SARS-COV-2 (COVID-19): The Essentials
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Dear Brothers and Sisters in Christ,

As promised, here is a brief summary of the essentials regarding Covid disease and Covid-vaccines.

Prologue (disclaimers):
1. This is not intended to be exhaustive or comprehensive; I am not a person inclined to *curiositas*. For simplicity and brevity sake, a semi-telegraphic style is employed. This document is meant to include the essentials as seen from my perspective of a scientist/doctor and moral theologian/bioethicist.
Many of you may already have a wealth of knowledge on the Covid issue and may not find this document informative.
2. The field is *evolving*. This means:
a) We do not have answers to many questions about either the disease itself or the vaccines.
b) We should not make hasty conclusions or unsound conjectures beyond what is permissible by the available empirical data. Scientific data in themselves are neutral, but the human heart with its ideological agenda can introduce therein subtle yet poisonous erroneous (or hasty) conclusion or interpretation.
FYI: By "data", I mean, scientific studies (both preclinical and clinical), also referred to as primary source. The evaluation of scientific studies needs to follow certain fundamental principles, e.g. the Material and Methods section of any scientific paper needs to be read with a razor sharp discernment.

Part I Origin of SARS-COV-2 virus
Two theories: Natural emergence vs. Gain-of-Function (GOF) research and laboratory escape
FYI: Natural emergence necessarily a series of traceable spontaneous mutations and/or recombination occurring over some period of time. GOF implies genetic engineering (e.g., production of virus chimera).
For this I refer you to the attached article (Wade 2021). This is secondary source article, but it is overall well written, grounded in scientific data instead of political conjectures.
Here, just to summarize the key points:
1. No intermediary animal host species has been identified – identification of intermediary host species would give supporting evidence of a natural emergence.
2. GOF research producing chimera virus ongoing for a couple of decades. Collaboration USA-China (including U. North Carolina – Wuhan Institute of Virology (WIV) – NIH funding – EcoHealth Alliance) on this project of "reprogramming the S (= spike) protein and generating chimeric coronaviruses capable of infecting humanized mice."
FYI: humanized mice = they are genetically engineered to carry the human version of ACE 2 (= angiotensin converting enzyme-2) receptor.
3. Several WIV researchers with Covid symptoms well before the 1st identified case of Covid outbreak.
4. Most important: the furin cleavage site at the junction of the S1 and S2 subunits of S protein.

(i) Furin cleavage site is found in many viruses, including coronavirus. Four genera of coronavirus: alpha, beta, gamma, delta. Betacoronavirus genus includes five subgenera: Sarbecovirus (e.g. SARS-COV and SARS-COV-2), Merbecovirus (e.g., MERS-COV, Embecorvirus (e.g. human coronavirus OC43 and human coronavirus HKU1, both causing common cold), Hibecovirus and Nobecovirus (both only found in bats)
SARS-COV-2 is the only virus in subgenus Sarbecovirus having the furin cleavage site, while even its closest relatives, bat coronavirus RaTG13 (sequence identity 97.7%) and pangolin coronaviruses (92.9%–90.7%), do not.
(ii): The furin cleavage site consists of four amino acids PRRA (proline-arginine-arginine-alanine) sequence, which are encoded by 12 inserted nucleotides in the S gene.
Most curious feature: the codons CGG-CGG encoding the arginine doublet. This codon doublet encoding the arginine doublet is not found in any of the furin sites in other viral proteins expressed by a wide range of viruses.
Arginine can be encoded by any of the following six codons: CGU, CGC, CGA, CGG, AGA or AGG. CGG is the least popular codon for arginine in coronavirus. Even in the SARS-COV-2, only two of the 42 arginines of the S protein are encoded by this codon – and both of them are found as a doublet in the PRRA motif of the furin cleavage site! The likelihood for this to have resulted from spontaneous mutation is minimal. Recombination is unlikely since this requires a closely related virus ("the donor") with this arginine doublet encoded by the CGG-CGG codon doublet.
FYI: Insertion of a furin site is a common practice in GOF research to make a virus more infective.

Part II Covid disease

1. The main problem with Covid is not about the infection rate since most people recover from Covid illness. The problem has to do with the severe manifestations of the disease among those in the high-risk/vulnerable group (elderly age group, people with comorbidity, especially obesity). This group is the group that needs to be "locked down". For the sake of this vulnerable group, however, other people must take the required precautions, especially when coming into contact with them. Imposing the lock-down on the non-vulnerable group goes against common sense.
a) The overall mortality rate is very low. High mortality mainly among the vulnerable age group
b) Spreading: 3 Cs = *crowded, closed space, close contact*. The 3cs are the conditions favorable for the spread of the virus.
c) The severe symptomatology which can lead to death has to do with an "overreacting immune system" ultimately resulting in micro-clots throughout the body, especially in the lungs. See further discussion below

2. S protein and ACE 2 receptors
SARS-COV-2 enters human cells via ACE 2 receptors.
FYI: Compared to other coronavirus, SARS-COV-2 demonstrates thousand fold affinity to ACE 2 receptors. Non- Covid coronaviruses are known commensals living in the respiratory secretions of the nasopharynx.
a) ACE 2 receptors are ubiquitous on epithelium throughout the body, from the cells lining the human airways to those lining the gastrointestinal (GI) tract to the olfactory neuroepithelium. The latter is a direct pathway into the brain cisterns.

