Some choices in taking or not taking the vaccine

10 February 2021 – Revised 10 Feb 2022

Introduction The problem of taking the vaccine or not taking it has to do with who you believe. The same problem has been prevalent the past year for all of us with reference to Wuhan flu, wearing masks, shutting the world down, watching players kneel, and the election. It is my opinion everything is just about half and half. Therefore, pick a side.

My research has convinced me to not take the vaccine and I have provided about an 1/8th of my papers, URLs, and notes noting I am not trying to sway anyone as we are a lot like Congress: *Our minds are made up*.

Keith and I have been working on this, Jim and I have been swapping stories, Bob has been in with some doubts about the vaccine, Ken has asked about it, and Wayne just sent me 9 pages of instructions on what to do, say, call, or find out about what is in the vaccine when you go to get the shot. The information surprised me as it does spell out the ingredients – it took 26 words. These pages also spell out some of my misgivings on the design of this particular vaccine which is the Pfizer-BioNTech.

My paper in the references "NOT taking the vaccine" goes along with the argument I like best and it is the video with Dr. Lee Merrit. She carries on from : Biologically manipulated bio-weapons to her office COVID KIT which has four ingredients listed in my pre-virus kit (also sent out last week).

Last year before Christmas it dawned on me of the critical time between coming down with the virus and getting treatment could be five or six days. All I could get from my doctors was (as I have indicated) is "Take the vaccine" – everyf'ing one of them said this – with little to nothing on what to do between Saturday midnight and Thursday noon on the way to the hospital to get on a ventilator – by myself…well, this task was finally met when I got all the meds/vits/Nebulizer all lined up and ready to go. Merrit meets the task by using five dollar historically working meds. As well see the splendid article below "HCQ treatment paper."

I now have the piece of mind over the questions about no animals were tested, few people were tested, the pharmaceutical industry buying out 'everybody,' the vaccines are not FDA approved, the vaccines are emergency experiments, and the vaccines are trials – and the industry is not responsible for any deaths…well, at least I know they need guinea pigs.

Conclusion
"Turn your TV off (except war movies) *Take your mask OFF* *Reopen your business* ***LIVE YOUR LIFE!***
Go visit with your family, have your neighborhood parties, we cannot live in a basement…No masks, NOT ANYMORE!
Notes on vaccine https://www.youtube.com/watch?v=3mPIomjWwd4&feature=youtu.be (30:11)

9 February 2021 Alex Newman on TNA with Dr. Lee Merrit, MD (Jan '21)


CDC Sleight of Hand Revealed in C(


COVID disease progression.pdf


The FDA didn't approve Pfizer's CO\


HCQ treatment paper.pdf


COVID Tests Gone Wild.pdf


Covid-19 white paper.pdf


NOT taking the vaccine - Duckduckg

There are about 30+ pages here on the issue. I do have more.
For the HCQ use: https://www.sciencedirect.com/science/article/pii/S0924857920304258
Revised 10 Feb '22 – See below for each of the .pdf files above listed in order below.

CDC Sleight of Hand Revealed in COVID-19 Death Rates

By Bill Sardi February 5, 2021

https://www.lewrockwell.com/2021/02/no_author/cdc-sleight-of-hand-revealed-in-covid-19-death-rates/

On March 24, 2020 the Centers for Disease Control fooled all Americans there were more deaths from COVID-19 coronavirus infection by changing the guidelines for determination of co-morbid conditions that contributed to death.

According to a breaking report at GreenMedInfo, this led to a 16.7-fold increase in deaths attributed to COVID-19, or ~425,000 COVID-19 related deaths on January 29, 2021 or 13 deaths over 10,000 Americans.
Using long-established guidelines, the accumulated death toll from COVID-19 would have been just 25,429 deaths on that same date, or less than 1 death per 10,000 Americans (see chart below). The entire fraud, which violates U.S. law, is described in detail in the October 12, 2020 issue of Science, Public Health Policy & The Law, and has gone unreported by the nation's news media.

		CDC GUIDELINES ISSUED MARCH 24, 2020			CDC GUIDELINES PRIOR TO MARCH 24, 2020		
SELECTED U.S./ STATES	POPULATION	ACCUMULATED COVID-19 DEATHS AS OF 1/29/2021	COVID-19 DEATH RATE PERCENT 0.01 = 1%	COVID-19 DEATH RATE PER CAPITA	COVID-19 DEATHS LESS 16.7-FOLD PER PRIOR CDC GUIDELINES*	COVID-19 DEATH RATE PERCENT 0.01 = 1%	REAL COVID-19 COVID-19 DEATH RATE PER CAPITA
U.S.	328.2million	425,000	0.0013	13.0/10,000	25,429	0.000077	0.77/10,000
STATES							
Illinois	12.67 million	20,853	0.0002	2.0/10,000	1,248	0.000019	0.19/10,000
Maine	1.34 million	558	0.0004	4.0/10,000	33	0.000024	0.24/10,000
Oregon	4.2 million	1,904	0.00045	4.5/10,000	114	0.000027	0.27/10,000
Utah	3.2 million	1,614	0.00050	5.0/10,000	97	0.000030	0.30/10,000
California	39.5 million	38,221	0.00097	9.7/10,000	2,288	0.000058	0.58/10,000
Florida	21.48million	25,672	0.0012	12.0/10,000	1,537	0.000071	0.71/10,000
Kansas	2.9 million	3,622	0.0012	12.0/10,000	217	0.000074	0.74/10,000
Texas	29.0 million	35,420	0.0012	12.0/10,000	2,120	0.000073	0.73/10,000
Tennessee	6.8 million	9,078	0.0013	13.0/10,000	543	0.000080	0.80/10,000
Mississippi	2.97 million	5,852	0.0019	19.0/10,000	350	0.00018	1.80/10,000
South Dakota	884,659	1,763	0.00199	20.0,10,000	105	0.00012	1.20/10,000
New York	19.45million	42,273	0.0020	20.0/10,000	2,531	0.00013	1.30/10,000
Sources: * Science Public Health Policy & The Law Oct, 12, 2020; Green Med Info Feb 1, 2021							

Excerpts from the report are provided below:

"Why would the CDC decide against using a system of data collection & reporting they authored, and which has been in use nationwide for 17 years without incident, in favor of an untested & unproven system exclusively for COVID-19 without discussion and peer-review? Did the CDC's decision to abandon a known and proven effective system also breach several federal laws that ensure data accuracy and integrity? Did the CDC knowingly alter rules for reporting cause of death in the presence of comorbidity exclusively for COVID-19? If so, why?
"The CDC published guidelines on March 24, 2020 that substantially altered how cause of death is recorded exclusively for COVID-19. This change was enacted apparently without public opportunity for comment or peer-review. As a result, a capricious alteration to data collection has compromised the accuracy, quality, objectivity, utility, and integrity of their published data, leading to a significant increase in COVID-19 fatalities. This decision by the CDC may have

subverted the legal oversight of the Office of Management & Budget as Congressionally authorized.

