

The science behind the catastrophic consequences of thoughtless human intervention in the Covid-19 pandemic

– Geert Vanden Bossche (DVM, PhD, March 13 2021)

I am herewith posting a list of a series of publications that have been instrumental in providing enlightening insights on the interplay between Covid-19 and the host immune system. They provide so to speak critical pieces of the puzzle that I have been putting together. Entire puzzles are rarely published. That's why publications rarely bring solutions to complex problems. For your convenience, I have allocated the publications I consulted to different categories. As you will appreciate, I have been tapping into several disciplines. To 'solve' a problem as complex as a viral pandemic, one has to draw from several different fields, including epidemiology, (molecular) biology, virology, immunology, genetics, vaccinology and even biophysics. Again, this is why 'finished' puzzles cannot be found in science journals specifically dedicated to a specific field of interest.

The publications attached support my scientific interpretation of how a natural pandemic develops and how its natural course can be profoundly disturbed by human intervention. For your convenience, I attach a synopsis of my science-based postulate below. I invite scientists from all over the world to read it and reflect on how we could shift gears and possibly intervene in ways that prevent the emergence of additional highly infectious Covid-19 variants and eventually enable eradication of variants that are circulating already.

Synopsis

I cannot emphasize enough how passionate I am about vaccines, but I cannot accept that we use vaccines which, instead of mitigating the Covid-19 pandemic, are now at risk of dramatically aggravating it.

The original Covid-19 strain was only causing mild symptoms, or even no symptoms at all, in the vast majority of healthy individuals. So, before recommending administration of any type of current Covid-19 vaccines to everyone, one should first make sure that the vaccine will reduce the rate of morbidity and mortality below the rates one could reasonably expect when letting the pandemic run its natural course. It's even more simple than that: if one analyses the dynamics of a pandemic caused by a natural, self-limiting viral infection (e.g., Influenza pandemic during World War One), it becomes obvious that the toll taken on human lives is no higher than what is strictly required for the virus to perpetuate. Without human intervention, a pandemic ultimately results in herd immunity. This potentially leaves the door open for the virus to become endemic with interspersed seasonal flare-ups (as we usually see, for example, with the influenza virus). No pandemic has lasted longer than two years, not even Spanish flu or Swine flu and, once herd immunity is established, resurgence of the virus is controlled by our immune systems thanks to their memory of previous encounters with the virus.

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Hence, in order for a vaccine to do better than the natural pandemic, it would need to expedite herd immunity. However, it's exactly the opposite what we are seeing right now: the vaccines are not able to prevent viral spread by vaccine recipients exposed to the emerging highly infectious strains. This is preventing herd immunity from developing. Whereas at the outset of the Covid-19 pandemic, innate immunity in healthy people provided for a solid first line of immune defense to Covid-19, this is no longer the case when highly infectious strains are increasingly dominating the scene. Healthy subjects, including children, are now increasingly exposed to circulating highly infectious strains while the quality or quantity of their antibodies is insufficient.

Why are the Covid-19 vaccines likely to enhance viral infectiousness? It is because they are prophylactic vaccines – designed to build immunity in individuals before they get exposed to the pathogen/virus. They are not suitable at all for administration to people during a pandemic because the likelihood that a vaccine recipient already comes under attack while not yet being endowed with a full-fledged immune response increases as the infectious pressure augments. This particularly applies in case of highly infectious circulating variants.

What happens when you get a vaccine? For an individual who has just received the first dose of vaccine, his or her body will be in the process of building an immune response. It could take several weeks for the immune response to be fully developed and if you are exposed to the virus during this time, your immune response may be too weak to effectively fight the virus. Even though the first dose may protect you from developing symptoms, the virus may still be able to replicate and transmit. Exerting high immune pressure without preventing viral replication and transmission is a recipe for selective viral immune escape. However, what we are now more and more observing is even more worrisome: even those who got fully vaccinated before exposure to Covid-19 are no longer controlling virus replication and transmission. This is because they're now increasingly infected by more infectious variants, the spike protein of which is different from the one comprised in the vaccine. Hence, the virus increasingly evades the vaccinal antibody response. We have already seen this in many care homes where highly infectious variants have been spreading within no time despite large vaccine coverage rates (i.e., up to 80-90%). The only benefit of these vaccines is that they may temporarily protect from severe disease and mortality (depending on the antigenic features of the infecting variant).

Selective immune evasion also favors further dissemination of highly infectious strains as mass vaccination is now increasingly turning vaccine recipients into asymptomatic spreaders. The latter transmit highly infectious virus to the unprotected or not yet infected subjects. This is exactly the opposite of what the vaccines were supposed to do. Indeed, there is now a general consensus that the vaccines will, indeed, fail to generate herd immunity. In addition, they will also fail to eliminate the steadily increasing number of highly infectious strains because the vaccinal antibodies do no longer match with the variant spike protein of the circulating strains whereas they're still hampering binding of natural antibodies to the virus¹.

The combination of immune escape and dominant circulation of highly infectious variants is a recipe for expediting viral resistance to the vaccine and long-lived suppression of our innate immune response against Coronaviruses in general. It is impossible to scientifically understand how this could have a happy end. Humanity, therefore, is at crossroads. Continuing mass vaccination with these 'leaky' vaccines (see 'leaky' vaccines under references from the literature) in the course of a full-blown pandemic inevitably implies that we will witness the emergence of more, more infectious variants putting people at a higher risk of severe disease.

In conclusion: while vaccination may help to momentarily protect an individual, mass vaccination of individuals during the height of a pandemic is going to worsen the global situation by encouraging the virus to select specific mutations enabling it to overcome 'suboptimal' immunologic hurdles. As a consequence, the global population will likely have to deal with a worse version of the virus and a worse health-care situation than earlier in the pandemic. We should stop using conventional prophylactic vaccines in the ongoing Covid-19 mass vaccination campaigns.

