# In May 2023 the WHO stated that the acute phase of the Covid-19 pandemic had ended. But when and how will the chronic phase of this pandemic end? Are we perhaps confusing chronicity with endemicity?

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Many are likely wondering by now whether my theory regarding the further evolution of the virus and the escalation toward a tsunami of C-19 hospitalizations and deaths will ever come to pass and whether such a frightening outcome is indeed the only scientifically plausible scenario for the chronic phase of this pandemic to end. I have asked myself that question hundreds of times. Essentially, people are wondering whether there might be another way to bring this SARS-CoV-2 (SC-2) pandemic to an end. That this pandemic is still very much ongoing is beyond the slightest doubt. The virus continues to systematically produce new variants, even though for quite some time now, these have taken the form of quasispecies[1]. The viral transmission rate is

still relatively high, especially because of the high intrinsic infectiousness of the circulating variants and since infections by the currently circulating variants are often *mild* or *asymptomatic*.

Even though symptomatic infections now frequently have a milder or more chronic course and viral concentrations in wastewater remain relatively low, it is crystal clear that the currently circulating SC-2 variants still cause C-19 illness, hospitalizations and even deaths. The interpretation by our health authorities and, unfortunately, also by many scientists and experts that this pandemic is gradually fading out to transition into a seasonal infection—like the flu—is, therefore, pure nonsense. It has long been evident that populations in highly C-19 vaccinated regions are unable to develop sterilizing herd immunity. In other words, *the SC-2 pandemic is far from over*; at most, it has taken on a different course (i.e., a more chronic progression) and has meanwhile caused many animal species to now also serve as reservoirs for the virus.

In my view, the relatively 'milder' course, as evidenced by the declining number of acute reinfections, can be attributed to a buffering effect exerted by migratory dendritic cells (DCs) patrolling the upper respiratory tract. Their lectin receptors strongly interact with viral sugars on the surface of the highly infectious circulating variants (see fig. 1 appended below). Upon exposure, an increasing number of these highly infectious variants are 'parked' on these DCs instead of being internalized into susceptible epithelial cells. This likely explains the decline in the rate of productive viral

infection. *This is the main reason why both laypeople and scientists are under the mistaken impression that the pandemic is waning and will soon transition into a seasonal epidemic that—according to our poorly educated health authorities—could be managed in a flu-like manner through annual vaccination of the most vulnerable in the population*! However, those among us who made just a bit more effort to understand these complex biological phenomena realize that suboptimal, vaccine-induced immune pressure on actively circulating respiratory viruses that easily mutate and/or tend to recombine in animal reservoirs not only promotes viral immune escape but may even be dangerous when the neutralizing capacity of vaccine-induced antibodies (Abs) against new variants decreases significantly. This is because such Abs markedly increase the risk of Ab-dependent enhancement (ADE) of infection. Both suboptimal virus neutralization and ADE of infection promote the further spread of the virus, respectively by facilitating natural selection of viral immune escape variants and 'enhancing' viral infectiousness.

The evolutionary dynamics of the C-19 pandemic now increasingly rely on viral replication and shedding in individuals with milder but prolonged symptomatic infections (commonly referred to as 'long Covid'), the vast majority of whom are C-19 vaccinees. This begs the question: *If vaccination is far from an effective method to curb the spread of currently circulating SC-2 variants, what alternative options do we have to contain this ongoing C-19 pandemic?*

Given the current immune-epidemiological situation in highly C-19 vaccinated countries, I can only think of two scenarios that could contribute to ending the pandemic -both, however, by triggering a hyperacute tsunami of C-19 hospitalizations and deaths, as I have been predicting.

