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Review Article

A New "Non-Epidemiological" Model of the Covid19 Pandemic - Based on Potential Infection Ceilings [Maximum Number of Infected Persons] and Blocks - Taking in to Account the Results of Simple Calculations of Virus Multiplication as Well as Infection and Infectiveness of Persons Part III- Two or Three Waves of Pandemic? What do they Really Mean?

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Abstract

The main objective of this part was further analysis what might be the reasons of "an unexpected excess of cumulative cases of infection redorded in statistics- from March to June 2020- over mine calculated data. It looked like this "strange excess"came from the infection of group of people [in Poland this group is very small one], so called group A [or I], apparently with the deficit of blocking sites in their blood. Such a group dominated during that early pandemic period. Alternative explanation, unconvincing in my opinion, which I discussed, would entail the assumption that this first wave was caused by „early" [„unmutated"] strains of SARSCoV-2, and that the second [and third] wave of the pandemic by „late mutants" [of the „British strain" type]].

I recalculated- with mine assumed total number of viruses on given time-statistical records of number infections on Poland and Belgium assuming there is no one population but two separate groups of people with different I_{max} and K_a [thus different kinetic coefficients for binding receptor/s with viruses and infection]. I estimated I_{max} and K_a using Eadie-Hofstee plot of linearizing of Hill equation -in analogy to that applied in enzymology for Michaelis-Menten equation. The results of such calculations were very consistent with statistical records. The same I repeated for Netherlands, Hungary and Portugal.

As there so called third wave of pandemic begun, first reported in Great Britain, I calculated once again for Poland but first of all for Great Britain but this time assuming there exist not two but three separate types [groups] of people. The data were consistent with the statistics.

Finally, I added to my previous data [apparently representing group B-with an efficient block] data for group A and C responsible for first [early] and third [last] waves of pandemic. But as the latter data were evidently too low I corrected them according my proposed approach. So, I obtained the most probably real values of number of infected, clearly sick, recovered and dead as well as so called active cases in Poland.

Keywords: *Covid19 Pandemic; Potential Infection Ceilings; Virus Multiplication*

What about so called "first-early -wave of pandemic"? An more exact analysis of statistical records for Poland and Belgium [Eadie-Hofstee plot-with number of viruses applied]

And now promised Y direction of analysis. What about that "surplus of statistical reports versus my calculations" - clearly visible from the beginning of pandemic-March to almost September ? There are only two possibilities: either my data are understated or the statistics

are too high.But in spite i do not estimate/believe the statistics as the reliable measure of the pandemic [compare earlier text] I can't expect that statistical data show us some mysterious excessive pool of cases of infection. So rather i had to omit quite a lot of cases in my calculations. But why and what cases?

Still with table 8. I tried to remove this "excess of early cases of infection". So I substracted the value about the plateau of this "excess" i.e. something like "the first wave of pandemic"-i.e. about 40000 infected [compare bit later] which is only a small percentage of the number of people reported as infected about November. Such an operation in fact annihilates "excess" ["first wave"] but even giving us the difference equal less than zero -without distinct decrease "the second wave [from half of September]. But if we substract just 80% of calculated "first wave" [compare bit later] we still obtain the numbers of infected in very first stage of pandemic although much smaller but still bigger than mine calculated. But from about half of May until all the October such corrected values represent about constant fraction of amounts calculated by me [less than 5% of mine].

Then I decided to examine the reported amounts of infected people from April to December in different countries, taking for granted that they are related to the assumed amount of viruses in the cloud [compare earlier]. In other words, I decided to treat the cumulative total number of infected persons curve as the sum of two curves: „earlier" and „later".

To eliminate the assumption that the statistical data in Poland are not entirely reliable, I decided to count it for other countries. Initially I chose Belgium, Hungary, Estonia and New York State in the U.S. Why? Belgium and New York [I wanted to find data for the New York metropolitan area, but found only for New York State] I chose because of the confidence in the statistical data and the high degree of pandemic development. But in general, I have chosen small and fairly homogeneous areas [without drastic differences in the topography and development of subregions]. Finally, I did not show the data for Estonia and New York State.

My approach and the results obtained are presented in table 9 and figure 9.

Thus, from the total number of infected [data cumulated per one million population] in Poland I first subtracted 1100 and in Belgium 7000, which is approximately the "plateau of the first wave". Then I put such a data together with the number of viruses [„in the cloud" - per million inhabitants]-as in my previous calculations. I assumed

Date	n	Number of viruses	Poland						Belgium				
			Number of infected persons I						Number of infected persons I				
			My calculations				I Statist. Recorded S	M/S % ..... as above but version II for gr. I-2	My calculations			I Statist. Recorded S	M/S %
			Group I-1	Group I-2 version I	Group I-2 version II	Σ I = (I-1)+(I-2) M ..... as above but version II for gr. I-2			Group I-1	Group I-2	Σ I = (I-1)+(I-2) M		
1.04. 2020	20	2,097 x 10^8	44	0	0	44 44	61	72,1 72,1	1713	0	1713	1502	114
22.04. 2020	22	8,389 x 10^8	158	0	0	158 ... 158	260	60,8 60,8	3549	0	3549	3976	89,3

13.05 2020	24	3,335 x 10^9	440	0	0	440 440	447	98,4 98,4	4847	0	4847	4876	99,4
3.06 2020	26	1,342 x 10^{10}	802	0,51=1	0,44=0	803 802	645	124,5 124,3	5335	0,6=1	5336	5238	101,9
24.06 2020	28	5,359 x 10^{10}	1007	2,04=2	1,81=2	1009 1009	859	117,5 117,5	5473	9,7=10	5483	5433	100,9
15.07 2020	30	2,147 x 10^{11}	1075	8,16=8	7,24=7	1083 1082	916	118,2 118,1	5508	39	5547	5621	98,7
5.08 2020	32	8,59 x 10^{11}	1093	33	29	1126 1122	1272	88,5 88,2	5517	155	5672	6400	88,6
26.08 2020	34	3,436 x 10^{12}	1099	131	116	1230 1215	1666	73,8 72,9	5520	612	6132	7381	83,1
16.09 2020	36	1,874 x 10^{13}	1100	516	463	1616 1563	1985	81,4 78,7	5520	2375	7895	8721	90,5
7.10 2020	38	5,498 x 10^{13}	1100	1989	1782	3089 2882	2756	112,1 104,6	5520	8506	14026	12955	108,3
28.10 2020	40	2,199 x 10^{14}	1100	6956	6523	8056 7623	7704	104,6 98,9	5520	23965	29485	34849	84,6
8.11 2020	41	4,4 x 10^{14}	1100	11912	11646	13012 12746	13783	95,1 92,5	5520	34386	39906	43408	91,9

19.11 2020	42	8,8 x 10^{14}	1100	18502	19176	19602 20276	20420	96,0 99,3	5520	43928	49448	47093	105
26.11. 2020	42,67	1,397 x 10^{15}	1100	20324	25208	21424 26308	24866	87,7 107,7	5520	48955	54475	48969	111,2
29.11 2020	43	1,76 x 10^{15}	1100	25579	28338	26679 29338	26028	103,4 113,7	5520	51005	56525	49751	113,6
10.12 2020	44	3,52 x 10^{15}	1100	37231	28421	38331 29521	29120	131,6 101,4	5520	55473	60993	51805	117,7

Table 9: *The comparison of my calculations of number of infected persons with the statistical records per one million habitants. In Poland And Belgium[but with separate two groups of people: I {A]and II [B] {C]alias for"two waves of pandemic: early[I] and main [II]"]. Constants for calcularions: Poland – group I: Imax= 1100 [0,11% of population]; Ka =5 x109. Group II – version I: Imax =54260 [5,43% of population]; Ka =1,61 x 1015. Group II- version II: Imax =41420 [4,14% of population]; Ka =1,09 x 1015. Belgium- group I: Imax = 5520 [0,55% of population]; Ka =4,66 x 108. Group II: Imax =60800 [6,08% of population]; Ka =3,38 x 1014. The number of viruses [in total cloud- per one million of habitants -ie supposedly from one „person zero"] was calculated according to equation: V = 200 x 2n [where n means number of succesive duplication period [t(2)] which length is 10,5 days].*

that the number of viruses is the same in Poland and Belgium on the same days [Well, taking in account the very slow initial-from infection of "person zero" with say 200 viruses taken- growth of total number of viruses such an assumption is quite justified: 21 days means there is e.g. 800 instead 200 viruses]. I assumed - as in my calculations so far [cf. summary table 7] that April 1 is the last day of the 20th duplication period [for viruses]: n = 36 according to my initial estimations [see much earlier].

Then, according to the formula {5}, there are [per million inhabitants-with a default of one "person zero"] 2,097 x 10^8 viruses. These and the following [For the following dates - in table 9] viruses' numbers were substituted for Hill equation {6} to obtain a calculated number of infected people. But first, the Hill equation constants, I_{max} and K_a, had to be established. Thus, I applied Eadie-Hofstee plot [77] of Hill equation [i.e. in fact Michaelis -Menten equation [59]] to linearize it.