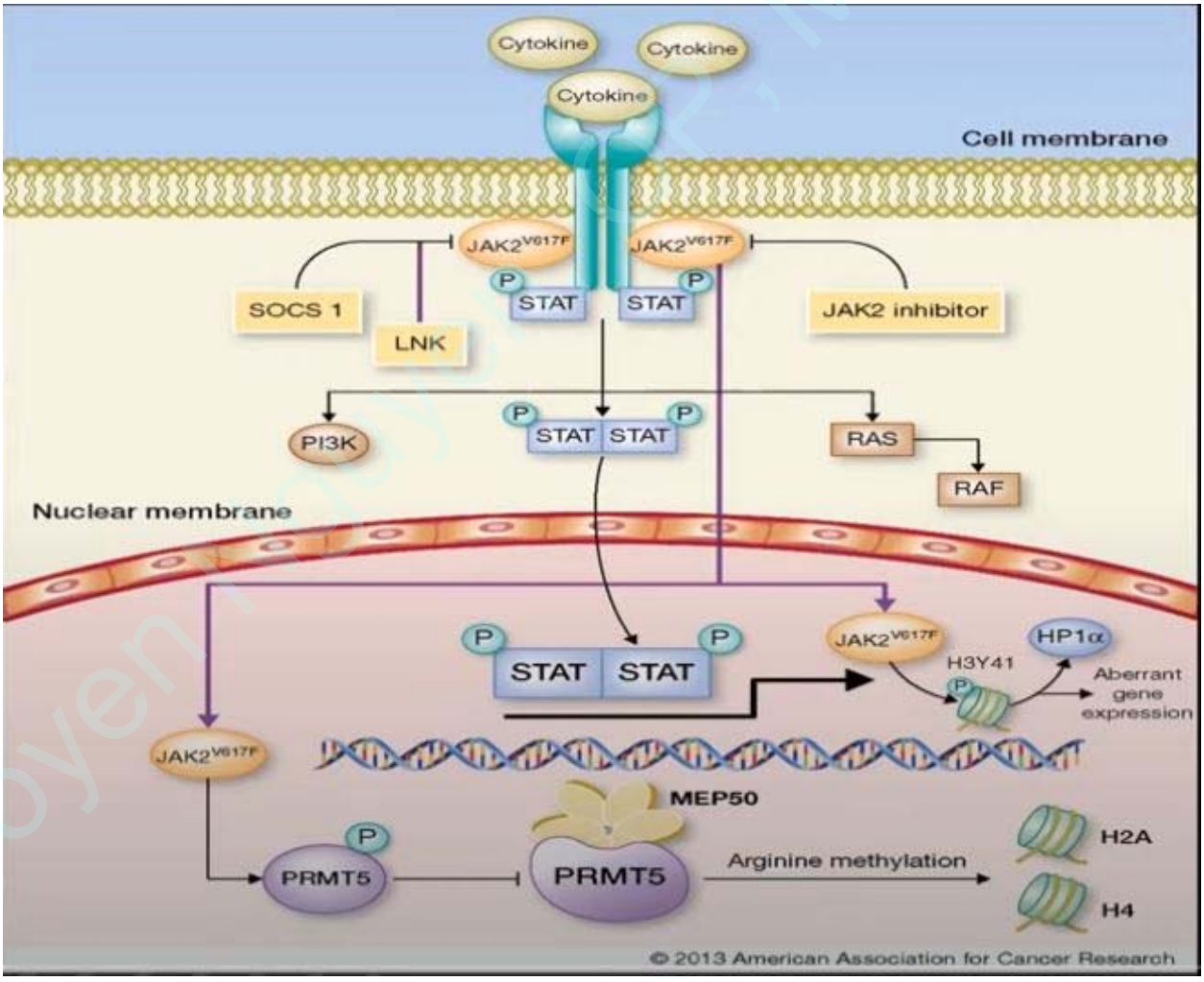
Consequence: protean manifestations of Covid symptoms especially as the disease progresses – from dyspnea (lung) to diarrhea (GI) to loss of smell and taste, to signs and symptoms of other affected organs (neurological, cardiac, renal, etc.)
b) S1 subunit helps bind the virion to ACE 2 receptor → furin, present on the cell surface, cuts the viral S protein at the furin cleavage site → activates S2 subunit → mediates the fusion of the viral and cellular membranes fusion → virus enters the cell, dissolves its own protein shells and releases viral RNA → viral RNA hijacks the cell replication machinery and starts to replicate itself; it also manufactures the necessary proteins to assemble new viruses (→ released to infect other cells) → the host cell dies → cellular debris and inflammatory response.

3. Pathophysiology of Covid disease
3.1. SARS-COV-2 activates pericytes via ACE 2 receptor (it is most predominantly present in pericytes) → loss of pericytes → increased endothelial permeability in the lungs.