I recently discussed one of these scenarios in a previous contribution that is available on the VSS website (https://www.voiceforscienceandsolidarity.org/scientific-blog/large-scale-flu-vaccination-could-facilitate-or-expedite-a-tsunami-of-case-fatalities). It involves the *reverse zoonosis of SC-2* in highly vaccinated C-19 populations, initially leading to a higher prevalence of mild to asymptomatic infections by *avian influenza viruses* in birds. The fulminant expansion of highly pathogenic avian influenza virus (HPAI) involving multiple genetic lineages (primarily belonging to the H5N1 subtype) that have evolved through genetic drift and reassortment, is clearly increasing the likelihood that a strain could emerge that is well adapted to humans and capable of enabling human-to-human transmission upon zoonotic spill-over to humans. Airborne avian influenza virus spread by birds could *theoretically* trigger rapid global panzootics in several mammalian species or even a global pandemic in humans, causing high morbidity and mortality rates, especially in immunologically naïve populations. While this possibility cannot be fully ruled out, I don't believe HPAI will sufficiently adapt to mammalian populations that have a high prevalence of species-specific anti-influenza Abs. Indeed, it is reasonable to assume that the better newly emerging avian flu strains bind to cell-surface receptors of susceptible mammalian

cells, the more likely they are to be recognized by pre-existing infection- or vaccine-induced Abs against species-specific seasonal flu viruses. For example, it is estimated that about 50–60% of the human population in the United States has such Abs. However, this recognition is *non-functional* (i.e., *suboptimal)*, because these Abs only *cross-react* with, but do *not cross-neutralize*, avian flu strains due to some antigenic similarity between certain epitopes on the hemagglutinin (HA) or neuraminidase (NA) proteins. Such cross-reactivity may lead to ADE of *disease* (ADED) in populations with high levels of anti-flu Abs.

Given the current surge in seasonal influenza cases—especially in C-19 vaccinated individuals—and the recommendations to vaccinate against seasonal influenza, the proportion of the population with high anti-flu Ab titers is now certainly increasing in many highly C-19 vaccinated countries. *It is therefore likely that, instead of witnessing a truly global avian flu pandemic, we will see an increasing number of individual case fatalities due to ADED in highly C-19 vaccinated populations*. This will not only affect individuals who developed high titers of infection-induced anti-flu Abs after suffering from serious breakthrough infections with seasonal flu, but also those with high vaccine-induced anti-flu Ab titers, regardless of their C-19 vaccination status. For this reason, *I strongly advise against vaccination against the seasonal influenza virus, even in the context of current multi-country flu surges*. On the other hand, individuals with weak innate immunity—such as those with underlying diseases or those whose cell-mediated innate immunity (CMII) was not adequately trained during the earlier phases

of the pandemic (when their C-19 vaccination prevented widespread acute SC-2 infections from training their CMII)—could also develop high anti-flu antibody titers. These individuals might consider taking antiviral medications at the early onset of symptoms.

**However, the current surge in viral respiratory infections unrelated to SC-2 (e.g., seasonal flu, Respiratory Syncytial Virus [RSV], and Human Metapneumovirus [hMPV]) in highly C-19 vaccinated populations is much more likely to trigger the end of the C-19 pandemic**.

Because of the redirection of cellular immunity[2] towards dendritic cell (DC)-mediated inhibition of virulence (see Fig. 2 below), the acute phase of C-19 disease increasingly transitioned into a more chronic form ('long Covid'). This transition induces a shift in population-level immune pressure—from targeting *viral infectiousness* to targeting *viral trans infection* and *trans fusion*. As the prevalence of chronic infections is now increasing, the occurrence of cryptic variants[3] capable of *intra-host* transmission[4] and shed by chronically infected individuals also rises. Over time, this gradually increases the likelihood of a 'suitable[5] cryptic variant emerging—one capable of intra-host transmission while overcoming the suboptimal immune pressure on viral *trans* infection and *trans* fusion (i.e., viral virulence) exerted by highly C-19-vaccinated populations exposed to co-dominantly circulating variants.