Eadie -Hofstee plot obeys the equation: $v = -K_M \times v / S + V_{max}$...{9}

[Where v is reaction velocity, V_{max} - maximal velocity, and K_M is Michaelis' constant. With my assumptions, the following formula becomes:

$I = -K_a \times I / V + I_{max}$.....{10} [designations identical as in formula {6}]

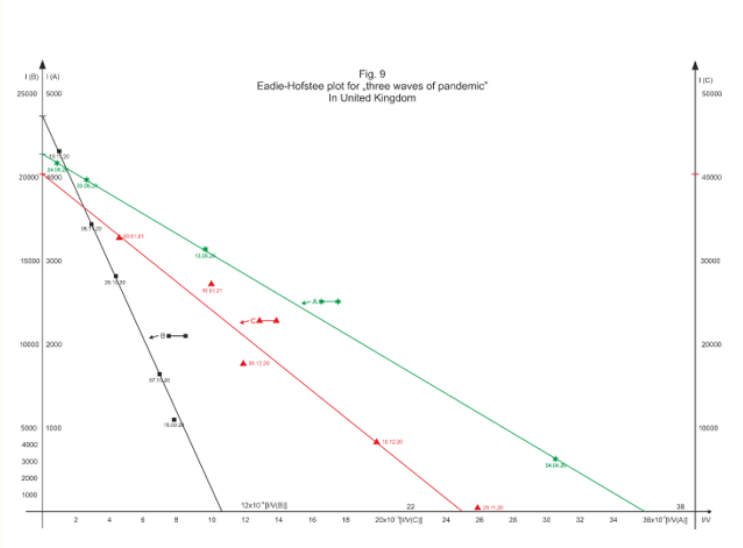

Figure 9: *The Eadie-Hofstee plot for "three waves of pandemic" in United Kingdom.*
On y axis: the number of infected people I [per milion of habitants] in Great Brittain [according to statistical records].
On x axis: the values of ratio I/V, where V is the total number of viruses in the entire population [that is, in the "cloud of human breaths and viruses"] - per million people - calculated according to the formula V = 200 x2n [my assumptions], where n show the number of successive doubling periods [of viruses] ;n=10, 5 days.The data come/are consistent with from the table 11.

Note [!], figure 9 presents data for United Kingdom [groups A, B and C i.e. respectively I, II and III]-compare table 12, but the method is always the same [so there is no point give many identical straight lines; of course the curves' slope and the scale on the x and y axes is different, adapted to the data [thus respectively the number of infected I on the Y axis and the ratio of the number of infected people to the number of viruses - on a given day "in the cloud" - that is I/V on the x axis].

From this plot/s [Figure 9] I obtained both I_{max} and K_a for Poland and for Belgium both for "something like the early/first wave of pandemic"-March-August [I_{max}^{I} and K_a^{I}] and for the "main wave of pandemic" ["later wave" or "second wave"-current one i.e. from the beginning of October]- I_{max}^{II} and K_a^{II}.

For "early wave" [i.e. that apparent excess of reported versus mine calculated data in table 8, I told earlier]-per million of habitants:

In Poland: $I_{max}^{I} = 1100$ and $K_a^{I} = 5 \times 10^9$

Aand for Belgium $I_{max}^{I} = 5520$ and $K_a^{I} = 4,66 \times 10^8$

And for the main wave ["second wave"]

In Poland: $I_{max}^{II} = 41420$ and $K_a^{II} = 1,09 \times 10^{15}$ [version Z]

And for Belgium $I_{max}^{II} = 60800$ and $K_a^{II} = 3,38 \times 10^{14}$

However such an estimation is not very exact [which is?], I mean that at some dates there are quite distinct deviations from the curves/lines drawn. And so for Poland there might be alternative values: $K_a^{II} = 1,61 \times 10^{15}$ and $I_{max}^{II} = 54260$ [version W].

And now we can calculate-with Hill equation- the total numbers of infected persons in the following days- applying the amount of viruses [estimated as I explained earlier] and taking the values of I_{max} and K_a obtained from Eadie-Hofstee linearizing plot [from both statistical records and my assumed amounts of viruses]. Of course one has first to calculate number of infected I for first set of constants [I_{max}^{I} and K_a^{I} - "first wave"], next for second set [I_{max}^{II} and K_a^{II} - "second-main wave"] and finally sum them.

Table 9 shows us clearly that both for Poland and Belgium such sums are almost identical with the statistical records. What is more- what at first might be looking strange-calculations for Poland give about the same figures and % [comparing to statistics] regardless we put the values of constants from above version Z or W. Most probably it is because the ratio of I_{max} to K_a is very close in both mentioned versions. Nevertheless the maximal total cumulative number of infected people in Poland resulting from these calculations seems to be strikingly different in both versions ": 2,084 million [version W] and just 1,59 million [version Z]. Mind you anyway I think that real cumulative number is more than 5 million [compare later]!

So I proved that:

1. My calculations give the results consistent with the reality in spite my calculations apply the amount of viruses what is highly unique [nobody in the world does some thing like that]

2. There are really likely two waves of pandemic exist: early [which is over about August/ September] and late/second or rather main one [current one?-compare later]

3. These waves [or phases] are very different, because:

    a. First concerns much smaller number of people than the main one

    b. First is much more quick in the beginning because there is incomparably lower K_a value. It means there is much higher apparent affinity of an infecting -anotherwords much, much lower amount of viruses is enough for infection and most probably also for resulting in getting sick, seriously sick and…. falling dead [compare later].

4. The fact that the analysis of statistical data shows the existence of a "borderline of infections" I_{max} [and as can be assumed by analogy-border of number of sick, healed and died from Covid19] is a strong [albeit indirect] confirmation that such a border exists ergo that only a specific people become infected [or more precisely multiplying viruses after receiving a potentially infectious dose], while others will not become infected because they do not have functional SARSCoV-2 receptors.

Serious evidence that in fact only some people have functional receptors for SARSCoV-2 [and others do not have them or have their levels very low, below a certain critical value] consists in the enormous amount of facts that many of us do know, in spite they are not recorded in the world statistics. It is about: very numerous cases that in one family living together many people do not get infected, even though other members of this family are infected [as confirmed by the tests], and even have symptoms, sometimes even severe, even died of „pulmonary ailments" [there are particularly many situations: an infected husband and a wife not, or *vice versa*]. Very often, even usually, one of the entire football team is infected, and it infects, for example, two, but not a further 12 players, training together and staying together very often and for a long time.

The same applies to people staying together in a sanatorium, a nursing home, a boarding house, or the famous ghost giant cruise ships that sailed for months because they were not allowed into any port. The fact that infected, but already sick [symptoms!] persons were isolated is no argument, because isolation [just after positive test done] usually only occurred when these people were already sick, *ergo* long infected and potentially contagious for a long time.

Two groups of infected persons-not two waves of pandemic!

However, I suggest clearly another interpretation of the term „two/three successive phases/waves of a pandemic". Look once again at table 9. The people infected - with an evidently „low affinity for viruses" [K_a of 10^{15}, not 10^9] do not appear in the first period of the pandemic, but there are already - although there are very few of them - in June and July. So rather, I believe that there are two/three [compare later] types of people being susceptible [i.e. with functional SARSCoV-2 receptors]: the first in whom the pandemic develops very quickly [*ergo* evidently reaches its ceiling much earlier] and the second in which the pandemic develops much more slowly [i.e. with a considerable delayed appearance of infection, morbidity, death, but also the production of specific antibodies].

And who are these people with such an evident delay in the development of symptoms? They are simply those who have a blockage for viruses in their blood [in the form of quite a distinct amount of the serum ACE2 released by ADAM17-sheddase-and a certain percentage of erythrocytes, perhaps already attacked by ROS].On the contrary, people from groups A ["very first wave"] and C [so called "third wave"-with "British strain"-see later] have the deficits of blocking sites for viruses [see later].

Now let us compare table 5 and 6 describing and taking into account the block - and my earlier text on inhibition of infection by V-SA [or V-B] complexes [Section 5, this part] and the resulting apparent decrease in the affinity of target cells for viruses resulting in a significant increase in K_a. I assumed then that it would be an increase from $K_a = 10^{12}$ to 10^{13}. But now I can see that it could be a much bigger increase, for example from 10^9 or 10^{10} to 10^{13} and more. In some early verses of table 6 in brackets {} are shown the numbers of infected which might be observed with $K_a=10^{11}$ -just horror.

By the way the mentioned values fully explain my earlier calculations [subtraction, multiplying by 0, 8…], where then I had not explained some figures. That was done and shown just to illustrate/highlight that an apparent surplus in amounts of infected people recorded in the pandemic statistics in comparison with my calculated data clearly almost disappears when we practically remove the „first wave of the pandemic" [March - July/August 2020]. In fact we substract quite a few people practically not having a block for SARS CoV-2 in the blood [or actually having very small amounts of it].

Summarizing, it was not the statistical data that had a surplus, but I simply did not take into account the presence of people „without a block" in my calculations. In my very early considerations i tried to calculate the course of pandemic just after the block. Later on I completely changed an interpretation assuming every body infected comes into block phase which is over when all blocking sites are saturated. Life is richer than the mind of man - I just could not even predict before that there might be people practically without a block for viruses. This is a group for which K_a is very small, [ie the amount of viruses needed to cause a half-effect, i.e. $I = I_{max}/2$]. Thus, the number of infected people [in this supposed „first wave of pandemic"] grows [relatively] very quickly and quickly reaches the ceiling level.

Indirectly, this piece of calculations is very good [though indirect] evidence that the blockage for viruses [in the blood] actually takes place in the vast majority of people. If there were no block, the consequences would be dire. Just at the beginning of July this year, almost all people in Poland - of all age groups, including young children - [but this applies to virtually all seriously affected countries in the world - I'm not talking about China] would be infected. Mind you, the number of sick people, including very serious sickness' cases, and those who died [in a possible causal relationship with the amount of viruses] would be shocking [compare later]. If only people who were really susceptible, i.e. those with functional SARSCoV-2 receptors [as I believe], would be infected, the effects would be tragic either in spite being smaller - over 90% of the population would not be infected, but still over 15% would [over 5 and half a million in Poland] already August 2020.

And now what about the world. If we look on curves of total cumulative cases of infection for many countries and areas of them we straightaway could see that in many, many of them the situation is about the same [in spite some quantitative differences] as in Belgium

and Poland-You can list here France, UK, Spain, Italy, USA and Russia. And in USA such a picture dominates ie the very similar type of curve is in majority of states including those states which unexpectedly excell in this sad statistic as South and North Dakota, Iowa and Nebraska [text from November 2020].

Table 10 shows us the same comparisons for three other countries:the Netherlands, Hungary and Portugal.