3.2. Bradykinin (BK) storm
(i) The maintenance of normal blood pressure (BP) entails the renin-angiotensin-aldosterone system (RAAS) which involves an interplay between ACE 2 (which lowers BP) and ACE (which has the opposite effect).
In the lungs, SARS-COV-2 causes decreased/downregulated ACE and increased/upregulated ACE 2, which in turn leads to increased BK → vasodilation and vascular hypermeability (= pulmonary blood vessels expand and leaky) → pulmonary edema
(ii) At the same time, upregulated synthesis of and downregulated degradation of hyaluronic acid (HA) in the lungs → increased levels of HA in the bronchioalveolar space of the lungs. HA can absorb more than 1,000 times its own weight in water to form a hydrogel.
(iii) Combination of (i) + (ii) = BK storm = induced leakage of fluid into the lungs combined with excess HA would likely could form a hydrogel impeding gas exchange (= oxygen uptake and carbon dioxide release) in the lung

3.3. Cytokine storm
(i) Normal anti-viral immune response requires the activation of the inflammatory pathways of the immune system. But exaggerated response of the host's immune system can cause severe disease if remains uncontrolled. Cytokines, an integral component of the immune response, are produced by several immune cells including the innate macrophages, dendritic cells, natural killer cells and the adaptive T and B lymphocytes.
During an innate immune response to a viral infection, pattern recognition receptors (PRRs) recognize different molecular structures that are characteristic to the invading virus. These molecular structures are referred to as pathogen associated molecular patterns (PAMPs). Binding of PAMPs to PRRs triggers the start of the inflammatory response against the invading virus resulting in the activation of several signaling pathways and subsequently transcription factors which induce the expression of genes responsible for production of several products involved in the host's immune response to the virus, among which are the genes encoding several pro-inflammatory cytokines. Three of the most important pro-inflammatory cytokines of the innate immune response are IL-1 (interleukin 1), TNF-α (tissue necrosis factor alpha), and IL-6. Tissue macrophages, mast cells, endothelial, and epithelial cells are the major source of these cytokines during innate immune response.

(ii) The “cytokine storm” results from a sudden acute and exaggerated increase in circulating levels of different pro-inflammatory cytokines including IL-6, IL-1β, TNF- α, and IFN-γ (interferon gamma), among others. This increase in cytokines results in influx of various immune cells such as macrophages, neutrophils, and T cells from the circulation into the site of infection → further activation of the cytokines in question, destructive effects on human tissue resulting from destabilization of endothelial cell-to-cell interactions, damage of vascular barrier, capillary damage, diffuse alveolar damage, multi-organ failure, and ultimately death. Lung injury is one consequence of the cytokine storm, progressing to severe acute respiratory distress syndrome (ARDS).
FYI: cytokine storm is not unique to Covid, it has been reported in other sever infections including influenza H5N1 virus.
(iii) JAK-STAT pathway (JAK =Janus kinase; STAT = signal transduce and activator of transcription) is the cytokine signaling pathway that initiates the cytokine storm. This pathway mediates the effects of a large number of cytokines and growth factors. To date, over 50 cytokines and growth factors have been shown to utilize the four JAK proteins and seven STAT molecules to regulate cell growth, survival, differentiation, motility, and immune responses.
FYI: diagram below for those interested in the details of the JAK-STAT pathway.



3.4. Heightened platelets activation in the lungs and increased serotonin
a) Day 1-7: via ACE 2 mechanism, SARS-COV-2 causes (i) pneumocyte injury and, (ii) pericyte injury → unstable endothelium → leaky alveolar capillaries
b) Day 7 and thereafter, in 20% cases: severe immune mediated platelet activation by immune complex and antibodies → excessive outpouring of platelet-derived inflammatory mediators out of proportion to alveolar injury → severe vasocentric injury in the lungs leading to progressive alveolar damage seen as "ground glass opacity" on CT scan around day 10-11
In 80% cases: no immune mediated platelet activation. The release of platelet-derived inflammatory mediators is proportionate to the viral alveolar injury. The result is disease resolution like in any other upper respiratory viral infections.
FYI: platelets have a thrombotic action (= involved in blood clotting) and also play a role in immune reaction. Not much is know about this second aspect. Conventional anti-platelet meds (e.g., aspirin) have no effect on the immune action of platelets.

c) SARSCOV-2 hyperactivation of platelets is ACE 2 receptor independent. Most likely multimodal mechanism (= involving more than one mechanism), in particular by activation of CD 147 on platelets, and by autoantibodies/immune complexes that activate platelet and cause platelet-leukocyte aggregation (→ thrombi). NB: The latter mechanism is both time and host dependent (= it occurs "late" i.e., after day 7, and only in a subgroup of patients).
d) Platelet activation → platelet degranulation → release a whole host of whole host of platelets mediators, in particular serotonin
e) Increased serotonin in the lungs leading to severe pulmonary vasoconstriction, pulmonary vascular shunting, pulmonary platelet trapping (→ a vicious cycle of further platelet activation and aggregation), increased respiratory drive (= inappropriate rapid breathing even though carbon dioxide level is not increased), and eventually, pulmonary fibrosis.
FYI: Severe alveolar capillary vasoconstriction in the lungs causes blood to back up into bronchopulmonary anastomoses thus producing a vascular shunt bypassing alveoli gas exchange. The result is severe hypoxemia.
f) Increased plasma serotonin can lead to shock, vasoplegia, high fevers, severe diarrhea, various neurological symptoms (e.g., myoclonus, hyperreflexia, agitation, hallucination, hyperactive delirium), coronary vasospasm, and renal failure by constricting renal artery flow, among others.