As previously explained, circulating, highly infectious SC-2 variants are increasingly being adsorbed onto migratory DCs patrolling the upper respiratory tract (URT; see Fig. 1 below, including a relevant reference from the literature). This not only dampens the rate of productive viral infection but also prevents antiviral immunity from being stimulated, as DCs cannot serve as antigen-presenting cells (APCs) unless the virus or antigen is internalized into these cells—rather than merely adsorbed onto them. As this diminishes viral production and shedding while failing to abrogate transmission due to the lack of sterilizing immunity, *the evolutionary dynamics of this pandemic seem delayed*. At this 'metastable' stage of the pandemic, viral *inter-host* transmission largely relies on weak but prolonged viral shedding by an increasing number of repetitively or 'chronically' SC-2-infected individuals. As this metastable equilibrium[6] creates a sort of steady-state situation, still enabling sufficient viral *inter-host* transmission for the virus to survive, viral *intra-host* transmission is not yet under sufficient cellular immune pressure for a new viral phenotype to be selected that can overcome the virulence-inhibiting effect of viral attachment to URT-patrolling DCs.

I have been wondering whether and how this metastable equilibrium could *suddenly* transition to a more stable, lower-energy state. In other words, the question remains as to when the pressure currently exerted on viral *trans* infection and *trans* fusion will become sufficiently high to trigger such a spectacular immune selection event. Departing from the metastable equilibrium currently evidenced by relatively low SC-2 wastewater levels, along with relatively low C-19 hospitalization and mortality rates,

*some external force would likely be required* to increase collective immune pressure on viral *trans* infection while promoting the emergence of new variants with enhanced *intra-host* transmissibility. Such a force could destabilize the current balance, leading to a more stable state in which viral propagation in highly C-19 vaccinated populations is no longer mitigated trained by population-level immunity.

*Outbreaks of other viral respiratory diseases, which are currently surging in several highly C-19 vaccinated countries, are likely to expedite the emergence of a new coronavirus (CoV) lineage that may prove highly virulent in highly C-19 vaccinated populations and could thereby provide enough energy for the metastable state of this pandemic to transition into a stable, low-energy state.*

*Why is this a scientifically plausible hypothesis, and what is the mechanism by which these viral respiratory epidemics could soon lead to the end of the C-19 pandemic?*

During the acute phase of the pandemic, the overwhelming prevalence of acute SC-2 infections inhibited training of CMII in C-19 vaccinees, as well as in unvaccinated individuals who suffered severe C-19 disease. Deficient or insufficient training of CMII in these individuals has now triggered outbreaks of other respiratory viruses (e.g., seasonal flu, RSV, hMPV), which were previously largely outcompeted by SC-2. Symptomatic infection by these viruses not only contributes to training the CMII system of these individuals but also activates broadly functional cytotoxic T lymphocytes (CTLs). These CTLs not only contribute to abrogating viral infection and

curb the spread of these other respiratory viruses but also helps reduce the transmission of circulating SC-2 variants. However, instead of enabling herd immunity[7], this additional reduction in the overall SC-2 transmission rate is likely to further increase the prevalence of more infectious circulating variants, thereby increasing their adsorption on URT-patrolling DCs. This is likely to further raise immune pressure on viral *intra*-host transmission while augmenting the prevalence of *chronic* SC-2 infection and associated diseases (i.e., 'long Covid'), thereby facilitating natural selection of cryptic, *intra*-host transmissible CoV lineages in chronically SC-2-infected individuals. Given the large-scale immune selection pressure on *intra*-host transmission, a newly emerging cryptic CoV lineage capable of *trans* infection and *trans* fusion of susceptible cells in distant organs would suddenly gain a significant fitness advantage. Its sudden and overwhelming dominance in prevalence would lead to the large-scale abolishment of the immune pressure collectively exerted by highly C-19 vaccinated populations on *intra-host* transmissibility. This would inevitably provoke a rapid and massive surge in *SC-2-associated disseminated intravascular coagulation* (SADIC) causing sudden death in all parts of the population that are unable to eliminate this newly emerging CoV lineage at an early stage of infection.