Date	Number of viruses V	Netherlands				Hungary				Portugal			
		Number of infected persons I				Number of infected persons I				Number of infected persons I			
		My calculations			Statist. Recorded S [M/S %]	My calculations			Statist. Recorded S [M/S %]	My calculations			Statist. Recorded S [M/S %]
		Group I-1	Group I-2	Σ I = (I-1)+ (I-2) M		Group I-1	Group I-2	Σ I = (I-1)+ (I-2) M		Group I-1	Group I-2	Σ I = (I-1)+ (I-2) M	
1.04.2020	2,097 x 10^8	839	0	839	772 [108,7]	54,7= 55	0	55	54 [101,9]	844	0	844	803 [105,1]
22.04 2020	8,389 x 10^8	1929	0	1929	1975 [97.7]	160	0	160	222 [72,1]	1925	0	1925	2138 [90,0]
13.05 2020	3,335 x 10^9	2855	0,43= 0	2855	2447 [116,7]	306	0	306	342 [89,5]	2832	0,47= 0	2832	2737 [103,5]
03.06 2020	1,342 x 10^{10}	3245	1,7= 2	3247	2646 [115,2]	396	0,46= 0	396	402 [98,5]	3210	0,95=1	3211	3236 [99,2]
24.06 2020	5,359 x 10^{10}	3360	6,8=7	3367	2819 [119,4]	428	1,86= 2	430	421 [102,1]	3321	3,8=4	3325	3901 [86,1]
15.07 2020	2,147 x 10^{11}	3390	27,3= 27	3417	2901 [117,8]	437	7,46= 7	444	436 [101,8]	3350	15,2= 15	3365	4613 [72,9]
05.08 2020	8,59 x 10^{11}	3397	109	3506	3199 [109,6]	439	30	469	476 [98,5]	3357	61	3418	5044 [67,8]
26.08 2020	3,436 x 10^{12}	3400	430	3830	3967 [96,5]	440	119	559	541 [103,3]	3360	244	3604	5474 [65,8]
16.09 2020	1,364 x 10^{13}	3400	2200	5600	5097 [109,9]	440	471	911	1501 [60,7]	3360	943	4303	6384 [67,4]
7.10 2020	5,498 x 10^{13}	3400	5633	9033	8764 [103,1]	440	1806	2246	3389 [66,3]	3360	3444	6804	7904 [86,1]

28.10 2020	2,2 x 10^{14}	3400	14276	17676	18394 [96,1]	440	6208	6648	6749 [98,5]	3360	10207	13567	12489 [108,6]
08.11 2020	4,4 x 10^{14}	3400	19176	22576	23495 [96,1]	440	10451	10891	11220 [97,1]	3360	15170	18530	17444 [106,2
19.11 2020	8,8 x 10^{14}	3400	23150	26550	26755 [99,2]	440	15876	16316	16526 [98,7]	3360	20044	23404	23639 [89,6]
26.11 2020	1,397 x 10^{15}	3400	25072	28472	28814 [98,8]	440	19651	20091	19657 [102,2]	3360	22748	26108	27276 [95,7]
29.11 2020	1,76 x 10^{15}	3400	25825	29225	29726 [98,3]	440	21441	21881	21651 [101,1]	3360	23879	27239	28677 [95,0]
10.12 2020	3,52 x 10^{15}	3400	27049	30809	33567 [91,8]	440	25998	26438	27124 [97,5]	3360	26406	29766	32608 [91,3]

Table 10: *The comparison of my calculations of number of infected persons with the statistical records per one million habitants. In netherlands, hungary and portugal [but with separate two groups of people: I {A]and II [B] {C]alias for"two waves of pandemic: early [I] and main [II]"] Constants for calcularions: Netherlands– group I: Imax= 3400 [0,34% of population]; Ka =6,4 x108. Group II: Imax =29200 [2,92% of population]; Ka =2,3 x 1014. Hungary- group I: Imax = 440 [0,044% of population]; Ka =1,475 x 109. Group II: Imax =33015 [3,3% of population] ; Ka =9,5 x 1014. Portugal– group I: Imax= 3360 [0,34% of population]; Ka =6,25 x108. Group II: Imax =29530 [2,95% of population]; Ka =4,165 x 1014. The number of viruses [in total cloud- per one million of habitants -ie supposedly from one „person zero"] was calculated according to equation: V = 200 x 2n [where n means number of succesive duplication period [t(2)] which length is 10,5 days].*

The consistency of my calculations [and thus :assumptions] with the statistical records is very good. There's no point to discuss these data in detail nor elongate the list of countries so studied. May be just note: 1. much lower abundance of people apparently lacking block in Hungary and 2. some discrepancies- about July and August- for Portugal. But if one applied -for Portugal-bit higher I_{max} for both "first and second waves of pandemic" [such denominations are easy in spite being incorrect] eg 4000 instead 3350 for I wave and 31000 instead 29530 for II wave the consistency would be excellent.

Well, my calculations agree with statistics. So why I do not believe in statistics? It should be expressed in another way! I believe in statistics but simply i have quite another way of interpretation. Simply the statistics give too low amounts of infected persons [thus showing new daily cases of infection and rejoicing if those numbers go down] as most probably almost all susceptible people are already infected [Table 7].

Why I am thinking this? Simply the values of K_a i.e. amounts of viruses needed to achieve half-effect i.e. 50% of infected people [From table 9 and 10] are too high. Just compare them [3 x 10^{14} - 1,1 x 10^{15}] with those in table 4 but not just for infection, but also for sickness, producing specific antibodies and death cases. The K_a values obtained for statistically reported cases of infection [Table 9 and 10] are about the same value as those shown in table 4 but…. for sickness, producing antibodies and even are not very much smaller than those for….death, being in any case rather delayed symptom of pandemic. They are an order of magnitude higher than those shown in tab.4 for resulting an infection. It can't be K_a values for infection are so high!

Well, but somebody might tell that these high values put into Hill equation gave the results consistent with statistical reports. That's right but anyway in spite there were very first cases of infection - belonging to this "main wave of pandemic" [group II-table 10]- about half of May in Belgium and beginning of June in Poland [Table 9] the tangible amounts, i.e. above 100 per million inhabitants, were observed only in August. This is 5 months later than the assumed and reported time of the start of the so-called pandemic, but according to my calculations it is already 10 months from the start of the pandemic, i.e. from the infection of the first „zero individual" [actually few such persons]. If we assume [for infection for the "main wave of pandemic" ie for susceptible people clearly containing the blocking sites in their blood] K_a be one order of magnitude smaller ie about 1-5 x 10^{13} the tangible amounts of infected persons [of that kind] would appear even in April [compare table 6-8]. Of course I mean K_a for infection reported in statistics for all countries mentioned in table 9-11.

On the other hand one could tell that such a high K_a values observed "for the main wave" just result from inproper choice of amount of viruses [about 2 x10^8 at 1 April- n=20-, so 200 viruses 210 days before, as 20 x t(2) ie 20x10, 5=210days-so assuming that our pandemic had begun in Europe-in China much earlier-in September 2019, ie much, much earlier than accepts it in the general opinion]. Indeed if we assumed less viruses is in the cloud about October-December 2020 lower value/s of K_a would be obtained in Eadie-Hofstee plot [or any else] -not similar to those for sickness and even death. But if you lower the amount of viruses in later part of pandemic you have to lower them for the earlier part either. But then the amounts of viruses "resulting the first [early] wave of pandemic" would have had to be improbably low. And even now they are looking very low- with K_a [for Poland, „first wave"] equal 5 x 10^9 the first infection ever would be observed with 4,4 million of viruses [in all the cloud], so at n = 14 ie 63 days before April 1st, thus about January 27th. With K_a 10 times lower [proportionally to such lowered K_a for main-current wave] first infection should be reported about December the 20th 2019.

I recalculated this more exactly assuming that the statistical data only for „the first wave of pandemic" [should be written "for first group of people -practically lacking the block"] concern in fact already sick people [very mildly however] not just infected.Then I assumed that average K_a for just infection is 71,4 times less- so 7 x 10^7 instead 5 x 10^9, but I_{max} is 2, 618 times more ie 2880 instead 1100. It is in an accordance with constants for K_a and X_{max} for infection and sickness shown in table 4 [and taking mean values for 4 different age groups]. Then first infection seems to have been at 14th November 2019 with128 infected at January 28th, 1229 at March 11th, 2658 already at April 22nd.

But look, if I added such the data to the previously calculated amount of infected persons [Table 7]-it does not change very much the whole picture -after September the 1st - as this first group of infected [*alias* "first wave"] is just a few [comparing with "main/? current pandemic"]. But of course such an operation resulted in an elimination of that strange surplus of statistical records versus mine calculation' results -from March to October 2020.

But the question is if those people -lacking the block-infected so early-would survive until now. Certainly not. My estimation [I do not show these data explicite just not to excessively prolong an already long work] based on analogy, similar to using with the arithmeric model in table 3, shows us that already at July the 15th there were about 16 -per milion-thus 608 death cases of those "first wave of pandemic". So it might be that about 1/3 to ½ of those people did not survive till now. Survivors might be even seriously ill.But some of them recovered [compare later].

From about June we have infected people evidently of two kinds/groups. [Notice, the people of all kinds exist all the time but the peak of infections for these groups takes place at different time]. Well, it turned out recently that there might be the third group of people ie alias "third wave of pandemic". I mean "the strain of virus" supposedly discovered in the end of November 2020 in United Kingdom [or that supposedly passed from the South Africa]. The problem is whether there are first, second, third mutations of virus [rather sets of mutations] or first, second and third wave of pandemic result from an existence of first, second, third kind/subpopulation of susceptible people? And if the third is the last one in this pandemic [i.e yet before the end of this pandemic]?

I will briefly discuss these issues bit later.

[Well, anyway one gets the impression the results for this "early wave of pandemic" fit more into the calculations of my arithmetic model „2 x 2 ... x2" than to the Hill equation, which works great for the later phases].

General remarks on the related situation in various countries around the world. Kinetic constants of viruses' binding and "the waves of pandemic"

So there is in the whole World one great long lasting pandemic with two [or rather three-see a bit later] waves resulting from the existence of groups of affected people, as I described earlier. First is less numerous group [about 1,1 pro mille in Poland, about 5,5 promille in Belgium, 4,5 promille in United Kingdom, only 0, 44 promille in Hungary, but probably much more, maybe even 2% in New York] who almost do not contain block.