“The CDC’s rules for data collection, published data, and statistical analyses are legally required to comply with the laws established by the Information Quality Act (IQA), enacted by Congress in December 2000 as Section 515 of Public Law 106- 554, which required the Office of Management and Budget (OMB) to “provide policy and procedural guidance to Federal agencies for ensuring and maximizing the quality, objectivity, utility, and integrity of information (including statistical information) disseminates by Federal agencies.”

“We allege that the complete absence of the appropriate Federal Register records is evidence that the CDC knowingly and willingly violated the IQA & PRA. As a direct consequence of implementing the two documents below without OMB approval, there was significant inflation of COVID-19 case and fatality data.

“COVID-19 was declared a pandemic on March 11, 2020 by the World Health Organization. As such, any data gathering related to this illness must be done with the utmost transparency to ensure the public and public officials have sound data upon which to make vitally important decisions.

“Yet, the CDC failed to follow the OMB Guidelines as required by Congress and, in doing so, violated the law and also violated the public trust.

“The Council of State & Territorial Epidemiologists (CSTE) position paper paved the way for unlicensed and medically untrained contact tracers to illegally diagnose patients without any medical examination or confirmatory lab testing. In fact, they could do so without even seeing or talking to the patient in question.
“By adopting both the March 24, 2020 NVSS COVID-19 Alert No. 2 and the April 14, 2020 CSTE position paper, the CDC knowingly and willfully compromised the integrity of data they collected, published, and analyzed. We allege the CDC intentionally violated federal law with respect to integrity of information.

“Tens of thousands of Americans have died without access to potentially life-saving medications like hydroxychloroquine or nutrient therapies like intravenous Vitamin C. Couple this with the tragic reality that so many Americans.

end

COVID-19 disease progression

Written by Franz Wiesbauer, MD MPH

Edited by Shelley Jacobs, PhD

Last update - 19th Nov 2020

Infection with SARS-CoV-2 (or COVID-19) can be classified into three stages of increasing severity:[1]

1. Stage I: The early infection or viral response phase during which symptoms of upper respiratory tract infection dominate.
2. Stage II: The pulmonary phase when the patients develop full-blown pneumonia with all its associated symptoms.
3. Stage III: The hyperinflammation phase when patients develop acute respiratory distress syndrome (ARDS), sepsis, and kidney and other organ failures.

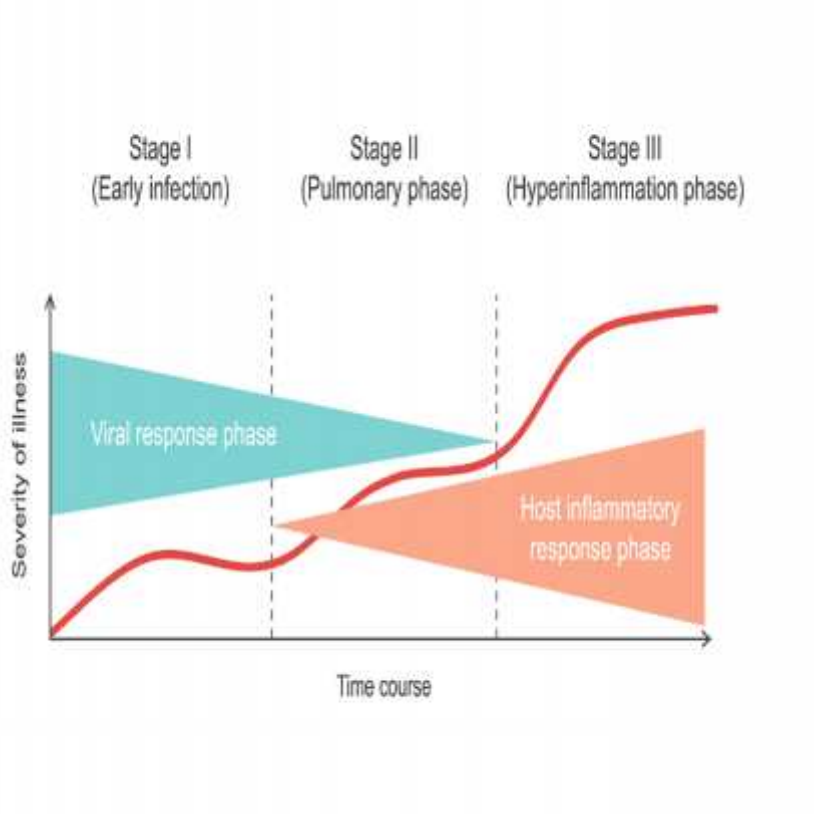


Figure 1. *Infection with SARS-CoV-2 (COVID-19) can be classified into three stages of increasing severity: early infection, pulmonary phase, and hyperinflammation phase (Adapted from Siddiqi, HK, and Mehra, MR. 2020).*

It's important to note that some patients only have milder symptoms associated with an upper respiratory infection or stage I, whereas others progress to more advanced stages.

Stage I: Early infection

Modes of transmission of COVID-19

Most of the time, the infection starts when an uninfected person inhales virus-laden droplets or aerosols into their nose and throat—47% of the time transmission comes from a presymptomatic person, through aerosol transmission or droplet inhalation, while 38% of the time it occurs from a symptomatic person through aerosol transmission or droplet inhalation. In 10% of patients, infection can also occur when someone touches a contaminated surface, then touches their face with their hands which contain the virus. And 6% of the time an asymptomatic person may be responsible for transmission, likely also through aerosol transmission or droplet inhalation.[2]

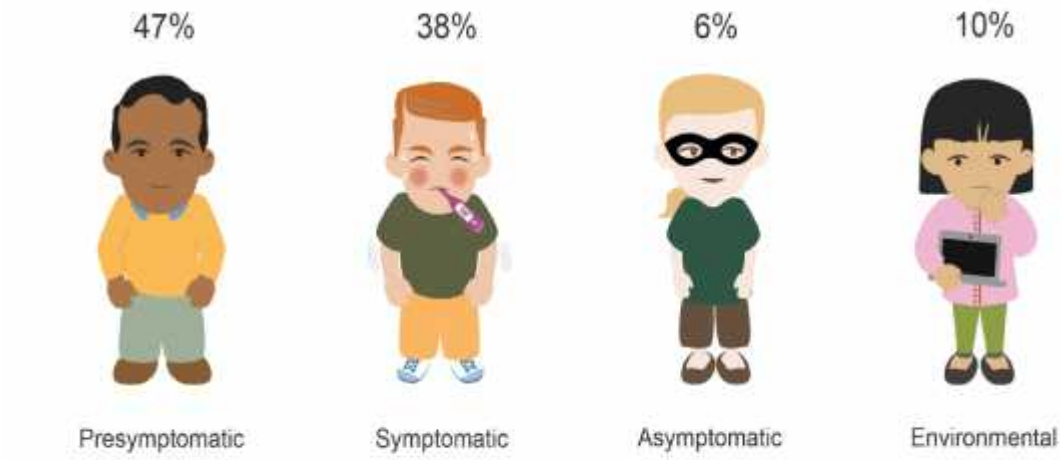


Figure 2. *The modes of transmission of COVID-19: 47% of cases are transmitted by contact with presymptomatic individuals, 38% from contact with symptomatic individuals, 6% from contact with asymptomatic individuals, and 10% are transmitted through environmental factors (Ferretti, L, Wymant, C, Kendall, M, et al. 2020).*

When are people with COVID-19 most contagious?