In other words, the current surge in non-SC-2 respiratory infections in highly C-19 vaccinated countries is expected to expedite the emergence and natural selection of a CoV lineage that proves highly virulent in individuals who failed to train their CMII, either during the acute phase of the SC-2 immune escape pandemic or during the

acute seasonal epidemics currently occurring during this chronic phase of the C-19 immune escape pandemic. Protection from virulence would require such individuals to prophylactically take efficient antivirals.

*Could training of the CMII by the ongoing other respiratory infections enable protection of C-19 vaccinees against a newly emerging virulent CoV lineage?*

The emergence of a new, cryptic CoV lineage with high virulence in highly C-19 vaccinated populations could lead to a hyperacute C-19 tsunami of hospitalizations and deaths, rapidly reducing large parts of the population and enable the remaining, largely unvaccinated population to end this immune escape pandemic through a combination of sterilizing natural immunity and diminished population density.

*Indeed, there is a considerable chance that even C-19 vaccinees who managed to train their CMII following exposure to the other, currently circulating respiratory viruses may still contract a highly virulent infection upon exposure to the newly emerging cryptic lineage (which I tend to call 'HIVICRON').*

This could particularly be the case in regions where there is currently a high prevalence of seasonal flu infections. Exposure to seasonal flu will provide large parts of C-19 vaccinated populations with high titers of anti-flu Abs, even in the absence of vaccination against seasonal flu! As mentioned above, these high titers could make

them highly susceptible to ADED when exposed to the rapidly spreading avian influenza strains.

**The bottom line is that highly C-19 vaccinated countries are on the brink of a major health crisis that could cause healthcare systems to collapse.**

Fig. 2 below summarizes the virological and immunological changes that occurred at the population level during the C-19 pandemic in highly C-19 vaccinated populations. The graph on the left-hand side illustrates the corresponding changes in the pandemic's evolutionary dynamics.

*However, no one knows when the collective DC-mediated immune pressure on viral virulence will rise to a high enough level to collectively trigger the natural selection of phenotypic variants capable of dramatically reducing this pressure and thereby fully unleash viral virulence*[8].

As we cannot measure the level of pressure collectively exerted on the viral infection or viral *trans* infection, and since we do not know the level of immune pressure the virus can tolerate before it gains a new function/ phenotype (i.e., either more infectious or more virulent), *it is impossible to predict when exactly the tsunami of CoV hospitalizations and deaths will occur and how high mortality rates will arise*.

No matter how much data is collected on the evolution of circulating and cryptic variants, including details about their sequences and prevalence, it will not change the uncertainty of any prediction. The same applies to data collected on the evolution of viral wastewater activity, hospitalization rates, and death rates.

It has taken me quite some time to realize that, regardless of how much surveillance and sequencing data are gathered, *any prediction about the timing and amplitude of the tsunami event remains uncertain*.

*The only thing that can be stated with certainty is that as the prevalence of long Covid increases and cases of acute severe C-19 disease rise simultaneously, we move closer to the predicted tsunami, and the surge of other respiratory viral outbreaks in highly C-19 vaccinated countries will only expedite that progression.*

A sudden exponential increase in the rate of hyperacute deaths will unambiguously signal the start of a powerful but brief CoV tsunami. *None of this, however, will affect the health of unvaccinated individuals who have been regularly exposed during the immune escape pandemic and do not suffer from underlying immunosuppressive conditions.*

In other words, *the transition from a metastable pandemic state to a stable post-pandemic state will likely occur as a short but spectacular surge in case fatalities, triggered once a CoV lineage shed by a chronically SC-2-infected individual and capable*

*of escaping DC*-mediated inhibition of viral *trans* infection is selected as the dominantly circulating lineage in highly C-19 vaccinated populations. *There can be no doubt that the virulence of such a CoV lineage will primarily affect C-19 vaccinees and unvaccinated individuals who previously suffered severe C-19 disease.*

**We're in the final stretch now, just staring at the horizon, fully aware that the tsunami could appear at any moment. What else or what more could I say?**

The laws of thermodynamics are increasingly making it clear to me that no single model, no single method of surveillance, sequencing, or any other tracking of viral mutants can exactly predict *when* Nature will proceed with this hyperacute event to restore a sound, low-energy equilibrium.