Summing up this section on pathophysiology: it is rather evident that Covid disease is primarily an immunological disorder in terms of pathophysiological mechanisms.

4. Disease progression (overlapping stages)
The above described pathophysiology is reflected in Covid protean clinical manifestations as the disease progresses through four stages: (i) viral proliferation, (ii) bradykinin storm, (iii) cytokine injury and, (iv) thrombosis. See diagram below (next page).
Principles regarding treatment:

- One must determine at which phase of the disease the patient is based on the clinical signs and symptoms. This will help to select the proper drugs to be used. For instance, it is useless to use antiviral therapy or hydrochloroquine (HCQ) at the phase of cytokine injury or of thrombosis. FYI: this is why it is reported that HCQ does not work in hospitalized patients

- The treatment at every phase, requires the use of a combination of different meds.
- One must treat the disease as soon as possible, especially the phase of viral proliferation. Ambulatory treatment (prior to the onset of cytokine storm) is effective, especially during the 1st ten days. Mortality/morbidity increases with disease progression.

FYI: I cannot give you details regarding treatment since Covid therapy needs to be supervised by physicians and requires a multi-drug approach.

a) Phase 0 intervention: nasal rinses to decrease the viral load + inhalers to dry up secretions in which the virus resides

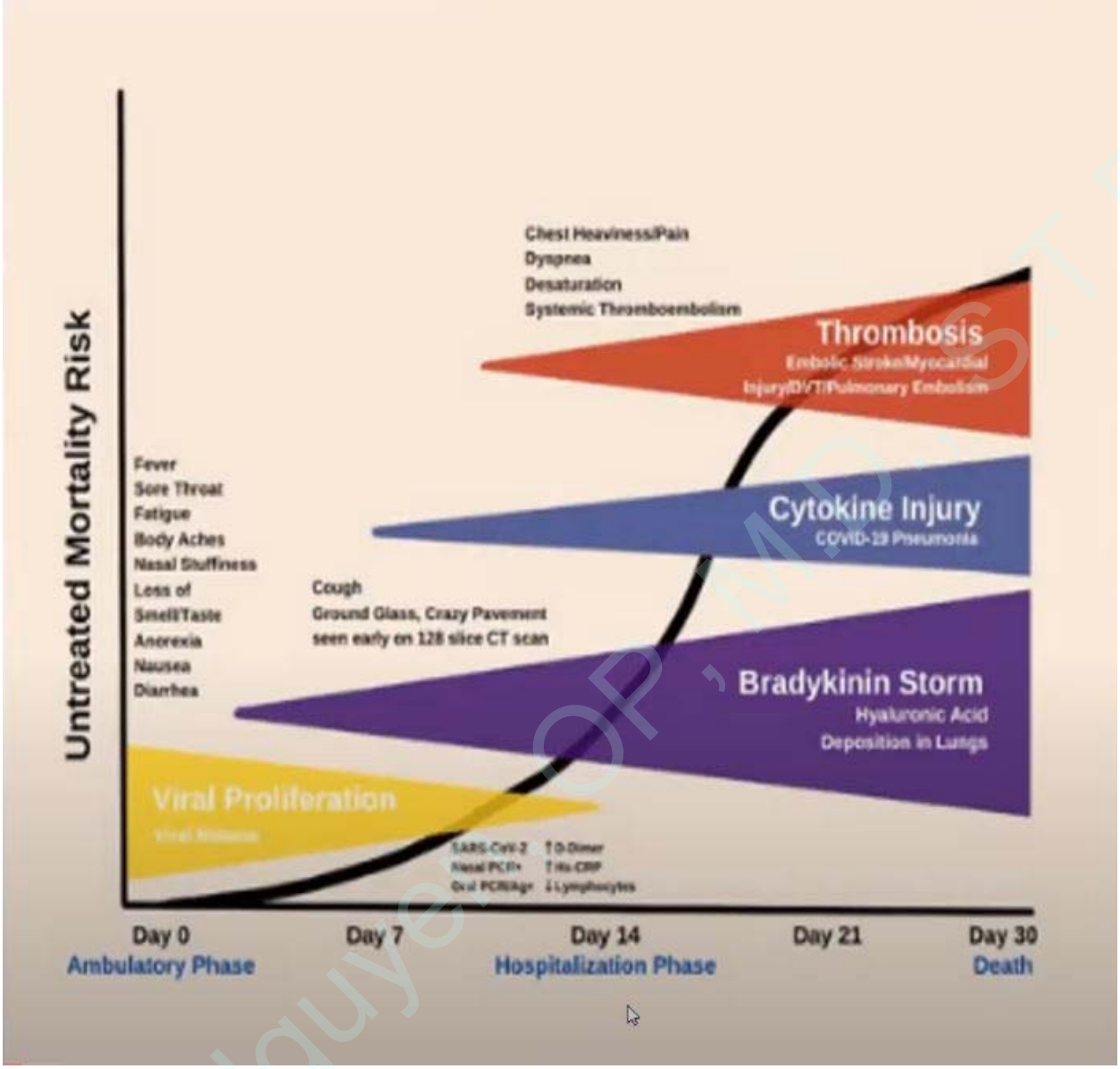
b) Phase 1 intervention to prevent ACE 2 activation (and therefore, prevent or shut down bradykinin storm) by using monoclonal antibodies targeting S protein to impede binding to ACE 2 receptors. Also use meds that decrease bradykinin and meds that deplete pool of serotonin stored within platelet