It might be that shedding activity of ADAM17 against membrane ACE2 is somehow down regulated up to almost zero in this group of persons. Then blocking activity might be limited just to some very small fraction of erythrocytes, according to my assumptions-table1-erythrocytes give from only 14% of total blocking affinity in the youngest group [< 25 years old] up to 84% for 65+. Yet it might be that for some reasons also the erythrocytes of such a subgroup much, much weaker bind to the viruses.

The second- dominating- group -its abundance depends on age, [and in my opinion also on some genes related to the so-called race/nationality, i.e. being more common in their representatives' genotype], thus the amount of functional receptors of SARSCoV-2 in the body], contains an effective blocking system for viruses. In Poland it is almost 14% of all people, in Belgium probably not less than 24%. My initial estimates for China, possibly - albeit to a lesser extent - also for Japan and Korea, would point to: 1/ very small % of susceptible people and 2/ surprisingly - domination among the population - ie susceptible people - people „of the first type, ie with block deficiency. In these countries, the pandemic developed rapidly [despite appropriate administrative measures „limiting" social contact"], but also quickly reached its ceiling. The other „kind of second wave of pandemic" [second subpopulation of people which appeared to be dominating in Poland and other countries i mentioned till now] is practically in China non-existent so far.

I would not like to „suppose" without sufficient arguments, but I vaguely see that for people of type I effects of infection [almost the only one observed in China] are more limited to the dangerous pulmonary symptoms [with heart disease and that of other tissues/organs arising indirectly rather than through attack of viruses]. For type/group II, which is dominant in the world [bit later i discuss the presence of type III], in addition to its course delay, very frequent, non-pulmonary type of disease might evolve.

It means that the first explanation of observed statistics of infection [I do prefer it]is the existence of two [bit later I will propose existence of three] groups/subpopulations differing with an affinity of their bodies to the viruses.

But there is another possible explanation for this supposedly first and second current wave of the pandemic. It would try to explain them not as manifestation of different subpopulations of people [possible recipients of virus] but as manifestation of quite new mutation of virus [rather set of mutations]. Below I will discuss this possibility in more detail i.e. what about binding of RBD [receptor binding domain] of virus with both functional membrane receptor of SARSCoV-2 [and serum ACE-2].

It is unlikely but cannot be completely ruled out. I will be discussing such a possibility in terms of kinetic constants of binding with virus not just molecular nature of eventual mutation. Let us imagine that the virus mutated in a special way quite early, already in about half of June [or even earlier]. This mutation caused that the mutant virus has a lower apparent affinity to the functional receptors than that earlier original virus. But here it is necessary to delve a bit again into enzymatic kinetics [to which, as I wrote before, the binding of the virus to the receptors shows a serious analogy].

Well, the measure of the affinity [of the enzyme to the substrate], that is the Michaelis constant $K_M = k_2 + k_3/k_1$ ……..{11}

Where k_1 is the rate constant for the formation of the enzyme-substrate complex, k_2 is the constant of the decomposition of this complex into free enzyme and substrate, and k_3 is the constant of the rate of the „appropriate reaction" [product formation -the slowest one] [57]. So in our situation K_a - measure of the affinity of the virus [its RBD] to the membrane ACE2 [but together with "other elements" in the membrane - i.e. the functional SARSCoV-2 receptor]:

So $K_a = k_3 + k_2/k_1$……..{12}

Where k_1 is the rate constant for the formation of V-FR complexes, k_2 for their decay, k_3 is the slowest reaction in the cascade of multiplication processes *ergo* an infection [in fact we don't know which exactly molecular event play there such a role]. Now imagine that the K_a value for this „early" virus is very low [*ergo* receptor affinity and thus infectivity is very high] due to the very high k_1 value [of „virus recognition"], but to some extent due to the low value of k_2 that is, the V-FR complex practically does not disintegrate [which would „prevent infection once"]. What's more, k_1 is so high not because of some particularly easy *ergo* quick recognition, but somewhat secondary i.e. that even initially recognized virus is almost immediately drawn into the target cell [thanks to fusion mechanisms involving priming enzymes and microdomain rafts]/i.e. the measurable values of k_1 might be very high in spite its real values -for some reasons impossible to be evaluated-are not so high; high measured k_1 values might come from an interfering with kinetic constants of some further steps of process.

But as an important element of FR consists in (membrane) ACE2 [mSA] it can be imagined, that analogously serum ACE2 [simply: SA] might bind viruses: V + SA ⇔ V-SA, where the rate constants of this reaction to the right and left are k_4 ["equivalent" to k_1] and k_5 ["equivalent" to k_2], respectively. And for this" early virus/es", the value of k_5 is so great, and k_4 so small that there is virtually no V-SA complex formation, and if they do, they immediately disintegrate.

For reaction V + SA ⇔ V-SA the equilibrium constant $K=k_4/k_5$ ……..{13}.

And now the mutation causes the value of K_5 to decrease very much and the value of k_4 to increase, so the equilibrium of the reaction shifts to the right and it is very easy to form V-SA complexes, i.e. to block the virus. However, at the same time, k_1 apparently but indirectly [!] decreases as relatively less viruses bind to the functional receptor [with quite a lot of them binding to serum ACE2] and so an apparent affinity of virus to the functional receptor decreases *ergo* K_a increases [for infection]; fusion of the viral envelope with the membrane still plays a major role [prevailing]. Although we cannot exclude such a possibility, but rather it is minimal, especially considering that usually in Science variants that are easier to explain should be chosen.

"Third wave of pandemic". Where/how it comes from?

And now for the last thing and somehow „last minute". It's about this „new variant of SARSCoV-2 discovered in England around December 1. Supposedly it has a greater ergo easier infectivity. So it resembles the „first wave" of the virus [around March]. And now is this caused with a significant new mutation [or maybe a set of several mutations and not necessarily only in RBD]?

But what is important here is whether this „second-and whatever else- wave" was the result of a mutated - as I wrote earlier - the virus [i.e. resulting in the change of reaction rate constants: k_4 and so on] or the result of - I wrote about it either- the existence of a special group/subpopulation of people - practically devoid of block in the blood for the virus?

So is the genetic apparatus of potential recipients [the „no block" group] or the genome of the virus itself [its mutations] of key importance ?! If the "last wave" concerned only the people apparently lacking of the block sites and this group was only one i.e. the same group as that being most affected during the very first wave then one could argue that: 1/ this group was relatively small, it got infected quickly, 2/ a large part of them died before November 1 and just around December 1 of such a group of people already does not exist.

Is it possible then „the emergence of people without a block for viruses"? At first glance it seems there is only one possibility. A certain - perhaps a large- percentage of people in this group previously infected [with this „fast path of infection"] - „as if healed", ie the viruses stopped multiplying, but these people did not acquire anti-SARSCoV-2 immunity and got infected:second time.

From who? It is not important, because with such an interpretation it is just important that the recipients of the virus have practically no block. But you can imagine that also the virus donor was a person - let's call him Q - maybe just before he died - not having a block for viruses who got infected around May 20. As I mentioned it is not important.

If that was the case it would mean that the number of potential recipients is negligible - about 1 per mille [because only those a/ without block b/ who survived and/who somehow recovered - maybe this recovery did not have the main immune component - maybe the viruses were destroyed by proteolytic enzymes, by ribonuclease/s or by ROS].

However, if these infections with the „new strain" are the return of the original virus [let's call it „early" because it may be the same form as it was at the beginning of 2020], where did it come from? After all - as I wrote before - it mutated around May 2020 [although it is rather problem of date when there is much bigger invasion of such viruses not the time of first appearing of mutation itself] - turning into a „slower" form [read: "easily binding with the block"]. Maybe it passed from people to some animals and „now", i.e. in early December it switched from these to people again?

Or maybe such a virus was preserved in the body of the previously „indicated" Q individual?

The situation then would become dangerous, because then all susceptible persons could become infected [including the repeated infections], especially when a person already infected - deprived of immunity - has only very few viruses of „former" form in the body]. And despite having a block, this „new virus" - as I explained, in fact the „old virus" - ie. from the beginning of 2020 - the block would not react. and, in turn, having two types of virus in the body of one infected person- „the one with greater infectivity, ie with a faster progression, will win.

If in such a case the numbering of viruses [ie estimation of total number of viruses in the cloud/population-which is to apply in my mode of calculation] - would start from the beginning? I do not think so, because all the time viruses somewhere multiply not necessarily infecting new persons. We must think that the majority of viruses is located in the bodies of people being infected long time ago.

Here I have to emphasize that I do not mean that the third [British, South African] mutant/strain is the same as those from the first [March] wave. It is certain that a lot of point mutations took place from March to November 2020. I would like simply to emphasize that like the viruses from the „first wave of the pandemic", it has/they have ["British strain"-compare later] virtually no affinity for the blocking sites in the blood, similar/identical to „first wave viruses".

Recently all talk on the "third wave of pandemic" because there are announcements on new strain [mutation/set of mutations] of SARSCoV-2 discovered in United Kingdom in November 2020. Till now every beginning of a „new wave of pandemic" has manifested itself in a rather sudden sharpening of the steepness of the curve of cumulative cases of infection [on a logarithmic scale], that is, the curve begins to flatten out, and here it „quite suddenly" becomes sharp [] „bends up"]. And this is the case from the second half of November 2020 - especially in United Kingdom, but also a bit less clearly in Poland. But this phenomenon is already noticeable in many countries of the world [including those presented in table 10 including Portugal].

Estimation of constants for "the third wave" i.e. for an infection of people of group/type C-an analysis of three groups in United Kingdom and Poland [once again]

Thus, I decided to calculate [for United Kingdom and also Poland] the number of infected people -analogically as I did before [Table 9 and 10] but [!] assuming there are three, not just two, subpopulations of people [A=I, B=II and C=III]. I estimated once again values of

constants [K_a and I_{max}] for these three groups with Eadie-Hofstee linearizing plot [exactly as I described earlier]. Then I calculated separately the number of infected people from supposed three separate groups/subpopulations. And once again I compared the abundance reported statistically with my calculations. As we can see in table 11 there is full consistence of data.