As it has always been my goal to share scientific truth—not only to warn people about the harmful outcomes of human intervention in this pandemic *but also to predict when Nature will retaliate*—it is now time for me to end my deductive research and communications on this disastrous yet intriguing phenomenon, brought about by the largest gain-of-function experiment ever conducted in the history of mankind, one that, however, involved the human species itself.

I have never been interested in the molecular details revealed by either virological or immunological analyses unless they could potentially help me predict the type and timing of the end of this pandemic. I believe I have largely succeeded in sharing

substantial parts of the biological truth. However, I eventually have to admit that *I will not be able to predict the exact moment when Nature will finally take back control over the health chaos orchestrated by mankind*. As I said, no single analysis can shed light on that. This is the complete uncertainty in which we currently find ourselves, despite living in an era of unprecedented technological revolution. We are nothing compared to Nature. From now on, I intend to spend more of the precious time that remains exploring and admiring its beauty, as well as that of the people who respect it. I will continue to participate in the Immune Biology Forum for as long as there is broad and genuine interest of those who want to learn more about the unimaginably disastrous consequences of this insane and unprecedented interference with the immune system of individuals and even with the collective immune protection of entire populations.

**Conclusion**

Connecting the dots between the overwhelming occurrence of a diverse spectrum of diseases, the ongoing evolution of the virus, the various manifestations of collective immune dysregulation, and the emergence of other viral panzootics (e.g., avian flu) and ongoing outbreaks of viral respiratory infections (e.g., seasonal flu, RSV, hMPV) in highly C-19 vaccinated populations, it becomes difficult to avoid the conclusion that these unprecedented phenomena—particularly their temporal and spatial overlap—are indirectly the result of reckless human intervention in the collective host immune response to the SC-2 pandemic.

The more the prevalence of chronic C-19 disease replaces that of acute C-19 disease, the closer we come to a tsunami of C-19 hospitalizations and mortality.

As long as cryptic SC-2 lineages emerging in chronically SC-2-infected individuals do not overcome the collective immune pressure exerted by highly C-19 vaccinated populations on viral virulence, the pandemic situation remains '*metastable*,' giving society the impression that the pandemic is subsiding. However, outbreaks of other respiratory illnesses in highly C-19 vaccinated countries are currently increasing the likelihood of the remaining immune pressure collapsing. As the sudden emergence of a new, 'suitable' SC-2 lineage is highly likely to result in a hyperacute CoV pandemic in highly C-19 vaccinated countries, I keep warning that society in these countries will be caught off guard.

**Who will clean up all the mess and restore order?**

In my view, these unprecedented phenomena and their spatial and temporal overlapping in highly C-19 vaccinated countries highlight the detrimental consequences of interfering with natural immunity at both, the individual and population level during a pandemic caused by an acute, self-limiting viral infection. When attempting to connect all these dots, I believe we must acknowledge that only Nature can ultimately bring an end to this immune escape pandemic, and its course will not be positively influenced by any of the vaccines that our public health

authorities or so-called ‘experts’, who have shown incompetence and ignorance all along, continue advising as the holy grail of public health interventions.

[1] A *quasispecies* is a well-defined distribution of highly similar but genetically distinct variants that is generated by a mutation-selection process. The composition of a quasispecies is dynamic, with certain variants becoming dominant under selective pressures from environmental changes such as the immune response or antiviral treatments. The diversity within a quasispecies gives the virus a survival advantage by allowing rapid adaptation to such environmental changes.

[2] The *redirection of cellular immunity* occurs following the *refocusing of humoral immunity* and involves a shift from cytotoxic T lymphocyte (CTL)-mediated killing/ elimination of virus-infected host cells at an early stage of infection to dendritic cell (DC)-mediated adsorption/ elimination of infectious virions (see Figs. 1 and 2).