It seems that infectiousness is highest in the one to two days before symptoms start (coinciding with a high rate of presymptomatic transmission), and that seven days after symptom onset there is very little chance that infections will be transmitted.[3]

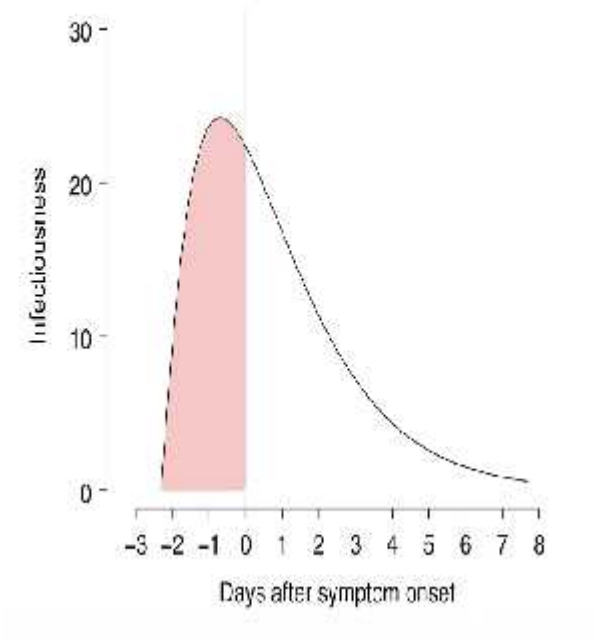
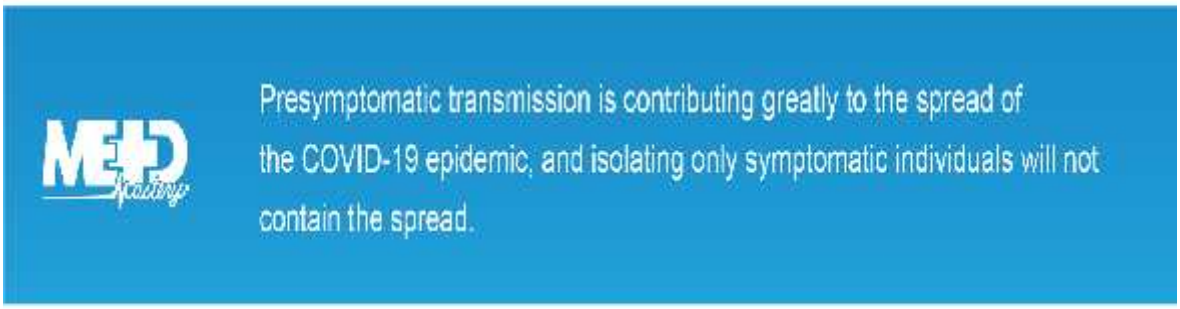


Figure 3. *The infectiousness of a person with COVID-19 is highest in the one to two days prior to symptom onset; whereas seven days after onset, there is little chance of transmission (Adapted from He, X, Lau, EHY, Wu, P, et al. 2020).*



Infection

The cells of the nose have a cell surface receptor called angiotensin-converting enzyme 2 (ACE2) ("Fig. 4a"). This receptor is also present in other organs, but cells of the nose exhibit a very high receptor density. And since the virus is inhaled through the nose, that's where it will likely attach first. Once bound to this receptor ("Fig. 4b"), the SARS-CoV-2 virus is able to enter the cells ("Fig. 4c"). And once inside, the virus uses the cell's machinery to make numerous copies of itself ("Fig. 4d–f") to invade even more cells.

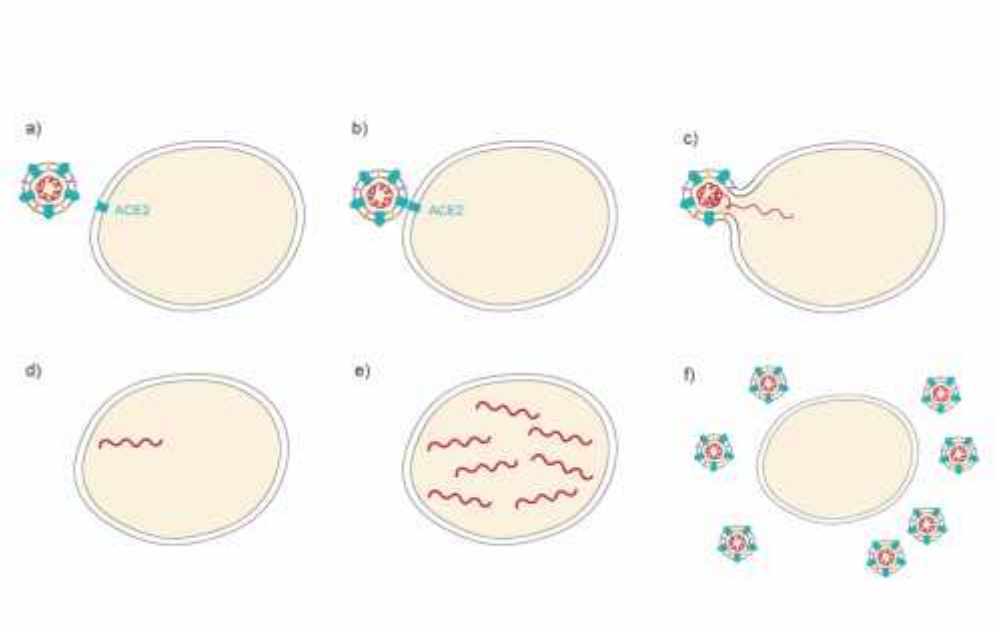


Figure 4. *Infection and replication of SARS-CoV-2 in human cells, a) virus particle in proximity to host cell with ACE2 receptor, b) virus binding to the host cell, c) insertion of viral RNA, d) viral RNA in the cell, e) viral RNA replication, f) new viral particles released.*

Watch a short animation of infection and replication of SARS-CoV-2:

Incubation

The median incubation period, the time between infection and the onset of symptoms, is 5.2 days, but it can be as long as 14 days in some cases.[3]

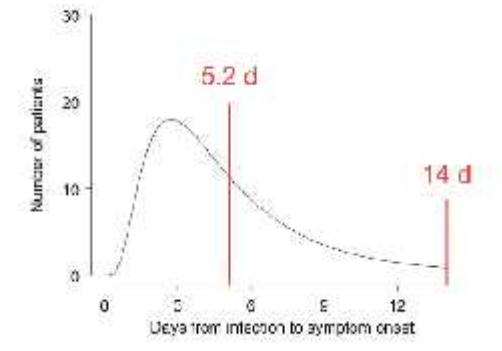


Figure 5. *The incubation period of SARS-CoV-2. The time between infection and the onset of symptoms is 5.2 days on average, but can be as long as 14 days (Adapted from He, X, Lau, EHY, Wu, P, et al. 2020).*

If a patient remains asymptomatic 14 days after exposure, the patient is unlikely to develop symptoms. This is why people have been told to self-isolate or quarantine for 14 days.