Date	n	Number of viruses	United Kingdom						Poland					
			Infected						Infected					
			My calculations				Statist.	ΣM/ Σ Stat.	My calculations				Statist.	ΣM/ Σ Stat.
			Group A	Group B	Group C	ΣM= A+B+C	Σ Stat.	%	Group A [% ΣM]	Group B [% ΣM]	Group C [% ΣM]	ΣM= A+B+C	Σ Stat.	%
01.04 2020	20	2,097 x 10^8	642 [100]	0	0	642	645	99,5	44 [100]	0	0	44	68	-64,7
22.04 2020	22	8,389 x 10^8	1825 [100]	0	0	1825	2085	87,5	158 [100]	0	0	158	269	-58,7
13.05 2020	24	3,335 x 10^9	3266 [100]	0,28=0	0	3266	3273	99,8	440 [100]	0	0	440	455	96,7
03.06 2020	26	1,342 x 10^{10}	4114 [100]	1,14=1	0	4115	3856	106,7	802 [99,9	0,7=1 [0,1]	0	803	652	-123,2
24.06 2020	28	5,359 x 10^{10}	4397 [99,9]	4,53=5 [0,1]	0	4402	4151	106	1007 [99,7]	2,8=3 [0,3]	0	1010	867	116,5
15.07 2020	30	2,147 x 10^{11}	4474 [99,6]	18,4=18 [0,4]	0,49=0	4492	4323	103,9	1075 [99]	11,2=11 [1]	0	1086	1023	106,2
05.08 2020	32	8,59 x 10^{11}	4493 [98,4]	73 [1,6]	1,93=2 [0]	4568	4549	100,4	1093 [96]	45 [4]	0,47=0	1138	1289	88,3
26.08 2020	34	3,436 x 10^{12}	4500 [93,8]	291 [6,1]	7,74=8 [0,2]	4799	4875	98,4	1099 [85,2]	179 [14]	1,91=2 [0,2]	1280	1686	-75,9
16.09 2020	36	1,374 x 10^{13}	4500 [80]	1096 [19,5]	30,6=31 [0,5]	5627	5608	100,3	1100 [60,8]	700 [38,7]	7,64=8 [0,4]	1808	2001	90,4
07.10 2020	38	5,498 x 10^{13}	4500 [53,8]	3741 [44,7]	124 [1,5]	8365	8057	103,8	1100 [29,2]	2636 [70]	31 [0,8]	3767	2836	-132,8
28.10 2020	40	2,199 x 10^{14}	4500 [31,2]	9429 [65,4]	489 [3,4]	14418	13926	103,5	1100 [11,3]	8550 [87,5]	122 [1,2]	9772	7902	-123,7

8.11. 2020	41	4,4 x 10^{14}	4500 [24,9]	12632 [69,8]	965 [5,3]	18097	17608	102,8	1100 [7,3]	1365 [91,1]	238 [1,6]	14997	14438	103,9
19.11 2020	42	8,8 x 10^{14}	4500 [20,8]	15204 [70,4]	1881 [8,7]	21585	21462	100,6	1100 [5,2]	19473 [92,6]	465 [2,2]	21038	21053	99,9
29.11 2020	43	1,76 x 10^{15}	4500 [18]	16944 [67,7]	3578 [14,3]	25022	23883	104,8	1100 [4,1]	24736 [92,6]	885 [3,3]	26721	26028	102,7
09.12 2020	44	3,52 x 10^{15}	4500 [15,5]	17966 [62]	6521 [22,5]	28987	26096	111,1	1100 [3,5]	28602 [91,3]	1614 [5,2]	31316	28757	108,9
30.12 2020	46	1,408 x 10^{16}	4500 [11,2]	18818 [46,7]	17018 [42,2]	40336	35946	112,2	1100 [2,9]	32400 [85,9]	422 [15,2]	37727	33858	111,4
10.01 2021	47	2,816 x 10^{16}	4500 [9,6]	18968 [40,6]	23258 [49,8]	46726	45326	103,1	1100 [2,9]	33133 [82,8]	5788 [14,5]	40021	36609	109,3
20.01 2021	48	5,632 x 10^{16}	4500 [8,7]	19044 [36,6]	28478 [54,7]	52022	52349	99,4	1100 [2,6]	33512 [80,3]	7100 [17]	41712	38332	108,8

Table 11: *The comparison of my calculations of number of infected persons with the statistical records per one million habitants. In Poland and United Kingdom [But with separate three, not two, groups of people: I {A], II [B] and III {C] alias for „three waves of pandemic: early, main" and current one: called „from the strain or mutation born in South Africa"].*

Constants for calcularions:Poland – group A [I]: Imax= 1100 [0,11% of population]; Ka =5 x109

Group B [II]: Imax =33900 [3,39% of population]; Ka =6,52 x 1014

Group C [III]: Imax = 9180 [0,92% of population]; Ka =1,65 x 1016

United Kingdom [see figure 9]

Group A [I]: Imax = 4500 [0,45% of population]; Ka =1,26 x 109

GroupB [II]: Imax =19120 [1,91% of population]; Ka =2,26 x 1014

Group C [III]: Imax =36720 [3,67% of population]; Ka =1,63 x 1016

The number of viruses [in total cloud- per one million of habitants -ie supposedly from one „person zero"] was calculated according to equation: V = 200 x 2n [where n means number of succesive duplication period [t(2)] which length is 10,5 days].

So I am convinced that there are three groups of people [possible virus recipients] A, B and C. The most numerous [however not in China] group is usually group B with quite big level of the blocking sites [first of all serum ACE2]. The least numerous [but not in China, where it is the most numerous, if not the only one] is group A, with the level of blocking sites close to zero. The "last" group C which in United Kingdom is very numerous [even possibly the most numerous], almost like group B [„main wave"], but in Poland three times less abundant, is similar to the group A [I] with their deficit of the blocking sites [especially serum ACE2] in the blood.

Now two questions arise. First why I am inclined to think that the real reason of emerging of successive waves of pandemic lies in the presence of different subpopulations of susceptible people and not in the existence of different sets of viruses' mutations. And the second question-of great importance for practice, as it is with the infectivity, morbidity and mortality of these three waves, especially the latter [and is it the last?].

Before I answer, I must emphasize that „it takes two to tango", i.e. that infection, and thus the further course of events [disease, its exacerbation, possible immune defense, and finally death] requires close interaction between viruses and target cells, i.e. not only the structure of the virus, but also its functional (or not) receptors - perhaps

even more so than the viruses themselves. Meanwhile, a lot is written and talked about the viruses themselves, and less about their recipients. There is no indication that the type of virus mutation determines the maximum number of infected people - let alone the mutation has a decisive impact on I_{max}. The mutations have some obvious influence, but probably the most important are the kinetic constants of binding of the virus by receptors, so to put it simply: „type of people".

Imagine there was 1] only one- in the beginning of pandemic, say in January/February 2020- kind [genotype and phenotype] of viruses [or one type of virus recipients] 2] there was no blocking sites [for viruses] in the blood 3] everybody possessing membrane ACE2 was susceptible [I do not mean some minor group- apparently the youngest- with the exceptionally high immunity against SARSCoV-2, whatever nature]. So imagine exactly what all the people on the world [except me!] think and write [and additionally one should know that we all live in the cloud of human breaths and viruses and the infection is just matter of time].

Then one should assume I_{max} not less than 600000 per million [of all groups of age together] i.e. 60% of all people and K_a of range the same as I found [Eadie -Hofstee plot] for different countries [Tables 9-12] for"the first wave of pandemic" [March-August] i.e. 10^7 - 10^9 [mind you, it looks similar as for those calculated with the simple arithmetic "2 x 2….. x 2" model [Table 3 and table 5]. Then July the 8^{th} it would have been [calculated with an equation {3}] 6000 [per milion of habitants] with K_a equal 10^8, but 54545 with Ka equal 10^7. But on August the 13^{th} [2020] it would have been respectively 53300 [per milion] or…300000. Thus before October practically everybody would have ben infected. It is/was impossible! The first pandemic [never mind virus mutations] must concern just much much lower number of people ["in the ceiling"].

So I am sure that the reason of existence of second and third waves of pandemic is the existence of different groups/subpopulations of people and not the new mutation [including the last British-or South African-strain]. Of course i agree that the mutations took place and even had some influence on binding of receptor [especially if such a mutation itself might change very much possibility of binding the block sites].

As for the second question, the matter is very ambiguous. Apparently, it is enough for the cumulative infection cases' curves to „bend up" over time to give the impression that the infectivity has increased significantly. And if there was a coincidence, because it was shown in parallel - by studying the viral genome sequence - that there was a set of mutations supposedly affecting the binding of RBD of spikes with membrane ACE2 and its neighborhood!? Then we would be already convinced that the mutation increased infectivity.

The comparison of my kinetic coefficients with those from experiments of Chan., *et al* [76]. Comments the titer of kinetic coefficients. If the British strain is more infectious?-Some comments the titer of kinetic coefficients.

In fact such a conclusion would have to be confirmed by meticulous studies of the kinetics of the virus-receptor binding [and possibly virus-blocking sites either] and the subsequent stages of the entire infection and morbidity process [not only in culture cells from one man, but also in a large specific group of people]. There are hardly any such papers ever. However, I found a job that sheds some light on these issues. Chan., *et al*. quite recently [2020] engineered soluble decoy receptors, in which the ACE2 ectodomain is engineered to block RBD of spike proteins [S] of coronaviruses with high affinity, potently neutralize infection. They found an engineered decoy receptor, $sACE2_2$.v2.4, tightly binds S of SARS-associated viruses from humans and bats, despite the ACE2-binding surface being a region of high diversity. So called wild type ACE2 and the engineered decoy compete for binding sites. Variant N501Y in the RBD, which has emerged in a rapidly spreading lineage (B.1.1.7) in England, enhances affinity for wild type ACE2 20-fold but remains tightly bound to engineered sACE22.v2.4.