[3] Cryptic SARS-CoV-2 (SC-2) variants may harbor novel mutations that are selected during chronic SC-2 infections and are not commonly seen in acute infections. Although they may eventually be shed into the population, they do not spread widely in populations that either do not exert immune pressure or exert a type of immune pressure that fails to provide these variants with a fitness advantage. These variants are, therefore, not regularly identified in wastewater analyses or through standard viral

surveillance and tracking methods, which is why they are referred to as 'cryptic'. However, *under specific collective immune pressure*, such as that exerted by highly C-19 vaccinated populations*****, *a specific emerging cryptic variant* could, however, gain a competitive advantage.

*****but not in previously SC-2 infected populations, as the latter do not exhibit a high prevalence of subneutralizing anti-Spike protein antibodies

[4] Chronic infections provide a prolonged environment for viral replication and evolution, enabling the virus to acquire mutations that improve its fitness within the specific host. These adaptations may make the virus better suited for *intra-host* transmission between cell types or tissues.

[5] For the purpose of this article, 'suitable' means 'being well adapted to overcome the suboptimal population-level immune pressure on viral *trans* infection and *trans* fusion.'

[6] 'Metastable' refers to a state where the equilibrium is only temporarily stable. This can be visualized by the position of a golf ball in a small hole on a steep slope. It is stable under small disturbances (e.g., in the case of weak selection pressure) as those will only make it roll around inside the small hole but then leave it to return to its resting position. However, if perturbed strongly enough (i.e., in the case of strong

selection pressure), it can transition to a much lower and stable energy state as a strong push will provide it with enough energy to escape the confines of the hole (i.e., to overcome the energy barrier preventing its transition to a lower energy state) and roll down the steep slope to a much lower position, which represents a more stable equilibrium.

[7] To explain why additional acquisition of CMII training in vaccinees at this late stage cannot longer contribute to herd immunity, I'd like to refer to my answer to a Q raised on the Immune Biology Forum (https://lnkd.in/e9ZRVsFQ):

"*You seem to be suggesting that C-19 vaccinees could benefit from exposure (to other currently circulating cold viruses). If so, then wouldn't that create counter pressure to the imminent tsunami? And if an inflection point is reached, herd immunity is then possible?*"

Answer: As the pandemic has transitioned into a chronic phase, other "cold" viruses are no longer being largely outcompeted by SC-2 variants (see more accurate explanation above in the text). This allows these viruses to cause illness, particularly in poorly trained C-19 vaccinees and those recovering from severe Covid-19. This situation is especially concerning for vaccinated individuals with underlying immunosuppressive conditions, as they are at higher risk of severe illness and may, therefore, require antiviral treatment.

However, for others, this could present an opportunity for innate immune system training. It is important to note, though, that while this may enhance their immune protection, it will not enable herd immunity. Why not?

Achieving sterilizing immunity requires the synergistic collaboration of the innate and adaptive immune systems unless cell-mediated innate immunity (CMII) becomes exceptionally robust, capable of eliminating all viral load independently of the adaptive immune system. This level of immune competence has now been observed in unvaccinated individuals who were repeatedly exposed to the virus during this immune escape pandemic. For C-19 vaccinated individuals, exposure to one or more of the other circulating respiratory viruses may now enable their CMII to synergize with their adaptive immune system, potentially sterilizing these new infections. *However, they won't be able to strengthen their CMII strongly enough to sterilize the remaining circulating SC-2 variants in the population such as to generate herd immunity*. There are two key reasons for this:

1. Other respiratory infections will be effectively contained, as sterilizing immunity prevents immune escape and recurrent infection.

2. The partial reduction—but not complete sterilization—of SC-2 transmission will rapidly cause the population to exert selective pressure on viral *trans* infection and *trans* fusion (see text).