Neutralizing anti-Covid-19 IgGs have high AFFINITY for S, whereas IgM have high AVIDITY for the virus; anti-S Abs may still weakly bind to S, even though they cannot prevent binding of the virus to ACE2 (as AFFINITY of S for ACE2 is much higher than for S-specific Abs). On the other hand, even weak binding of highly specific IgGs to S can prevent binding of multimeric IgMs as binding of the latter is not S-specific. Indeed, multimeric IgMs don't interact with individual antigens but bind through multivalent interactions with repetitive patterns on the surface of the virus ('ensemble effect'). So, despite their high AVIDITY for the virus, IgMs cannot outcompete S-specific IgGs for binding to S.

Supportive references from the literature

Topic 1: Natural antibodies (B-1A cells, sIgM, natural Abs & innate immunity to CoV and Covid-19)

[https://www.thelancet.com/journals/lanchi/article/PIIS2352-4642\(20\)30131-0/fulltext](https://www.thelancet.com/journals/lanchi/article/PIIS2352-4642(20)30131-0/fulltext)

doi: [https://doi.org/10.1016/S2352-4642\(20\)30131-0](https://doi.org/10.1016/S2352-4642(20)30131-0)

<https://www.frontiersin.org/articles/10.3389/fimmu.2020.02139/full>

doi : <https://doi.org/10.3389/fimmu.2020.02139>

<https://www.nature.com/articles/s41385-020-00359-2>

doi : <https://doi.org/10.1038/s41385-020-00359-2>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5526850/>

doi : <https://doi.org/10.3389/fimmu.2017.00872>

<https://www.frontiersin.org/articles/10.3389/fimmu.2020.595535/full>

doi: <https://doi.org/10.3389/fimmu.2020.595535>

https://journals.lww.com/shockjournal/fulltext/2020/11000/therapeutic_potential_of_b_1a_cells_in_covid_19_2.aspx

doi: <https://doi.org/10.1097/SHK.0000000000001610>

<https://www.frontiersin.org/articles/10.3389/fphar.2020.01309/full>

doi: <https://doi.org/10.3389/fphar.2020.01309>

<https://pubmed.ncbi.nlm.nih.gov/23692567/>

doi: <https://doi.org/10.1111/nyas.12137>

<https://www.nature.com/articles/s41467-020-20247-4.pdf>

doi: <https://doi.org/10.1038/s41467-020-20247-4>

<https://pubmed.ncbi.nlm.nih.gov/20948548/>

doi : <https://doi.org/10.1038/nri2849>

<https://www.sciencedirect.com/science/article/pii/S1939455120303793>

doi : <https://doi.org/10.1016/j.waojou.2020.100476>

Topic 2 :

- **Role of natural Abs and NK cells in asymptomatic carriers**
- **Substantial transmission by asymptotically infected subjects ; protection of asymptomatic carriers not due to Abs**

<https://www.medrxiv.org/content/10.1101/2020.12.18.20248447v1>

doi : <https://doi.org/10.1101/2020.12.18.20248447>

<https://pubmed.ncbi.nlm.nih.gov/33391280/>

doi : <https://doi.org/10.3389/fimmu.2020.610300>

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7608887/>

doi : <https://doi.org/10.1371/journal.pone.0241536>

topic 3 :

- **Natural Abs facilitate MHC class I-restricted antigen presentation**

- **Conserved, CoV-associated cell surface-expressed MHC cl. I peptides**

<https://www.nature.com/articles/nm933>

doi: <https://10.1038/nm933>

<https://pubmed.ncbi.nlm.nih.gov/19439480/>

doi : <https://10.1128/JVI.00079-09>

topic 4 :

- **Abs may bind to Sars-CoV-2 without neutralizing the virus/ preventing infection**

<https://www.pennmedicine.org/news/news-releases/2021/february/antibodies-to-common-cold-coronaviruses-do-not-protect-against-sars-cov2>

Topic 5 :

- **Natural and vaccine-induced immune escape**

<https://www.biorxiv.org/content/10.1101/2020.12.28.424451v1>

doi : <https://doi.org/10.1101/2020.12.28.424451>

<https://science.sciencemag.org/content/371/6527/329>

doi: <https://10.1126/science.371.6527.329>

<https://journals.plos.org/plosbiology/article?id=10.1371/journal.pbio.1002198>

doi: <https://doi.org/10.1371/journal.pbio.1002198>

topic 6 :

- **Mechanism of viral shedding and clearance**

[https://www.thelancet.com/journals/lanmic/article/PIIS2666-5247\(20\)30172-5/fulltext](https://www.thelancet.com/journals/lanmic/article/PIIS2666-5247(20)30172-5/fulltext)

doi: [https://doi.org/10.1016/S2666-5247\(20\)30172-5](https://doi.org/10.1016/S2666-5247(20)30172-5)

topic 7 :

- **Dynamics of humoral anti-Covid-19 immune response and potential for reinfection**

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7641391/>

doi : <https://doi.org/10.1099/jgv.0.001439>

topic 8 :

Lessons learned from Smallpox vaccines and Influenza pandemic 1918

<https://pubmed.ncbi.nlm.nih.gov/20860482/>

doi: <https://10.2217/fmb.10.98>

<https://www.cnbc.com/2020/09/28/comparing-1918-flu-vs-coronavirus.html>

<https://www.smithsonianmag.com/science-nature/compare-flu-pandemic-1918-and-covid-19-caution-180975040/>

<https://theconversation.com/what-makes-a-wave-of-disease-an-epidemiologist-explains-141573>