But in fact Chan., *et al*. [76] measured an affinity not of just real functional receptors of SARSCoV-2 but artificial proteins.What is more these proteins were [for analytical reasons and also for eventual possibility to apply them in future] bound to IgG antibodies [natural ACE2 were bound with the same IgG as well]. So in fact the measured affinity constants were even more similar to the constants for binding of block ie serum ACE2 and viruses than those of viruses with natural membrane functional receptors.

But certainly the measured data gives us an idea of even an order of magnitude of these constants for the V-RF bond. And now there is problem hardly ever solvable. In my interpretation K_a is presented as number of viruses [supposedly in the cloud i.e. the whole population] needed to contribute in an infection of 50% of all susceptible persons. Meanwhile K_a in experiments of Chan., *et al*. [76]-and similar ones-were expressed as nM [as K_a -see equation 12-and K-see equation 13 are in an analogy to applied by Chan., *et al*. [76] $K_D = k_{off}/ k_{on}$, where k_{off} {equivalent of k_2} is in s^{-1} and k_{on} {equivalent of k_1} is in $M^{-1} \times s^{-1}$].

But "suddenly" everything is at least "partially unlighted". One virus [more strictly just RNA from one SARSCoV-2] has a mass $1,635 \times 10^{-17}$g [one mol of viruses would weigh $9,81 \times 10^6$ g: 30000 nucleotides x 327]. Thus 10^8 viruses' RNA weigh 1,64 ng; 6×10^8 viruses represents 1 fmole of viral RNA, but 6×10^{14} represents 1 nmole of viral RNA. So my assumed K_a for group B ["main wave of pandemic"] is in range of 0, 1 - 1 nM [in terms of RNA content]near exactly those for binding of RBD of British strain of SARS CoV-2 with ACE-2 soluble decoy engineered receptor.

How it happen to be such a consistence and how theoretically explain a real chain of molecular events taking place from very first binding of RBD with membrane ACE2 to the infection first and … thousandth infected person…and their kinetics? And explain how from nM [of viral RNA?] we have got number of infected people?! It is the task for future scientists equipped with the mathematical machines.

Another puzzles are that 1] so called wild type ACE2 has K_D [equivalent to my K_a] almost 10 times bigger than the decoy and 2] what is with the real functional SARSCoV-2 receptors in membranes [I guess K_a for them are much lower than for soluble decoys].

But there is another doubt. Chan., *et al*. [76] [and generally Covid19 molecular virology] assume that the bigger infectivity of so called British strain [comparing to other strains] comes from the bigger affinity of binding ie smaller value of K_D [K_a]. But look on equation {12}. Real measure of infection/infectivity is the kinetic coefficient k_3 ie for the slowest ever molecular event in the infection chain [unfortunately nobody knows which one is the candidate]. And -in analogy with enzyme kinetics in which:

$V_{max} = k_3 \times S$……..{14}

Where V_{max} means maximal velocity and S means concentration of substrate, we might propose:

$I_{max} = = k_3 \times V_{max}$……..{15} [where- bit misleading -V -as before in my hands-means number of viruses [analogous with substrates of reaction-compare earlier text]-but V_{max} means the maximal possible amount of viruses bound to all functional receptors [the value I do not know, who does].

And now $K_a = k_3 + k_2/ k_1$……..{12} and Chan's $K_D = k_2/ k_1$. It means that Chan and other scientists claiming about infectivity of….British strain of virus did not consider real infectivity and infection [as they did not take into consideration k_3]; just they estimate the binding [k_1]. So it looks like British strain indeed binds easier [in my interpretation: certain group of people easier bind viruses] but not necessarily there is a greater infectivity i.e. the number of infected people [with about the same amount of viruses "around"] does not necessarily increase over time.

And now let us assume different values of kinetic constants and compare them with those estimated experimentally by Chan., *et al*. [76] but also with K_a in terms of number of viruses needed to attain $I=I_{max}$]. All such data are shown in the table 12.

Group [subpopulation] or * experiment [ ]: RBD of SARSCoV-2 strain.../ s ACE2 from.....		k_1 [k_{on} *] [$M^{-1} \times s^{-1}$]	k_2 [k_{off} *] [s^{-1}] [M]	$K_a = k_2 + k_3 / k_1$		k_3 [s^{-1}] @	I_{max} = the ceiling ie the maximal number of infected people[per 1 million] to infect I_{max} &	Calculated amount of viruses V_{max} [on average]	
				Number of viruses in the cloud				To infect one person &&	
A		1×10^{13}	$1,67 \times 10^{-2}$	$1,67 \times 10^{-15}$	1×10^{9}	$1,67 \times 10^{-4}$	4500	$2,69 \times 10^{7}$	5970
B		3×10^{9}	5×10^{-2}	$1,67 \times 10^{-11}$	1×10^{13}	1×10^{-5}	19120	$1,91 \times 10^{9}$	100000
C		1×10^{8}	$1,67 \times 10^{-1}$	$1,67 \times 10^{-9}$	1×10^{15}	1×10^{-4}	36720	$3,67 \times 10^{8}$	10000
-----	* Y449K/ wild type	2×10^{6}	9×10^{-2}	$ $4,5 \times 10^{-8}$	# $2,71 \times 10^{16}$	----------	----------	---------	--------
-----	* N 501Y/ wild type	$2,2 \times 10^{6}$	$1,8 \times 10^{-3}$	$ $8,2 \times 10^{-10}$	# $4,94 \times 10^{14}$	----------	----------	----------	----------
-----	* N 501 Y/ engineered decoy	$3,6 \times 10^{5}$	$1,1 \times 10^{-4}$	$ 3×10^{-10}	# $1,81 \times 10^{14}$	----------	----------	----------	----------

Table 12: *The kinetic constants of binding of SARSCoV-2 with receptors in thrre subpopulations of susceptible people A, „first wave", B "second wave" and C "third wave of pandemic" in United Kingdom [compare table 11]- my aasumptions versus some Chan [] * data.*

@ I adopted the titre of k3 in s-1 ie exactly as for k2 ;but I am not able to explain that in terms of molecular events; also the values of k3 are chosen arbitrarily just to obey two regularities:

1. That always k3 is the least in the set of constants : k1, k2 and k3.

2. To show that real infectivity [measured with k3 value] is the biggest for people of group A, much smaller for group B and for group C [supposedly as for binding of British strain B.1.17] is slightly less than for subpopulation A [most affected between March and August] but still much greater than that for the subpopulation B [mostly affected between September and December].

& The equation for Imax is : Imax = = k3 x Vmax…….{15} [Vmax means the maximal possible amount of viruses bound to all functional receptors [the value I am not able to explain, but can easily calculate from eq.{15}.

*&& Then by dividing Imax by such calculated Vmax I obtained something like k3 but in terms of number of viruses needed to infect one person from given subpopulation. These data →apparently show that the true infectivity of these three population groups is increasing according to direction shown with arrows: B→ C→ A. $ * these are the values of KD from the paper of Chan., et al. [ ]*; it corresponds my Ka.*

 # KD was presented there in terms of number of viruses [to make possible the comparison of my calculated data with those of Chan., et al. [ ] -as 1 mole contains 6,02 x 1023 viruses, so [M] x 6,02 x 1023 gives us number of viruses.

It is visible that: 1/ both changes in k_1 and k_2 contribute into the final values of K_D [K_a] 2/ this concerns even more to serum ACE2 decoy binding, as for supposedly much infectious British strain [withN501 Y RBD] the proper binding is even smaller than for RBD of wild type of supposedly less invasive viruses as k_1 value is smaller but the change in k_2 value decides on final value of K_D 3/the values of K_D of Chan., *et al*. [76] for binding viral RBD with ACE2 are quite similar [in range of magnitude] to my assumed [and estimated-compare table 9-11] values for groups/subpopulations B [main group in Poland/main wave everywhere] but hardly A [first affected-in February/March 2020]; but it seems that there are quite possible such bigger values for k_1. Anyway the values of k_3 not included [not even mentioned] by Chan., *et al*. [76] and all the molecular virology-are decisive.

But it is quite possible [as „it takes two to tango"], that the type of group -given person belongs to decides to the great extent-with which viruses the infection is possible. I mean that it might be that for instance people from group C are infected first of all with "British strain" of viruses [B.1.1.7 with N501Y "main" mutation] much easier than with other strains [supposedly older ones-but in fact it is not the time of appearing of this mutation deciding but mutual matching of virus RBD and that of the entirety of functional receptors]; it might be that -for instance- this mutation [i.e. replacement of N with Y i.e. asparagine for tyrosine] had happened long time ago but simply there were not people with the receptors with a certain affinity just to such viruses. Or they even were in the neighborhood but the all characteristics of kinetic constants [i.e. the size of k_1, k_2 and k_3] decided that first mostly an infection of people from another group [i.e. A or B not C] took place.

General problems

And now only last two questions: 1/ would be this third i.e. actual wave the last one or there would be fourth, fifth and so on? And 2/ what is real numbers of infected persons as well that of certainly sick, seriously sick and -most important-recovered, who certainly produced enough specific antibodies to contribute into the complete destruction of viruses in their bodies.

As for the first question: The current „third wave" [in my opinion, simply an evident increase in infections of susceptible people primarily of special type C-compare the previous text] is in my opinion the „end" of the pandemic. Why? Simply viruses [multiplying somewhere] have no more binding sites almost anywhere - soon there will be no possibility of further multiplication and even re-infection. Of course, they may still „slowly come to light" delayed effects of previous infections, before even several months, especially extra-pulmonary, especially intestinal and in the nervous system. These can also be diseases related to indirect or long-term effects, for example, of a cytokine storm, and not only directly the „virus attack" on target cells. Certainly, there will be a „delayed wave of deaths" and an increase in the incidence of sickness within younger people, including children under 10 years of age.

As for the second question: I would like to emphasize that in general, the current increase in the number of infected, i.e. the daily increases of reported cases of new infections, so the „third wave", can be [and has been for a long time, e.g. from November 2020] „a flood of artifacts".