Symptom onset

As mentioned before, in most patients, the disease starts as a mild infection with upper respiratory tract symptoms. In some patients, the infection will worsen and enter the lungs and cause pneumonia by the end of the first week or the beginning of the second week.

The terminal alveoli in the lungs are also lined with cells rich in the ACE2 receptors. As the virus enters these alveolar cells, pneumonia develops. White blood cells release chemokines in order to kill virus-infected cells.

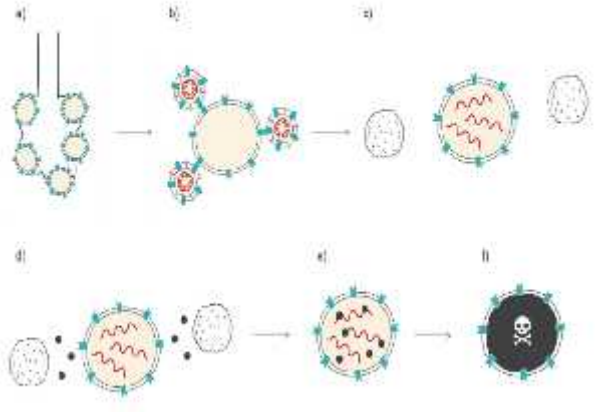


Figure 6. *Immune response to SARS-CoV-2 in the alveoli, a) host alveolar cells with ACE2 receptor, b) virus particles binding to ACE2 receptor on the host cells, c) immune cells approaching the virus-infected cell, d–f) immune cells releasing chemokines and killing the virus-infected cell.*

Check out a short animation of how the immune cells respond to SARS-CoV-2 in the alveoli:

Pus, a collection of fluid and dead cells, is left behind and interferes with the lungs' ability to transfer oxygen to the blood and CO_2 out of it. By this point, the patient will likely have a worsening cough, fever, and rapid, shallow respiration. It is at this stage where most patients with COVID-19 would need to be hospitalized for close observation, management, and possibly oxygen therapy.

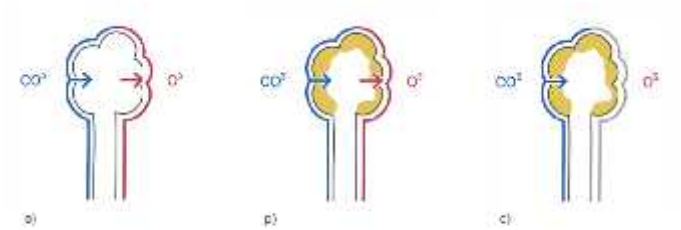


Figure 7. *The pus causes a barrier to gas exchange in the alveoli, reducing the efficiency of respiration, a) normal gas exchange in the alveoli, b) accumulation of pus in the alveoli, c) reduction of oxygen transfer in the alveoli.*

Stage II: Pulmonary phase

This pulmonary phase is divided into two distinct parts. Stage IIA is the pneumonia patient without hypoxia and Stage IIB is the pneumonia patient with hypoxia who will likely require hospitalization and oxygen supplementation.

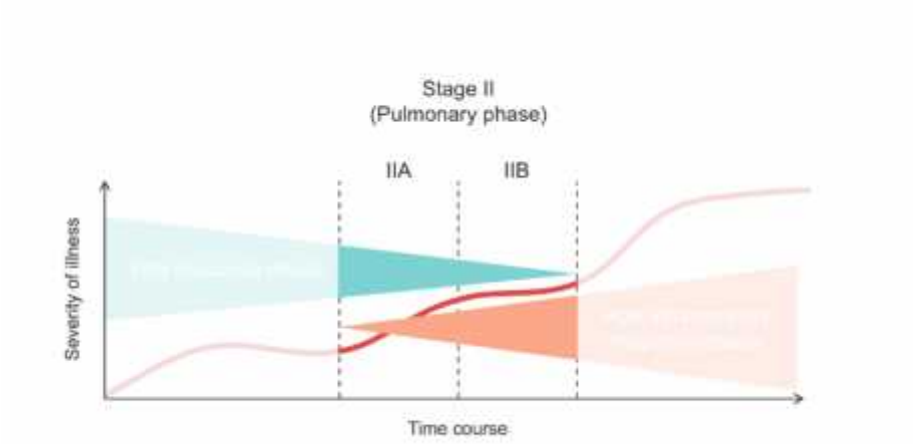


Figure 8. *Stage II of a COVID-19 infection, the pulmonary phase, is divided into two distinct parts: Stage IIA, the pneumonia patient without hypoxia, and Stage IIB, is the pneumonia patient with hypoxia who will likely require hospitalization and oxygen supplementation (Adapted from Siddiqi, HK, and Mehra, MR. 2020).*

Studies in China and the US suggest that most patients, on average, are admitted to the hospital about one week after symptoms begin. Patients in the pulmonary phase of the disease can quickly progress to the hyperinflammatory phase where the infection runs wild.

