{2\pi A} + \gamma}$$

The most important is $e^{-\frac{P}{V_0} t}$. Thus, also for the situation with inserting $n^*(t)$ into the lungs, the obtained in [1-3] condition $P > 0$, or

$$A > \frac{kS}{\beta} \quad (10)$$

is necessary. Inequality (10) means that for small creatures (small S) it is easy to have the sufficiently high frequency of the breathing. Already such a not very small creature as the dog has such an intensive breathing (such a high A) that its lungs are well ventilated, and the probability of infection by coronavirus is small [3].

Comment: We often see here the advantage of the small size. This motivates the question of whether only the cold in the "ice period" was the cause for the death of the huge animals, like dinosaurs, or there also were some diseases, associated with viruses in the air – while the respiration of the huge animals could not clean their lungs sufficiently well [3-8]. Another noticeable point is that the well-known criticism of Jonatan Swift regarding the large-size people appearing in his story about Gulliver is insufficient. The problem was also with the Lilliputians who could not behave like usual people, but more like mouths. (Besides the

problem of the respiration, consider the thermal conditions, why the smallest mammal in water i.e. in the sea is the dolphin, and in the air, i.e. on the ground the mouse).

On the Moment of the Start of the Artificial Ventilation of the Lungs

Not being able, unfortunately, to solve here the biological problem of creation of the cover for the added molecules, let us make one more mathematical-phenomenological step. Introducing n_c the critical level (which has to be known) for $n(t)$ when the artificial ventilation of the lungs has to start, and using the above equations, we write the equation from which the moment of the starting the artificial ventilation can be found:

$$n(t) \approx \frac{N(t)}{V_0} \approx \frac{N(0)}{V_0} e^{-\frac{P}{V_0} t} = n_c \quad (10)$$

from which

$$t = \frac{V_0}{P} \ln \frac{N(0)}{V_0 n_c}$$

Since in the factor, the influence of V_0 is stronger than it is inside the logarithm, a decrease in V_0 decreases t . Furthermore, since the size of the living creature relates to both V_0 and S , let us expand P , having

$$t = \frac{V_0}{\beta A - kS} \ln \frac{N(0)}{V_0 n_c}.$$

Making S smaller, we increase the denominator of the factor, additionally decreasing t . Thus, for the same n_c , for children the ventilation of the lungs should start earlier.

Conclusion

Unfortunately, we were not able to consider (suggest) the needed cover of the added molecules. It is only clear that this cover can be much simpler than the protein cover of the lungs' cells. The latter cover is many-functional, while the needed cover of the added molecules has only to provide the "gluing" of the CVM, making them competing.

The big plus of the present discussion is that if the idea works, then it is relevant to any mutation of the CVM, because it only uses the presence of the protein arrows.

Last, but not the least from

$$N = kSN^* + N(0)e^{-\gamma N^*} \quad (6 \text{ repeated})$$

we can find minimum of the function $N(N^*)$. The condition $\frac{dN}{dN^*} = 0$ gives

$$N^* = \frac{1}{\gamma} \ln \frac{N(0)\gamma}{kS}$$

Analysis of this interesting connection between N^* , $N(0)$, and γ is left to the Reader. Let us only notice that a small S increases N^* .

Acknowledgments

I am grateful to Mr. Eithan Gluskin (The Israeli Medical System) for drawing my attention to the fact that an interaction between two CVM cannot be as that between such a molecule and a cell of the lung. I was explained that the lung has a sophisticated (and multifunctional) protein cover over its cells. This cover "catches" the coming CVM, helping it (them) to be reproduced. It became obvious that some covering of the added CVM is needed, but this cover need not be so complicated as that of the lungs.

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