As SC-2's intrinsic infectiousness is already nearing its upper limit (recent increases have only been marginal), the virus's survival will now likely require all brakes on viral intra-host transmission to be lifted. This, in turn, is expected to drive natural selection of a virulent CoV lineage and trigger a tsunami of hyperacute fatalities. Tragically, this is likely to affect a large part of C-19 vaccinees as only a minority of healthy C-19 vaccinees will have an opportunity to train their CMII following exposure to other currently circulating respiratory viruses (e.g., seasonal Flu, RSV, hMPV).

[8] For the purpose of this article, 'virulence' refers to the capacity of the virus to *trans* infect and *trans* fuse host cells in susceptible individuals. Virulence, therefore, greatly depends on the immune status of the exposed individual.

Figures:
Fig. 1:

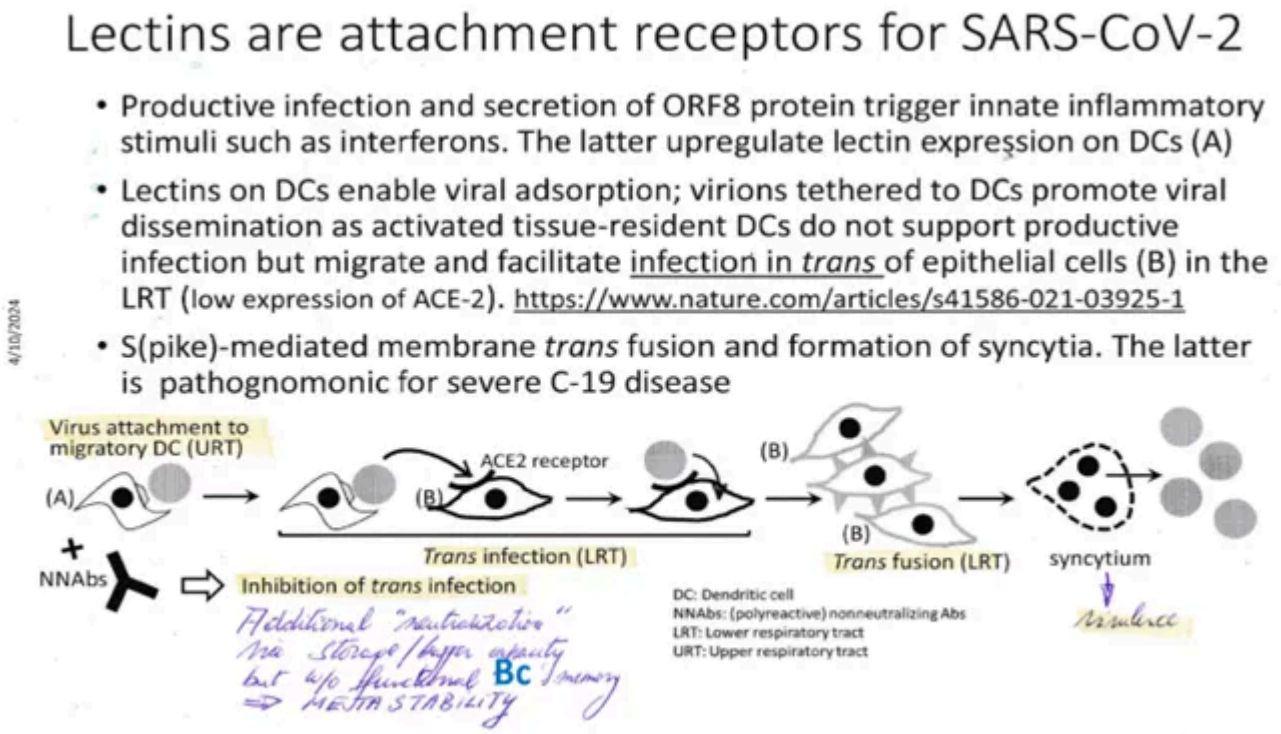
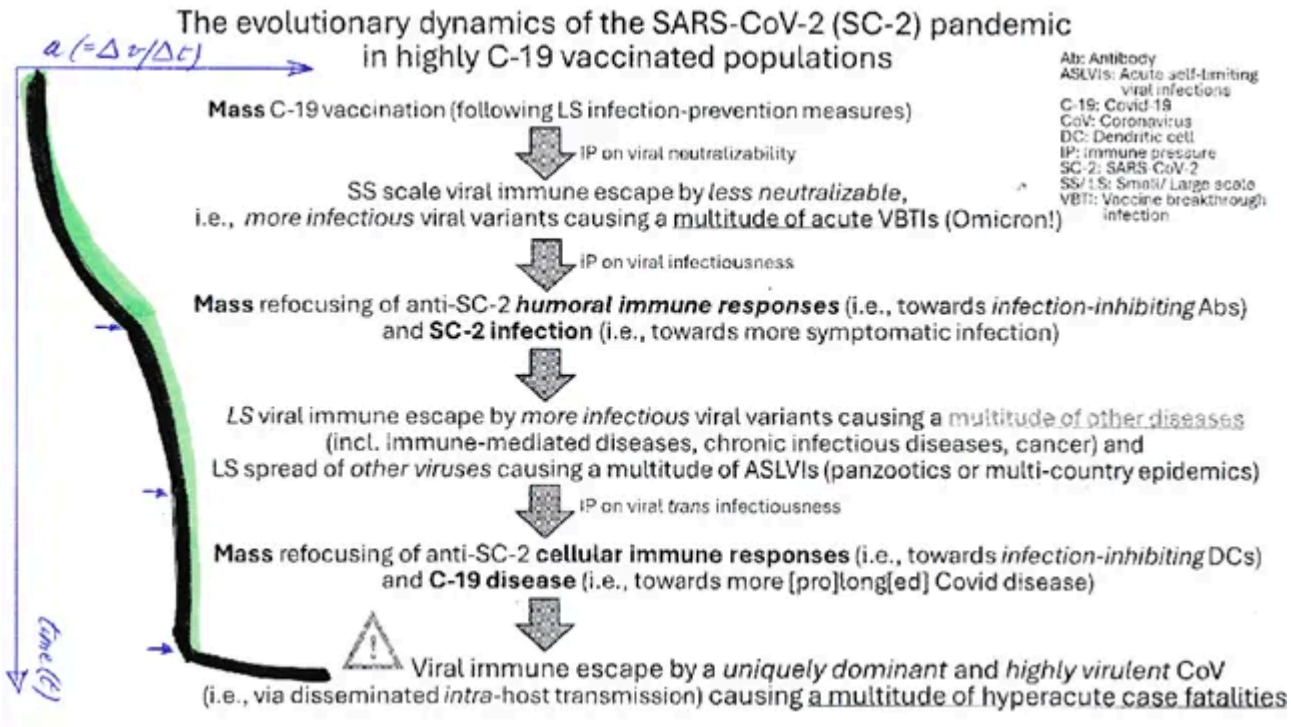

Lectins are attachment receptors for SARS-CoV-2
• Productive infection and secretion of ORF8 protein trigger innate inflammatory stimuli such as interferons. The latter upregulate lectin expression on DCs (A)
• Lectins on DCs enable viral adsorption; virions tethered to DCs promote viral dissemination as activated tissue-resident DCs do not support productive infection but migrate and facilitate infection in trans of epithelial cells (B) in the LRT (low expression of ACE-2). https://www.nature.com/articles/s41586-021-03925-1
• S(pike)-mediated membrane trans fusion and formation of syncytia. The latter is pathognomonic for severe C-19 disease
4/10/2024
Virus attachment to migratory DC (URT)
(A)
ACE2 receptor
(B)
Trans infection (LRT)
(B)
(B)
Trans fusion (LRT)
syncytium
NNAbs
Inhibition of trans infection
Additional "neutralization" via storage/hyper capacity but w/o functional Bc memory ⇒ META STABILITY
DC: Dendritic cell
NNAbs: (polyreactive) nonneutralizing Abs
LRT: Lower respiratory tract
URT: Upper respiratory tract

Fig. 2

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