Stage III: Hyperinflammation phase

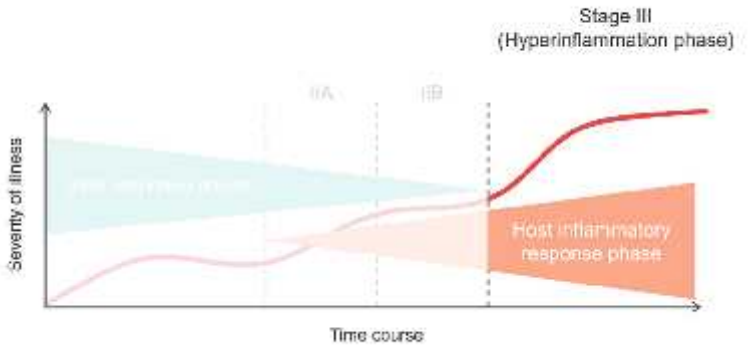
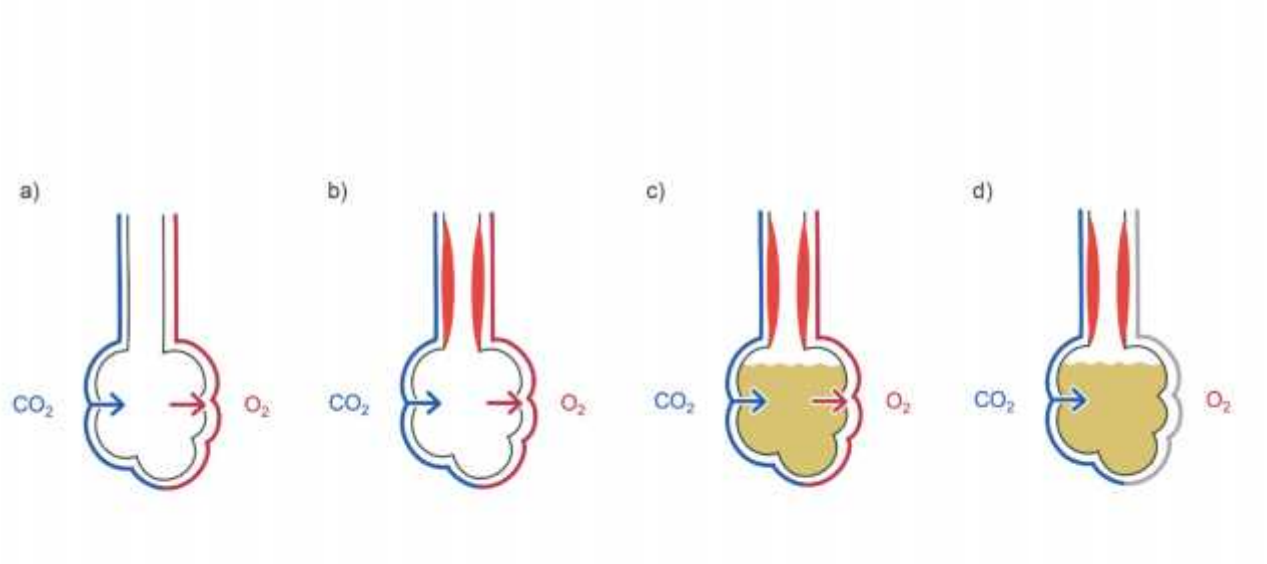


Figure 9. *Stage III of a COVID-19 infection, the hyperinflammation phase, in which the infection runs wild and patients often deteriorate suddenly (Adapted from Siddiqi, HK, and Mehra, MR. 2020).*

These patients often deteriorate suddenly, usually developing ARDS. Acute respiratory distress syndrome involves inflammation and fluid build-up in the lungs, which prevents oxygen transfer from the air to the



blood.

Figure 10. *Fluid build-up in the alveoli and inflammation prevents oxygen transfer in patients with acute respiratory distress syndrome (ARDS), a) normal alveoli with efficient gas exchange, b) inflammation of the airway as seen in ARDS, c) inflammation of the airway and fluid build-up in the alveoli as seen in ARDS, d) decreased oxygen transfer due to fluid accumulation and inflammation of the airway.*

Blood oxygen levels drop rapidly and the patient struggles harder to breathe. Patients with ARDS usually require mechanical ventilation in the intensive care unit (ICU). On average, patients are intubated between 14.5 days after symptom onset. Depending on the country, and the ICU setting, approximately half of ARDS patients will recover, and half will die.

How long does it take COVID-19 patients to recover overall?

One study of hospitalized patients in Wuhan, China showed that the median duration from symptom onset to discharge from the hospital was 22 days, and the median time from symptom onset to death was 18.5 days.[4]

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5.

Recommended reading

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SCIENCE Los Angeles Times

The FDA didn't 'approve' Pfizer's COVID-19 vaccine. Here's why

A health worker prepares a dose of the COVID-19 vaccine from Pfizer and BioNTech. The Food and Drug Administration authorized its emergency use but didn't formally approve it.
(Associated Press)

By KAREN KAPLANSCIENCE AND MEDICINE EDITOR

DEC. 12, 2020 5 AM PT
https://www.latimes.com/science/story/2020-12-12/why-fda-didnt-approve-pfizer-covid-19-vaccine-eua

A lot of things are different when you're in the midst of a global pandemic. A case in point: How federal regulators scrutinize and authorize new vaccines.
The U.S. Food and Drug Administration ushered in a new phase of the fight against COVID-19 on Friday by giving its blessing to a vaccine made by Pfizer Inc. and BioNTech. It's the first such vaccine to get a green light from the FDA, and immunizations will begin in a matter of days.
There are plenty of reasons why it passed muster. Clinical trial data indicate that:
• It was 95% effective at preventing cases of COVID-19 in both Latinos and non-Latinos.
• It was 100% effective in Black people.
• It was 94% effective in people who were at least 56 years old. (The older you get, the greater the risk of a serious case of COVID-19.)
• It was 95% effective in those who had at least one medical condition that made them more likely to develop a serious case of COVID-19.
• It was 96% effective for people who were obese, another condition that makes people more vulnerable to COVID-19.
Yet none of this was enough for the vaccine to win official FDA approval. What it got instead was a more limited emergency use authorization.
Why?
Blame it on the pandemic.

During a public health emergency, it's imperative to develop new medicines and vaccines as quickly as possible. But even when speed is of the essence, the FDA still takes the time to be sure patients aren't subjected to untested therapies that do more harm than good.
So the agency uses an alternative evaluation process that's designed to vet things more quickly than the usual FDA approval regimen. If a drug or vaccine passes muster, it's granted an emergency use authorization, or EUA.
An EUA can be used on a brand-new medical product or on an existing one that has already been approved for another purpose. They're not limited to vaccines — under the right circumstances, an EUA can be granted to anything used to "diagnose, treat, or prevent serious or life-threatening diseases or conditions," the FDA explains.

What are those conditions?

For starters, the country needs to be in an official, specific kind of public health emergency. Secretary of Health and Human Services Alex Azar declared that the coronavirus triggered a nationwide public health emergency that began on Jan. 27, but that declaration doesn't count because it was issued under the Public Health Service Act.

The legislation that matters is the Federal Food, Drug and Cosmetic Act, a law passed in 1938 that empowered the FDA to regulate medications, among other things. Azar issued a public health emergency under this law that was effective as of Feb. 4.

Another important condition for issuing an EUA is that "there are no adequate, approved, and available alternatives" to the product being authorized. That's certainly the case with vaccines against COVID-19, a disease that only came on the scene at the end of 2019.

An experimental vaccine being considered for emergency use authorization still must be tested in multiple rounds of clinical trials. In Phase 1, the candidate vaccine is given to a small number of healthy people at gradually increasing doses to make sure it's safe and well-tolerated. A Phase 1 trial might provide some preliminary information about ideal dosages as well.

Next comes a Phase 2 study, which involves more volunteers testing various doses. At this point, the study will branch out to include people with a variety of health conditions, not just those who are in great shape. This is also when a vaccine is first compared head-to-head against a placebo.

If no safety issues crop up, things proceed to Phase 3. Thousands of study volunteers from a variety of backgrounds are randomly assigned to receive either the vaccine or the placebo. This type of study helps

researchers measure the effect of the vaccine. For instance, in Phase 3 trials of the Pfizer-BioNTech vaccine, eight of the 17,411 people who were given the vaccine wound up becoming sick with COVID-19, compared with 162 of the 17,511 who got the placebo. Using that and other data, researchers determined that the vaccine was 95% effective at preventing COVID-19.