I mean, I do not deny that, for example, on January 25, 2021, Ms. XYZ's test result turned out to be positive [and the lady had not been tested before and did not complain in principle earlier]. This Lady could just get infected [that is, download a minimum dose of 200 viruses] e.g. on November 2, 2020, and now she has got [?] about 50, 000 viruses and she started to feel clearly sick. Or she got infected just 2020 December 13th [e.g. from a person almost dying from Covid19] with a large dose, say 6, 400, not just 200 viruses. Another possibility is that this infection [01/21/2021] was the second infection, and the first happened a month ago, but it is doubtful that - even if she had done the test earlier - it turned out to be positive [she still had too few viruses, so that even a super-sensitive test, let alone antigenic one, showed their presence].

Generally the best arguments that statistical records, namely the reported number of cumulative [and daily] cases of infection, are too low [not only in Poland -all over the world, including countries I presented -tables 9-11] are the following [compare an earlier text]: 1/ the tests are not made randomly 2/ the tests are rather for people even mildly sick than those absolutely asymptomatic ones 3/ thus values of

main constants [I_{max} and K_a]-revealed by Eadie-Hofstee plot- are too low thus making that first victims [from any group of people] of Covid19 came clearly too late eg not in February but in April, not in August but in October and so on.

So one should just to multiply I_{max} [estimated with Eadie-Hofstee-EH- linearizing plot] 3 - 4 times thus estimating the an final number [ceiling]. If we did the same with the actual statistical records of cumulative cases of infected people then we would obtain the real data which clearly would show that in fact practically all susceptible people were already infected [well with so called "third wave"- „with a British strain"- the daily increases from December the 1st to say today ie. February the 9th are of course unfortunately real.

However there remains how to estimate number of sick S, clearly sick S, seriously sick S_{ser}, recovered and dead. So one should then first assume K_a for an infection as 10 - 40 times lower than that estimated with above [EH] method.

The estimation of pandemic parameters for different groups of people [recipients]-my method;with use of statistical records

But how estimate more accurately a number of affected eg sick people? I mean: without the direct assumption some in fact arbitrary values of $K_{a.}$

I will show such a method with the data presented in table 13.

Time n	Date	Total number of viruses	Number of persons affected [per 1 million of habitants]						
			@ Infected I *1/ *2/	Sick S *1 *2/	Producing antibodies A	Clearly sick S *1/ *2/	Seriously sick S_{ser} /*1 *2/	Recovered REC *3/	Dead D *4/
20	01.04 2020	2,097 x 10^8	44 [44]	0	0	0	0	0	0
22	22.04 2020	8,389 x 10^8	158 [158]	1 [1]	0	0	0	0	0
24	13.05 2020	3,335 x 10^9	440 [440]	4 [4]	2	0	0	0	0
26	03.06 2020	1,342 x 10^{10}	802 [802]	15 [15]	8	3 [3]	0	0	0
28	24.06 2020	5,359 x 10^{10}	1007 [1005]	54 [52]	28	10 [8]	2 [0]	2	0
30	15.07 2020	2,147 x 10^{11}	1075 [1071]	204 [200]	100	33 [29]	6 [2]	4	0
32	05.08 2020	8,59 x 10^{11}	1093 [1079]	437 [423]	324	115 [101]	20 [6]	14	0

34	26.08 2020	3,436 x 10^{12}	1099 [1050] /1047	550 [501] /498	540	339 [290] / 287	68 [19] /16	49	3
36	16.09 2020	1,874 x 10^{13}	1100 [930] /919	589 [419] /408	692	589 [419] /408	229 [59] /48	170	11
38	07.10 2020	5,498 x 10^{13}	1100 [683] /643 &	598 [181] /141	776	598 [181]//141	479 [62] /22	417	40
40	28.10 2020	2,199 x 10^{14}	1100 [376] /238	600 [<0] <0	794	600 [<0] /<0	595 [<0] / <0	724	138
42	19.11 2020	8,8 x 10^{14}	1100 [324] /90	600 [<0] <0	796	600[<0] <0	600 [<0] <0	776	234
44	10.12 2020	3,52 x 10^{15}	1100 [312] /60	600 [<0] <0	799	600 [<0] <0	600 [<0] <0	788	252
46	31.12 2020	1,408 x 10^{16}	1100 [301] /3	600 [<0] <0	799	600 [<0] <0	600 [<0] <0	799	298

Table 13: *Parameters of pandemic -for group a of population in poland [calculated on the basis of statistical records of infected people]- so called "first wave of pandemic". The number of n, so date, and total number of viruses in the total cloud [population] per 1 million of habitants shown is exactly the same as in the table 10. *1/: in parentheses [ ] there are numbers of affected but minus those people REC who apparently recovered as they were producing the amount of specific IgG antibodies. anti SARSCoV-2 being enough to cause the complete destruction of viruses [the number is shown at *3/]. *2/: after / : previous numbers [shown at *1/] but minus the number of dead people[shown under *4/]; so as there are substracted numbers of both recovered and dead people,these values could correspond the number of so called "active cases".*

@ These numbers are taken from table 9 [for Poland] for group I [anotherwords A] ie something like so called "first wave of pandemic".

Constants for calcularions: Poland – group I : Imax= 1100 ; Ka =5 x109.

Remaining explanations –crucial for the calculation of data shown in table 13 –see text.

Now I am going to show how to calculate the number of affected people [of any kind]- ie infected, infectious, sick, clearly sick, seriously sick, dead but also producing specific antibodies A, producing much more of them A, and at least really recovered REC ie certainly producing the amount of antibodies enough to completely destroy all viruses [provided that first they did not die]- for group I [A] in Poland: The number/ s of infected people [for given group /subpopulation of people -estimated as I explained earlier -see table 9-11] on a logarithmic scale [decimal logarithms] - were plotted on the graph [on y-axis; and x-axis is time, i.e. the number of periods of doubling -of viruses - amount -n]. I previously assumed that an infected person has-shortly after infection- a minimal dose of 200 viruses in the body.

After n = 1 [i.e. after 10.5 days] it has 200 x 2 = 400 viruses, etc. In turn, I previously assumed [compare earlier] that in order for an infected person to be infectious [Inf], he must have more than 4000 viruses [V], to be sick [S]: > 40,000 V; to produce antibodies [A]: > 10000V; to be infectious with a high degree of certainty [Inf]: > 32000 V; to produce clearly more antibodies [A]: > 80000V; that she was clearly sick [S]: > 300000V; that she would be seriously ill S_{ser} [risk of death]: > 2 x 10^6 V; that she would - most certainly - recover [REC] [i.e. produced antibodies to kill 100% of viruses- > 3 x 10^6 viruses {of course, since it kills viruses, there will be zero of them ;at least 3 million of them if the person for some reason did not produce antibodies at all}; that they would die [D]: > 5 - 6 million V.

Such amounts of viruses will be- on average- after the time given below [Δn] from the moment of infection ;in parentheses there is shown the time required for this ["kind of affect"], but assuming that there a block takes place ie considerable number of „blocking sites" in the blood of an infected person exists - compare the previous text]: Inf: 4.32 [8.05]; A: 5.64 [9.37]; Inf: 7.32 [11.1]; S: 7.64 [11.2]; A: 8.64 [11.7]; S: 10.55 [12.55]; S_{ser}: 13.29 [13.87] REC: 13.87 [14.24]; D: 18.2 [18.4].

The above numbers come from an equation: V = 200 x 2^n. So [for Inf] 4000 = 200 x $2^{n,}$, thus n = 4,32; for REC: 3 x 10^6 = 200 x $2^{n,}$, thus n = 13,87 and etc. The values in parentheses [for persons with an apparent block of viruses in blood] come essentially from table 6; more strictly as above but from equation: V_{free} = 200 x 2^n x 0,0758.

[As 1,00 - 0,0758 = 0,9242 ie 92,42% of viruses is -on average-blocked in the blood;this is visible clearly for very first time of infection but of course for just one "person zero" [n < 12, table 6].

!! And now we shift the straight line- [„curve"] of the decimal logarithm of the number of infected persons - to the right by the above Δn value; then we read [y-axis] the corresponding values of lg S, lg A, lg REC etc, and from them we calculate the values of S, A, REC, etc.

Of course, first we have to assume the maximum values of X_{max}. I_{max} is 1100 [See table 9-group I for Poland]-ie about 54,5% people of group I [A]; for S [as well as S and S_{ser}] I assumed 600 [i.e. about 54,5% people of group I [A]]; and for A [also A and REC] 800 [ie about 72,7% people of group I [A]] and for D 300-ie about 27,2% people of group I [A]] The latter is the least certain, but as it turns out later it has some justifications]. Later on [Table 14] I adopted for groups A and C-those apparently without a block: S_{max} as 60% of I_{max}, and A max [so REC_{max}] as 80% of I_{max}.

Evidently from about October the 7th all [or the majority of them] of these infected.

People [rather "active cases"] are clearly or even seriously sick. You need to „know how to read" the data in table 13. With corrections for the number of recovered people and the dead, we have on December 31, 2020 only [we are talking about cumulative data] people who died [300 out of 1100 in this group] or convalescents [respectively 800] - but we have practically no sick [they all recovered or died]. However, on October 7, we have analogically only 40 dead and only 417 convalescents [out of 1100], and out of 643 „active cases" as many as 141 are ill - clearly, not slightly - almost asymptomatic [S and not S], and of them as many as 22 are in severe-threatening condition [S_{ser}].

After such a calculation one should multiply obtained data by 3,5 [2,5-5?]or this multiplication might be made first [to obtain "more realistic" I_{max}, thus I. The same such [a bit laborious] way has to be done thereafter with remaining two groups of people [eventual recipients

of virus] i.e. B and C; the summing would give us the total picture of pandemic. Later on [Table 14] I did such calculations for groups A and C, but for main one [at least in Poland] group B ["second wave ie roughly from September to December"] I simply used my calculated data I had presented in table 7 [per 1 million of habitant]. I mean the number of infected people I but of course further calculation with Δn method were made exactly as I explained before [and as I did for groups A and C]. If one would like to calculate this for three mentioned groups of people for any country/area [Portugal, Russia, Iowa State, etc] one should follow the succesive steps of my method [as for table 9-11 and 13].