FYI: monoclonal antibodies against S protein have been approved for emergency use (EUA) only.

c) Phase 2 intervention: at this point the virus has entered the cell. The body tries to contain it by means of intracellular endosomes. Here, use HCQ which can enter the cell to prevent endosomes from breaking, thereby preventing viral replication. Also use antiviral agents and protease inhibitors to stop the production of new viruses.

d) Phase 3 intervention to prevent cytokine storm. At this point, using antiviral agents and/or HCQ will not help. The virus is long gone.

e) Phase 4 intervention to treat thrombosis and other complications (caused by increased serotonin).



Part III Covid vaccines

1. The moral issue: is it licit to get oneself vaccinated with Covid vaccines, of which the production and/or preclinical testing use cell lines derived from aborted fetuses, when there is no other type(s) of vaccine(s), of which the production and/or testing do not involved cell lines derived from abortion?

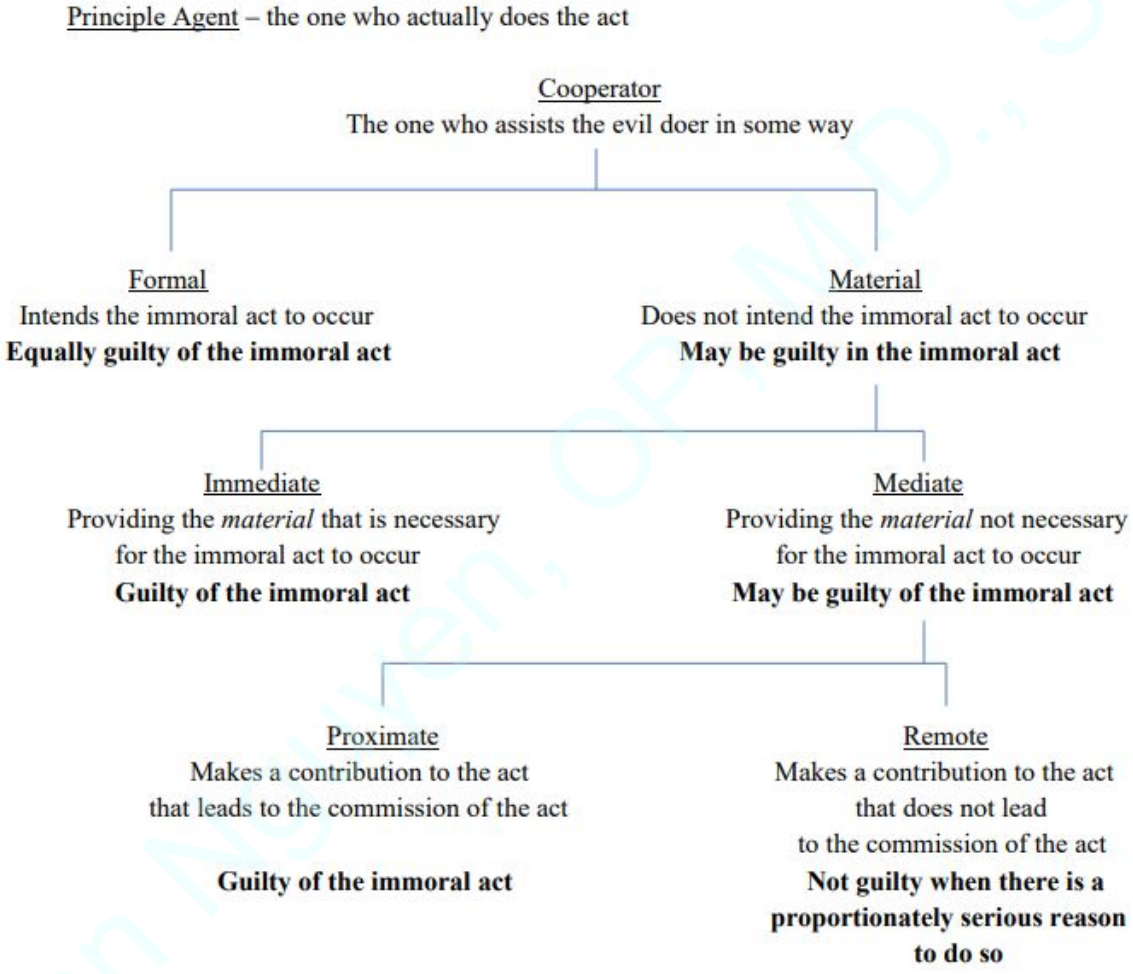
1.1. The "No" argument comes down to this: these vaccines are "tainted" and to take them amounts to an act of cooperation with evil (= cooperation with the abortion industry), thus acting contrary to Catholic faith and morals.