To be considered for an EAU, a Phase 3 vaccine trial should include "well over 3,000" participants, and at least half of them should be tracked for at least two months after receiving their final dose.

Once sufficient data are in hand, the FDA can decide whether emergency use authorization is warranted. Doctors and scientists on the agency's staff pore over the study results. So do the independent scientists and health experts on the agency's Vaccines and Related Biological Products Advisory Committee.
In the case of a vaccine, authorization can be granted if "the known and potential benefits outweigh the known and potential risks," the FDA says.

The agency also assesses the company's ability to consistently produce high-quality doses of its vaccine. Granting emergency use authorization isn't the end of the story. Once an authorized vaccine goes out to the public, its manufacturer must keep track of any serious side effects that befall those who take it, especially adverse events that result in hospitalization or even death.

The FDA, the Centers for Disease Control and Prevention and other government agencies will do their own safety monitoring as well. If the FDA ever determines that the benefits of the vaccine no longer outweigh the harms, the emergency use authorization can be revoked.

An EUA can last only as long as a public health emergency is in effect. But scientists anticipate that the coronavirus will continue to circulate in humans even after the COVID-19 pandemic ends. In that case, vaccine makers that want to keep their products on the market will need to have regular FDA approval — and to get it, they'll need to keep their Phase 3 clinical trials going.

And that's probably what they'll do. The FDA said it expects vaccine makers who receive emergency use authorizations to "continue to collect placebo-controlled data in any ongoing trials for as long as feasible" so they can apply for regular approval.

International Journal of Antimicrobial Agents

journal homepage: www.elsevier.com/locate/ijantimicag

COVID-19 outpatients: early risk-stratified treatment with zinc plus low-dose hydroxychloroquine and azithromycin: a retrospective case series study

https://www.sciencedirect.com/science/article/pii/S0924857920304258 (Read this on line.)

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a r t i c l e i n f o
Keywords:
SARS-CoV-2
COVID-19
Outpatients
Zinc
Hydroxychloroquine
Azithromycin

a b s t r a c t
The aim of this study was to describe the outcomes of patients with coronavirus disease 2019 (COVID-19) in the outpatient setting after early treatment with zinc, low-dose hydroxychloroquine and azithromycin (triple therapy) dependent on risk stratification. This was a retrospective case series study in the general practice setting. A total of 141 COVID-19 patients with laboratory-confirmed severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in the year 2020 were included. The main outcome measures were risk-stratified treatment decision and rates of hospitalisation and all-cause death. A median of 4 days [interquartile range (IQR) 3–6 days; available for n = 66/141 patients] after the onset of symptoms, 141 patients (median age 58 years, IQR 40–67 years; 73.0% male) received a prescription for triple therapy for 5 days. Independent public reference data from 377 confirmed COVID-19 patients in the same community were used as untreated controls. Of 141 treated patients, 4 (2.8%) were hospitalised, which was significantly fewer (P < 0.001) compared with 58 (15.4%) of 377 untreated patients [odds ratio (OR) = 0.16, 95% confidence interval (CI) 0.06–0.5]. One patient (0.7%) in the treatment group died versus 13 patients (3.4%) in the untreated group (OR = 0.2, 95% CI 0.03–1.5; P = 0.12). No cardiac side effects were observed. Risk stratification-based treatment of COVID-19 outpatients as early as possible after symptom onset using triple therapy, including the combination of zinc with low-dose hydroxychloroquine, was associated with significantly fewer hospitalisations.

1. Introduction

In December 2019, the novel severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) started as an outbreak in Wuhan,

China. This coronavirus has spread rapidly as a pandemic around the world [1], causing coronavirus disease 19 (COVID-19) pneumonia, acute respiratory distress syndrome (ARDS), cardiac injury, liver and renal injury, thrombosis and death [2].

As of June 2020, the diagnosis and treatment of COVID-19 have been almost exclusively studied from an inpatient perspective, including intensive care with mechanical ventilation. Only one study has described the characteristics and key health outcomes of COVID-19 diagnosed patients in an outpatient setting [3]. This

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is surprising as primary care physicians often see COVID-19 patients first. Thus, they could play a critical role in early diagnosis, treatment and management of disease progression and virus spread. This assumption is supported by the established principle in medicine that speed of eradication is linked to the outcome of life-threatening infections [4].

The early clinical phase of COVID-19 has not been the focus of much research so far, even though timing of antiviral treatment seems to be critical [5]. The optimal window for therapeutic intervention would seem to be before the infection spreads from the upper to lower respiratory tract and before severe inflammatory reaction ensues [6]. Therefore, diagnosis and treatment of COVID-19 outpatients as early as possible, even based on clinical diagnosis only, may have been an underestimated first step to slow down or even stop the pandemic more effectively. Based on clinical application principles of antiviral therapies, as demonstrated in the case

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R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214

of influenza A [7], antiviral treatments should be used early in the course of infection.

Due to the lack of a vaccine or SARS-CoV-2 specific therapies, the proposed use of repurposed antiviral drugs remains a valid practical consideration [8]. One of the most controversial drugs during the current SARS-CoV-2 pandemic is the well-known oral antimalarial drug hydroxychloroquine (HCQ), routinely used in the treatment of autoimmune diseases such as rheumatoid arthritis

and systemic lupus erythematosus (SLE) [9,10]. HCQ is currently listed as an essential medication for SLE by the World Health Organization (WHO) [11]. With more than 5.6 million prescriptions in the USA, HCQ was the 128th most commonly prescribed medication in 2017 [12]. In the meantime, the first observational studies concluding beneficial therapeutic effects of HCQ as monotherapy or in combination with the antibiotic azithromycin were reported just a few weeks after the start of the SARS-CoV-2 outbreak [13]. All studies that used HCQ with rather contradictory results were in hospitalised and often sicker patients [13–16], and one publication was recently withdrawn [17,18]. As of June 2020, no studies of COVID-19 outpatients treated with HCQ at an early stage of the disease have been reported.

The antiviral effects of HCQ are well documented [19]. It is also known that chloroquine, and probably HCQ, have zinc ionophore characteristics, increasing intracellular zinc concentrations [20]. Zinc itself is able to inhibit coronavirus RNA-dependent RNA polymerase (RdRp) activity [21]. It has been hypothesised that zinc may enhance the efficacy of HCQ in treating COVID-19 patients [22]. The first clinical trial results confirming this hypothesis were recently published as a preprint [23]. Nevertheless, many studies with HCQ as monotherapy or in combination with the antibiotic azithromycin have been inconclusive so far [13–16]. In all of these studies, HCQ was used later than 5 days after the onset of symptoms when hospitalised patients most likely had already progressed to stage II or III of the disease [6]. Regardless of the established antiviral effects of zinc and that many COVID-19 patients are prone to zinc deficiency, dependent on co-morbidities and drug treatments [22], none of these studies were designed to include zinc supplementation as combination treatment.