By the way data in the table 13 clearly show that by the end of 2020 year people from group A [I] are either dead or certainly recovered. So is rather excluded they might be victims of the third wave unless, after all, they got infected a second time [??].

The applied mode of estimation of number of affected people presented here is based upon "rule of analogy" and data coming from the tables 3, 5 and 6.

Summarizing table for Poland and final conclusions

And now the final table 14 presenting briefly all the pandemic from the beginning of 2020 upto April 2021-but only in chosen dates.

Date	Group A				Group B				Group C				All the population = A + B + C			
	I /AC	S /&	REC	Died D	I [AC]	S &	REC	Died D	I [AC]	S &	REC	Died D	I [AC]	S &	REC	Died D
28.01	74/ 74	0	0	0	0	0	0	0	0	0	0	0	74/ 74	0	0	0
18.02	276/ 276	0	0	0	0	0	0	0	0	0	0	0	276/ 276	0	0	0
11.03.2021	920/ 920	0	0	0	0	0	0	0	0	0		0	920/ 920	0	0	0
01.04	2143/ 2143	6	0	0	6	0	0	0	0	0	0	0	2149/ 2149	6	0	0
22.04.2021	3211/ 3211	17	0	0	25	0	0	0	0	0	0	0	3236/ 3236	17	0	0
03.06	3803/ 3772	195/ 164	29	2	407/ 407	0	0	0	0,78=1	0	0	0	4211/ 4180	195	29	2
15.07	3847/ 3541	1698/ 1392	288	18	6918/ 6918	0	0	0	12,54=13	0	0	0	10778/ 10472	1698/ 1392	288	18
26.08	3850/ 1607	2239/ 0	1995	248	54954/ 54949	17/ 12	5	0	201/ 201	0	0	0	59005/ 56757	2256/ 8	2000	248
16.09	3850/ 736	2291/ <0	2630	484	100000/ 99976	66/ 42	22	2	783/ 783	1	0	0	104633/ 101495	2358/ <0	2652	486

07.10	3850/ 0	2312/<0	3020	891	123027/ 122920	257/ 153	98	6	2920/ 2920	4	0	0	129797/ 125840	2573 <0	3118	897
28.10	3850/ 0	2312/<0	3083	962	131826/ 131399	1000/ 573	407	20	9177/ 9176	13/ 12	1	0	144853/ 140575	3325 <0	3491	982
19.11	3850/ 0	2312/<0	3083	962	134896/ 133039	3981/ 2124	1778	79	19772/ 19767	42/ 37	5	0	158518/ 152806	6336/ 429	4866	1041
10.12	3850/ 0	2312/<0	3083	962	138038/ 129967	13804/ 5733	7762	309	27788/ 27768	155/ 135	19	1	169676/ 157735	16271/ 4135	10864	1272
31.12	3850/ 0	2312 /<0	3083	962	138028/ 106449	23442/ <0	30903	676	30922/ 30855	575/ 508	63	4	172800/ 137304	26329/ <0	34049	1642
20.01 2021	3850/0	2312 /<0	3083	962	138028/ 76795	37154/ <0	60256	977	31819/ 31570	2188/ 1939	234	15	173697/ 108365	41654 /<0	63573	1954
09.02 2021	3850/ 0	2312 <0	3083	962	138028/ 59403	44668/ <0	77625	1000	32052/ 31111	7244/ 6303	891	50	173930/ 90520	54224/ <0	81599	2012
2.03. 2021	3850/ 0	2312 <0	3083	962	138028/ 49932	52480/ <0	87096	1000	32110/ 28762	13490/ 10142	3162	186	173988/ 78694	68282/<0	93341	2148
23.03 2021	3850/ 0	2312	3083	962	138028/ 47903	53211 /<0	89125	1000	32130/ 21919	17378/ 7156	9550	661	174008/ 69822	72901/ <0	101758	2623
13.04.2021	3850/ 0	2312	3083	962	138028/ 47903	53211/ <0	89125	1000	32130/ 14042	19055/ 967	15849	2239	174008/ 61945	74578/ <0	108057	4201

Table 14: *Final. The number of affected people of three different subpopulations/groups [A, B, C] during the pandemic of COVID19 in poland -2020/21. Per 1 million of habitants-calculated with me -exactly as in tables 11 and 13-explanations in text.*

The calculations followed the above method. Only some parameters were taken into consideration: number of infected [I], number of clearly sick S, number of recovered REC and number of dead D-but this allows us calculation of AC [active cases] = I-REC -D, as well as correction of S number considering number of REC and dead D.

[S_{corr} = S-REC -D]. In the table 14 I omitted for bigger clarity remaining parameters [as Inf, Inf, A, A, S, S_{ser}]. The number of clearly sick S-supposedly for people with > 300000 viruses in the body seems to be quite important -much more than just number S [> 40000 V] which by its nature contains all people with so mild sickness level that they are almost asymptomatic. Of course I can't judge if they have to be hospitalized as certainly have so called seriously ill S_{ser} [> 2 x 10^6 V].

As REC I assumed [and so calculated as I explained] the people with the number of viruses in the body > 3 x 10^6. So it might mean there is a "crossroad" -about 2 million viruses in the body. Either the person's illness will get much worse - he will become seriously ill - at risk of death - or he will produce enough of specific antibodies that the viruses present in the body will die completely. Either recovery or death. Of course, given this - to write that „a person being completely and certainly recovered contains 3 million viruses in the body [or even > 3 x 106] is an" internal contradiction „.

The most important observations and conclusions can be found in the summary table 14:

- Until mid-April, and practically even until June 2020, the pandemic was almost asymptomatic [of course, compared to what followed later.
- Until June, infections and disease of people from group A predominate, but since July already group B. Group C [„third wave"] dominates over group B from the end of October, but only when we take into account the increase in the number of infections [daily, weekly], not cumulative cases.
- The maximum number of active cases took place around mid-December 2020; most of the active cases are then people from group B, but almost 20% of them come already from group C.
- The number of active cases after exceeding a certain maximum decreases; for group A [but the same tendency is observed for group C] - i.e. people without blocking sites - it drops to zero; these people - especially from group A „either recover or die".
- However, for people from group B [the most numerous - in Poland, but also in many countries of the world], having an effective virus blocking system, the number of active cases decreases, but at the end of the pandemic there are still a lot of them [about 1/3]; by the end of the pandemic, about 1/3 remain either completely or nearly asymptomatic but without immunity.
- It seems that the mortality [in %] is higher for people without viral block, i.e. for people from groups A and C.
- Since mid-December the number of convalescents has sharply increased; later this increase is slower, but clear.
- At the end of the pandemic, there are practically no people who are clearly ill [that is, apparently they are, but they are - statistically speaking - single cases]; but here is repeated what was already observed from the end of August to mid-November - then the number of clearly ill people dropped to zero [mathematically to < 0, as simply the number of people recovered - mainly from group A - exceeded the number - calculated - clearly ill and there were still few clearly sick people from group B, let alone C].
- The dynamics [shape of cumulative data curves] is completely different for infected, clearly sick, convalescents, deceased and active cases.

The best confirmation of my calculations, assumptions and reasoning comes unexpectedly…within data from media. Look, it has been reported that just about 2% of teachers tested recently in Poland [about 03.02.2021] has positive result. If 50% of them is 4 - 65 years old it gives us 1/6 x 500= 83 of susceptible [according my assumptions, table 1]; remaining 50% [25 - 45 years old] gives us 1/12 x 500 = 41, so together 121 [per 1000] susceptible [having the functional receptors], so according my assumptions: 118 - 121 already infected [cumulative data]. So if we substract about 80% recovered [even unconsciously, so 95-it gives us finally not more than 24 active cases, thus < 2,4% of positive tests.

So it remains to have confidence in the collective wisdom of all mankind and in Science, which will offer us effective vaccines and medicines, and most importantly, more complete knowledge, not limited to fashionable supermethods.

As for the SARSCoV-2 mutant attack: you just have to judge and act as I wrote above. The same is true of other deadly viruses -let's hope not coming from our own genome and still present in it.

Conclusion

Generally, my calculations-based upon the asumed number of viruses within the cloud [of breaths and viruses] ie all the population fit after some interpretation-to the statistical records.

In Poland, Belgium, Netherlands, Hungary, Portugal and United Kingdom [but similar situation is in the majority of most seriously affected countries all over the world] it looks as there were/are two waves of pandemic: earlier [March - August 2020] and main current one. But [!] about November there begun the "third wave". But in fact there is one pandemic but with three groups of people infected: a/ very small A without the block [or with much lower level of block] taking place with an infection quicker [small K_a] and b/ much more abundant ["main", at least in Poland] group B with an infection going slowly [much bigger K_a] for people apparently having quite significant level of block [in their blood] and c/ group C -without a block operating [similar to A]. In United Kingdom [most probably in Portugal either] group C is the most numerous, also the abundance of group A is bigger than in Poland.

The alternative interpretation of the "second wave"-the main one or the current one- in my opinion much less probable- is that about May there was "big mutation" [I do not mean quite a lot of "small" mutations taking place all the time from the very beginning of pandemic] substantially increasing binding of SARSCoV-2 with the blocking sites in the blood. Eventually "the new strain of virus" supposedly discovered in the December 2020 in Great Brittain might be just the "old version"of virus [from about March] hardly binding with the block sites.

Probably the survival of a block-i.e. "alleviated pandemic" [quoting is because usually word "pandemic" concerns all the population not the single person] - does not result to develop immunity to reinfection, or such an immunity concerns only for small fraction of susceptible infected people.What is worse, it seems that even when the block is already saturated also does not induce immunity in all infected people.

The pandemic is clearly coming to an end [December 2020 and once again February 2021] due to „viruses having difficulty meeting free functional receptors".

Bibliography

77. Atkins GL and Nimmo IA. "A comparison of seven methods for fitting the Michaelis–Menten equation". *Biochemical Journal* 149.3 (1975): 775-777.

78. Chan KK., *et al*. "An engineered decoy receptor for SARS-CoV-2 broadly binds protein S sequence variants. Preprint". *Bio Rxiv* (2020).

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