FYI: I will not delve into the scientific inaccuracies (= analogous to fake news) propagated by several pro-life groups that hold this position. My approach is first, check the credentials of their so-called scientists/"doctors" and the veracity of their claims. Examples of "red flags": (i) the claim that babies got aborted via C-section is in itself an *absurdum*, (ii) scientists/authors whose

qualifications are only a master degree in biology, or whose books are not peer-reviewed, or who practice homeopathic medicine without evidence of peer-reviewed research-publications.

1.2. *Sed contra* to the "No" argument

a) Common sense: how do you cooperate with evil if the evil action took place in the past way back in the 1970s? One can cooperate in some activity only if such activity is in the here and now, or in the near future. In philosophical terms, it is metaphysically impossible to cooperate with a past event. This applies to the different categories and subcategories of cooperation with evil (see the chart below)



The issue is not about not cooperating with evil, but rather about not creating scandal: it comes down own to this perception that we (Catholics) are behaving as hypocrites, who on the one hand decry abortion, and on the other hand, have no qualm getting vaccinated with "tainted" vaccines. This perception is unfounded. We should not conflate the issue of abortion with the issue of vaccines (there are not two sides of the same coin).

b) The principle of double effect can be invoked to further support the liceity of getting the "tainted" vaccines. I will not go delve into this issue since point (i) is sufficient to argue against the "No" argument.

c) Scientific grounds: Here we must understand the nature of cell lines derived from aborted fetuses. When we speak about fetal cells or cell lines, we need to be specific (something that the "No" camp does not do!): some types of research need fresh fetal cells every few weeks, others use unaltered fetal cell lines, still others use altered fetal cell lines. Covid vaccines fall into the 3rd category.
(i) Human cells are diploid (= 2n) 46, XX or 46 XY. No diploid human cells of fetuses can be maintained in *in vitro* culture beyond the exceptional maximum of 11 months. Most can be maintained for 5-6 months and requires continuous subcultivations (twice a week) since the primary culture is viable for 1-3 weeks only. The critical threshold is around 40-50 subcultivations when cell degeneration takes place, and this phenomenon is independent of any extrinsic factors (= factors in the culture media).
FYI: Fifty subcultivations roughly equal 10 months
The point is: cultured fetal cells can only undergo a specific number of cellular divisions, and (ii) cryogenically preserved fetal cells can 'remember' how many times they have divided when they have frozen. This is known as the Hayflick factor. In non-technical terms, normal human fetal cells are "mortal". Malignant cells are "immortal". Hence the only way to make normal cells to become immortal is by transforming them into tumor cells. This is what took place with the fetal cell lines HEK 293 and PERC6 used in Covid vaccines.
(ii) The principles of what I describe below for HEK 293 cell line also apply to PRC6 Cell line
HEK 293: This cell line originated from the kidney of an aborted human female embryo and was originally immortalized in 1973 by the integration of a 4 kbp adenoviral 5 (Ad5) genome fragment including the E1A and E1B genes, at chromosome 19.
During subcultivations, the proportion of transformed cells increased such that, by the 7th subcultivation, there are no more normal fetal cells (→ hence in no way can Covid vaccines contain fetal cells), the cell population is quasi tetraploid (= when tested at the 8th subcultivation. By 1997, the cells have been subcultured more than 100 times and considered an established cell line. Several progeny cell lines have been produced from the parental HEK 293 cell line.
The point is: HEK-293 cell lines, whether original/parental or derivatives/progeny, although of human origin, no longer have the normal diploid human karyotype; they produce viral-specific tumor antigen and they do not produce certain proteins expected of normal cells. In brief, they are genomically and transcriptomically abnormal cells. If you were to implant these cells into a woman's uterus, no way could they form an embryo/fetus/baby. They are worse than the cells we found in human cancers.
Last but not list, HEK293 cell lines have been used to produce therapeutic agents such as recombinant erythropoietin, interferons and a whole hosts of others for necessary treatment purposes, as well as to test numerous types of meds.

2. Medical issues
2.1. Background information about mRNA vaccines (key points and quotes taken from Pardi 2018 review article)
a) "The first report of the successful use of *in vitro* transcribed (IVT) mRNA in animals was published in 1990, when reporter gene mRNAs were injected into mice and protein production

[= antibody production] was detected". But the application of mRNA vaccines "has until recently been restricted by the instability and inefficient *in vivo* delivery of mRNA. Recent technological advances have now largely overcome these issues, and multiple mRNA vaccine platforms against infectious diseases and several types of cancer have demonstrated encouraging results in both animal models and humans."
The point is: contrary to popular belief, mRNA vaccine technology did not arise "yesterday" with Covid pandemic.
b) There are several beneficial features of mRNA vaccines over conventional vaccines (such as live attenuated and inactivated viruses), subunit vaccines, as well as DNA-based vaccines. In particular, with regard to safety: "as mRNA is a non-infectious, non-integrating platform, there is no potential risk of infection or insertional mutagenesis. Additionally, mRNA is degraded by normal cellular processes, and its *in vivo* half-life can be regulated through the use of various modifications and delivery methods. The inherent immunogenicity of the mRNA can be down-modulated to further increase the safety profile".
FYI: Ongoing research to evaluate different mRNA vaccine modalities especially with respect to safety concerns
c) Two categories of mRNA vaccines:
(i) Vaccines against infectious agents for prophylactic purpose. Conventional vaccine approaches have been ineffective against challenging viruses such as HIV1, herpes simplex virus, respiratory syncytial virus, etc. There have been several clinical trials (some ongoing) against HIV1, influenza virus, and Zika virus.
(ii) Vaccines against cancer for therapeutic purpose. Cancer vaccines seek to stimulate cell-mediated immune responses (such as those from cytotoxic T lymphocyte) to clear or reduce metastatic tumor burden. FYI: Here, the buzz word is "gene therapy".