This first retrospective case series study of COVID-19 outpatients was done to show whether (i) a simple-to-perform outpatient risk stratification might allow for a rapid treatment decision shortly after onset of symptoms and (ii) whether the 5-day triple therapy with zinc, low-dose HCQ and azithromycin might result in fewer hospitalisations and fatalities compared with relevant public reference data of untreated patients.

2. Methods

2.1. Setting

This retrospective case series study analysed data from COVID-19 outpatients with confirmed SARS-CoV-2 infection treated in a community in New York State, USA, between 18 March 2020 and 14 May 2020. The outcome of patients treated with a specific triple therapy was compared with public reference data of patients in the same community who were not treated with this therapy.

2.2. Confirmation of COVID-19 diagnosis
The COVID-19 diagnosis was confirmed if patients tested positive for SARS-CoV-2 by PCR of nasal or pharyngeal swab specimens (majority of tests by Roche, Basel, Switzerland; 99.1% sensitivity and 99.7% specificity; other tests used with lower frequency included: DiaSorin: 500 copies/mL; Thermo Fisher: 10 genomic copy equivalents/reaction; Seegene: 1250 copies/mL; Hologic: TCID50/mL: 1 × 10–2) or retrospectively by IgG detection tests [DiaSorin: sensitivity 97.6% (≥15 days after diagnosis), specificity 99.3%; Diazyme: sensitivity 91.2%, specificity 97.3%]. Only patients who had a record of a positive test result were included in the analysis. The PCR assays were authorised by the US Food and Drug Administration (FDA) without clinical sensitivity/specificity data owing to the urgent nature of the pandemic. Only one positive test was necessary for the patient to be included in the retrospective analysis.

2.3. Patients
Sequentially consecutive COVID-19 outpatients aged >18 years at diagnosis were included in the analysis as the treatment group. All patients were White. Patients received a prescription for triple therapy only if they met one of the following risk stratification requirements during a medical office-based or telehealth consultation: Group A, age >60 years, with or without clinical symptoms; Group B, age ≤60 years and shortness of breath (SOB); or Group C, age ≤60 years, clinically symptomatic and with at least one of the following co-morbidities: hypertension, hyperlipidaemia, diabetes mellitus, obesity [body mass index (BMI) ≥ 30 kg/m2], cardiovascular disease, heart failure, history of stroke, history of deep vein thrombosis or pulmonary embolism, asthma, chronic obstructive pulmonary disease (COPD), other lung disease, kidney disease, liver disease, autoimmune disease or history of cancer. Pregnant women, if any, were also included in this group.
Laboratory-confirmed COVID-19 patients from the same community who were not treated with the described triple therapy and their related outcome data represented the untreated control group, which comprised both low-risk and high-risk patients (public reference data).

2.4. Procedure and treatment
Data for treated patients were collected from electronic health records in the year 2020. Demographics, as reported by the patient, and current medical history of hypertension, hyperlipidaemia, diabetes mellitus, obesity (BMI ≥ 30 kg/m2), cardiovascular disease, heart failure, stroke, asthma, COPD, other lung disease, kidney disease, liver disease, autoimmune disease, history of cancer, thyroid disease psychiatric disorder or pregnancy were collected.
The presence of the following clinical symptoms of treated patients was documented: cough/dry cough; fever; SOB; changes to or no smell or taste; sore throat; headache; runny nose/clear rhi-

norrhoea; sinus congestion; diarrhoea/vomiting; cold symptoms; feeling sick; weakness; and low back pain. If reported, the number of days since onset of symptoms was documented.
The following vital signs, if available, were collected and documented: heart rate (beats/min), respiratory rate (breaths/min), systolic and diastolic blood pressure (mmHg), body temperature (°C), oxygen saturation measured by pulse oximetry (O2 %), body weight (kg) and/or BMI.

The main co-medications were characterised based on primary care prescriptions active at the time of diagnosis, documented as categorical variables, included beta-blockers, angiotensin-converting enzyme inhibitors, angiotensin 2 antagonists, calcium channel blockers, hydrochlorothiazide, statins, bronchodilators, antidiabetics and insulin.

Only diagnosed COVID-19 patients who met the defined risk stratification requirements of group A, B or C received a prescription for the following triple therapy for 5 consecutive days in addition to standard supportive care: zinc sulfate (220 mg capsule once daily, containing 50 mg elemental zinc); HCQ (200 mg twice daily); and azithromycin (500 mg once daily). No loading dose was used. Patients who did not meet the risk stratification requirements received standard of care to treat common upper respiratory

[7][8]910[11][12][13][13–16]1718[19][20][21][22][23][13–16][6][22]

tract infections. Patients were not treated with HCQ if they had known contraindications, including QT prolongation, retinopathy or glucose-6-phosphate dehydrogenase deficiency. As usual and following best practice, patients were informed about possible drug-related side effects. Reported events, if any, were documented as required.

Selection of the used zinc supplement and of drugs, dosages and the combination thereof were based on treatment guidelines, positive reports from other countries such as South Korea, emerging first clinical evidence, and based on the discretion of the treating physicians.

2.5. Outcomes
Two outcomes were studied: COVID-19 related hospital admission and all-cause death during time of follow-up of ≥28 days in the treatment group and in the untreated control group (public reference). The outcome of COVID-19 patients in the untreated control group was reported by the responsible health department.

2.6. Statistical analyses

Only patients in the treatment group who met the defined risk stratification requirements and who received at least one prescription for HCQ, with or without zinc, for 5 days were included in the retrospective analysis and were categorised accordingly. If the patient's electronic health record did not include information on a clinical characteristic, it was assumed that the characteristic was not present. In the group of the public reference data, only confirmed COVID-19 patients who were not treated in the respective general practice with triple therapy were included in the analysis. For this untreated control group, only outcome data for hospitalisation and all-cause death were available and used for the statistical comparison with the treatment group.

No sample size calculations were performed. Descriptive statistics are presented as median and interquartile range (IQR) for continuous variables and as frequencies (%) for categorical variables. For comparison with the results of other studies, the mean and standard deviation were calculated as needed. Normality of distribution for continuous variables was assessed by the Shapiro–Wilk test. A two-tailed Student's t-test was used for parametric analysis, and a Wilcoxon signed-rank test was used for non-parametric data analysis. For calculation of correlation, the point-biserial correlation coefficient was applied if one variable was dichotomous. Associations between two categorical variables were calculated with the $\chi 2$ test. The odds ratio (OR) was calculated for comparison of the outcome of the treatment group with the untreated control group. An α value of 0.05 was considered as a significance level. Data were analysed using Microsoft Excel for Microsoft 365 MSO (32-bit), the Excel add-on Real Statistics, SigmaStat 4 and Sigma Plot 14.0.

2.7. Study approval

The study was approved by the Western Institutional Review Board and was exempt under 45 CFR § 46.104(d)(4). Ref. number: D4-Exemption-Zelenko (06-16-2020). The analysis was conducted with de-identified patient data, according to the USA Health Insurance Portability and Accountability Act (HIPAA), Safe Harbor. For that reason, exact dates and locations are not mentioned in this study.