2.2. Background information about immune system
FY: I am writing this section for your benefit, namely to address the (unfounded) concerns that mRNA vaccines are reprogramming the immune system. Here, the buzz word is "reprogramming", and upon seeing/hearing this word, many people jump to the conclusion "the vaccine is gene therapy and it will change our genes forever"!!!

The term "reprogramming", understood in immunological terms, refers to vaccines rendering the immune system to become more effective against the pathogen or antigen in question.
Two broad arms of immune system:
(a) Adaptive immune system (B cells, T helper cells, cytotoxic T cells) which includes both humoral and cellular immune responses, and which has long term memory (= memory cells, "remembering the particular antigen/pathogen encountered in a given infection)
One of the function of vaccines is to train the adaptive immune system to produce memory cells to defend the body rapidly against that antigen/pathogen in the future before the latter can cause damage to the body.
In a nutshell, the adaptive arm "learns", when exposed to a particular antigen (either through infection or vaccination) by creating memory cells.
(b) Innate immune system (macrophages, monocytes, dendritic cells, natural killer cells, etc.): how does this system learn?
From what we know thus far, the innate arm does not create memory cells. Innate arm "learns" to respond to various antigens in one of two ways:

(i) An enhanced response to some antigens – referred to as "trained immunity"
(ii) To respond less to some antigens – referred to as "innate immune tolerance" by decreasing cytokines production
NB: This "learning" process of either trained immunity or innate immune tolerance is also referred to as "functional reprogramming of cells of the innate immune system." Such "reprogramming" is known to occur with certain vaccines such as BCG (Bacillus Calmette Guerin), MMR (measles, mumps and rubella).

c) The question one may ask is: do Covid mRNA vaccines also produce a similar learning process of the innate arm? → yes, it does.
Foehse et al, May 2021study of Pfizer Covid vaccines → high levels of antibodies to S protein produced after the 1st vaccination, and even stronger responses after the 2nd dose. Pfizer vaccine has been also reported to activate T helper cells and cytotoxic T-cells specifically against the virus. In a nutshell: Pfizer vaccine induces learning in adaptive arm.
In addition, it was also found that, similar to BCG and MMR vaccines, Pfizer Covid vaccine "also modulates the production of inflammatory cytokine by innate immune cells upon stimulation with both specific (SARS-CoV-2) and non-specific (viral, fungal and bacterial) stimuli".
In sum, Pfizer Covid vaccination "reprograms" both adaptive and innate arms

FYI: (i) In general, innate memory is considered as a non-specific short-lived phenomenon, as opposed to adaptive memory that is long-lived and highly specific.
(ii) One reason why children respond better to Covid is because of innate arm "reprogramming" from continuous exposure to respiratory viral infections.
(iii) "it is important to remember trained tolerance and potentiation do not exclusively depend on the priming stimulus [e.g., vaccines]. A myriad of environmental factors and other variables affects trained innate memory, including individual history of pathogen/antigen exposure, organ and tissue microenvironment, health and metabolic conditions, gender and age."

2.3. Effectiveness and safety
Normally, the production and testing of a vaccine require around two years (or more) before it can be rolled out. This time window is necessary for evaluating the efficacy and safety of the vaccines in question. For several reasons (which I will not discuss here), this was not possible with Covid.
Moreover, rare serious side effects such as vaccine-induced thrombotic-thrombocytopenia (VITT), which occurs in AstraZeneca (AZ) and Johnson&Johnson (J&J) vaccines, may not necessarily be detected during clinical phase 3 trials but only become observable after millions of people have been vaccinated with AZ/J&J.
Therefore, at this point, no answers or only partial answers to questions (see some of the questions listed below) regarding effectiveness and safety.

2.3.1. Effectiveness
a) Some questions
(i) How long does vaccine-induced immunity last (six months, a year, or lifetime)?
(ii) How effective is a particular vaccine against SARS COV-2 variants?

(iii) Any potential interaction between the immune responses to Covid vaccines and the response to other vaccinations, especially the influenza vaccine?

b) What we know so far
(i) Natural immunity in patients who had Covid: Denmark study analyzing of infection rates during the 2nd Covid surge, comparing infection rates between individuals with positive and negative PCR test during the 1st surge → 0.65% of PCR-pos. tested positive again, compared to 3.27% of PCR neg. who tested positive during the 2nd surge → estimated protection against repeat infection after previous SARS-COV-2 infection was 80.5%. However, among those aged 65 years and older, observed protection against repeat infection was 47.1%
In another study: immunity memory observed at 8 months post-Covid infection.
The point is: (i) natural immunity is not 100% effective; (ii) in older people, it drops below 50% because of immune senescence (= natural age-related changes in the immune system of older people). Therefore, we should not expect vaccine-induced immunity to do better than natural immunity.
(ii) Vaccine-induced immunity: Israel study of Pfizer → "The incidence rate of SARS-COV-2 infections among adults aged 16 years and older was 91.5 per 100 000 person-days in the unvaccinated group and 3.1 per 100 000 person-days in the fully vaccinated group, with an estimated vaccine effectiveness (adjusted for age group, sex, and calendar week) against SARS-COV-2 infection of 95·3%".
In this study, vaccine-immunity observed beyond seven months.

2.3.2. Safety issues
Two current most serious side effects: blood clotting and bleeding
a) Vaccine-induced thrombotic-thrombocytopenia (= blood clotting and decreased platelets), 5-20 days after vaccination with AZ or J&J.
FYI: AZ and J&J use an incompetent adenoviral vector to deliver the vaccine into the cell, whereas Pfizer and Moderna use lipid nanoparticles as mRNA delivery tools.
No case of VITT reported in other adenoviral vector-based vaccines, such as Sputnik V, and AD 5-based Covid-19 vaccine.
(i) Incidence of VITT: presumably rare. It only came to light after mass vaccination of millions got vaccinated "The highest incidence was reported from Norway, in which five cases were reported from among approximately 130,000 individuals vaccinated with ChAdOx1 nCoV-19 [= AstraZeneca] suggesting an incidence of 1 in 26,000". In other reports: only a small number of cases after ten of millions got vaccinated.
(ii) The data suggests a preponderance of females (18-50 age range). Risk factor: birth control pills, hormonal replacement therapy, obesity, hypertension. FYI: obesity is associated with increased endogenous estrogenic hormones and other co-morbidities.
(iii) VITT patients present with clinical features resembling those of autoimmune heparin-induced thrombocytopenia.
A distinctive feature of VITT: thrombosis in unusual sites, especially (i) cerebral veins (→ severe headache as a presenting symptom), splanchnic (splenic, portal, mesenteric) veins, and adrenal veins (→ risk for adrenal failure).
Just like in heparin-induced thrombocytopenia, AZ and J&J VITT show high levels of antibodies to platelet factor 4 (PF-4) pointing to an immune-mediated mechanism which still needs to be further elucidated. It is "unclear if PF-4 antibodies are autoantibodies against PF4 induced by

the strong inflammatory stimulus of vaccination or antibodies induced by the vaccine that cross-react with PF4 and platelets".

b) Vaccine-induced thrombocytopenia (= bleeding), observed in Pfizer and Moderna.
→ can lead to severe bleeding, including brain hemorrhage.
This side effect is very rare. The incidence of thrombocytopenia among vaccinated people does not appear to be higher than that in the general population. A predisposing factor is history of ITP (immune thrombocytopenia).
The pathophysiological mechanism of Pfizer/Moderna Covid VIT is still unknown.
FYI: Occurrence of VIT has been in other vaccinations including MMR, Hepatitis and B, diphtheria-tetanus-acellular pertussis, varicella, and influenza.
Vaccines, just like viral infections, can induce thrombocytopenia by immune mediated mechanisms. A most likely mechanism, is "molecular mimicry": an epitope on the vaccine antigen structure shares a similar structure with a self-peptide (= an antigen on cell surface, namely of platelets) leading to the production of autoantibodies production. Notably, this "molecular mimicry" mechanism could be induced not only by the vaccine antigen, but also from other constituents of the vaccines such as adjuvants.

Summing up this section on effectiveness and safety: the decision to get or not to get vaccinated has to be made by the individual, taking into account of the person's own medical history and social context, and of what is currently known about the vaccines. For instance, if you are a heath worker coming in contact with patients, then you should get vaccinated unless there are other overriding health factors. It is not ethical that an organization, be it governmental or ecclesiastical, manipulatively "nudge" into "Yes, you must take the vaccine, otherwise you cannot attend school or travel, or etc.", or "No, you should not take any of the vaccines because they are all "tainted" and therefore you commit a sin". Obviously, if there were a clean, effective and safe vaccine, then no Catholics should go for the "tainted" ones

Epilogue
I hope that this document has been informative to you. Some of medical/scientific jargon has to be included, there was no way to get around them. Some of the information are from publications in May/June 2021.
I would like to share a personal story: I got fully vaccinated (Pfizer) by the end of March. Nevertheless I continued to use face mask. The first time I did not use it in an enclosed space and in close contact with other people was on Pentecost Sunday May 23rd during our meeting in Aquinas Hall. Ironically, two days later (Tue), I started having a hint of an itchy throat. Then I had to fly to Austin, TX to deliver a one hour lecture on Brain Death to the Texas Right to Life. Fifteen minutes after I finished the lecture, I lost my voice!! The next day, my cold symptoms became overt (general aching, paroxysmal coughing). I had to fly back home. Obviously, the flights could only make my cold worse. The paroxysmal coughing (especially during the night) did not end until this past Thu June 10th. But until today June 13th, my voice is still hoarse!
I have a family history of asthma which renders me vulnerable to upper respiratory infection. People recover from a cold after a week, in my case, it can go for two weeks!
The point I would like to make is: Covid or not, it helps to protect oneself with such thing as a multilayer scarf or even a mask. The older one gets, the more vulnerable one becomes to respiratory viral infections.