3. Results

3.1. Patients

In accordance with available public reference data, 712 confirmed SARS-CoV-2 PCR-positive COVID-19 patients were reported for the respective community at the defined time point of the analysis. Of these 712 patients, 335 presented as outpatients at a general practice and 127 were treated with the triple combination therapy. Of these 127 patients, 104 met the risk stratification cri-

teria and were included in the analysis (Table 1). Of the 335 patients, 208 did not meet the defined risk stratification criteria and were treated with standard of care and recovered at home. The SARS-CoV-2 infection of 37 additional patients who were clinically diagnosed with COVID-19 who met the risk stratification criteria and who were also treated with triple therapy was later confirmed by IgG tests (Table 1). These patients were included additionally in the analysis resulting in a total number of 141 patients, all with a confirmed SARS-CoV-2 infection by PCR or IgG tests. None of these patients were lost to follow-up for the defined outcome. The outcome of the remaining 377 positively tested but not treated COVID-19 patients, e.g. from other practices of the community, served as public reference (Fig. 1). Analysis of the 141 patients in the treatment group showed that all of these patients (100%) received a prescription of HCQ, 136 (96.5%) of zinc sulfate and 133 (94.3%) of azithromycin, while 1 patient (0.7%) received doxycycline instead. Instead of triple therapy, 1 patient (0.7%) in the treatment group received HCQ only, 7 patients (5.0%) received HCQ and zinc, and 4 patients (2.8%) received HCQ and azithromycin.

3.2. Baseline characteristics of the patients

Table 2 shows the baseline demographics and clinical characteristics of all 141 patients in the treatment group and for the risk stratification groups A, B and C. Of the 141 patients, 69 (48.9%) belonged to group A, 48 (34.0%) to group B and 24 (17.0%) to group C. The age ranged from 18–80 years and the median age was 58 years (IQR 40–67 years). The median age of patients in groups A, B and C was 67, 39 and 45 years, respectively. A total of 103 patients (73.0%) were male with a male-to-female ratio of 2.71. The most common co-morbidities included hypertension (28%), obesity (28%), hyperlipidaemia (23%) and diabetes mellitus (18%), whilst the least common co-morbidities were liver disease (2%), heart failure (1%) and stroke (1%). One patient (0.7%) was pregnant at initiation of treatment. There was a positive and significant correlation between age and hypertension ($r = 0.3309$, $P = 0.001$), hyperlipidaemia ($r = 0.26306$, $P < 0.001$) and cardiovascular disease ($r = 0.16757$, $P < 0.05$), whilst asthma was negatively correlated with age ($r = –0.30867$, $P < 0.001$).

The median time between onset of clinical symptoms and medical consultation was 4 days (IQR 3–6 days; available for 66/141 patients; mean 4.8 ± 2.7 days) (Table 3). There was no significant correlation between age and days from onset of clinical symptoms to consultation ($P > 0.05$). Days from onset of symptoms to consultation were not significantly different between the groups ($P > 0.05$).

The most common clinical symptoms included cough (87.2%), fever (77.3%), SOB (46.1%) and changes to or no smell or taste

(30%), whilst the least common clinical symptoms were sinus congestion (16%), diarrhoea/vomiting (5%) and low back pain (3%). Table 4 shows the symptoms of all patients and stratified by groups A, B and C. There was a significant negative correlation between age and changes to smell or taste ($r = –0.43$, $P < 0.001$). No patient had a clinical diagnosis of pneumonia.
Table 5 shows the vital signs, if available, for all patients. Many patients consulted the general practice during the COVID-19 crisis
3

Fig.1
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
Table 1
COVID-19 diagnostics by PCR and IgG tests of patients in the treatment group

COVID-19 diagnostic [n (%)]	Risk-stratified group			All patients (N = 141)
	Group A (N = 69)	Group B (N = 48)	Group C (N = 24)	
SARS-CoV-2 PCR test	51 (74)	39 (81)	14 (58)	104 (74)
SARS-CoV-2 IgG test	18 (26)	9 (19)	10 (42)	37 (26)

COVID-19, coronavirus disease 2019; SARS-CoV-2, severe acute respiratory syndrome coronavirus 2.
Table 2
Baseline demographic and clinical characteristics of patients in the treatment group

Characteristic	Risk-stratified group			All patients (N = 141)
	Group A (N = 69)	Group B (N = 48)	Group C (N = 24)	
Age (years) [median (IQR)]	67 (64–69)	39 (24–47)	45 (36–50)	58 (40–67)
Male sex [n (%)]	46 (67)	40 (83)	17 (71)	103 (73)
Co-morbidities/coexisting conditions [n (%)]				
Any condition	44 (64)	31 (65)	24 (100)	99 (70)
Hypertension	27 (39)	4 (8)	8 (33)	39 (28)
Hyperlipidaemia	21 (30)	7 (15)	5 (21)	33 (23)
Diabetes mellitus	16 (23)	4 (8)	5 (21)	25 (18)
Obesitya	20 (29)	10 (21)	10 (42)	40 (28)
Cardiovascular disease	9 (13)	1 (2)	3 (13)	13 (9)
Heart failure	2 (3)	0 (0)	0 (0)	2 (1)
Stroke	1 (2)	0 (0)	0 (0)	1 (1)
Asthma	2 (3)	9 (19)	2 (8)	13 (9)
COPD	0 (0)	0 (0)	0 (0)	0 (0)
Other lung disease	6 (9)	5 (10)	4 (17)	15 (11)
Kidney disease	1 (2)	3 (6)	2 (8)	6 (4)
Liver disease	1 (2)	2 (4)	0 (0)	3 (2)
Autoimmune disease	2 (3)	4 (8)	4 (17)	10 (7)
History of cancer	6 (9)	2 (4)	1 (4)	9 (6)
Thyroid disease	7 (10)	4 (8)	2(8)	13 (9)
Psychiatric disorder	7 (10)	4 (8)	5 (21)	16 (11)
Pregnancy	–	–	1 (4)	1 (1)

IQR, interquartile range; COPD, chronic obstructive pulmonary disease.
a Body mass index (BMI) ≥30 kg/m2 .
Table 3
Patients with reported days since onset of symptoms in the treatment group

Characteristic Risk-stratified group All patients
(N = 141)
Group A (N = 69) Group B (N = 48) Group C (N = 24)
Patients with reported days [n (%)] 32 (46) 25 (48) 9 (38) 66 (47)
Days since onset of symptoms [median (IQR)] 4 (3–6) 3 (3–6.5) 4 (3–5.5) 4 (3–6)
IQR, interquartile range.
Table 4
COVID-19 diagnostics and baseline reported clinical symptoms of patients in the treatment group
Clinical symptom [n (%)] Risk-stratified group All patients
(N = 141)
Group (N = 69) Group B (N = 48) Group C (N = 24)
Cough/dry cough 60 (87) 39 (81) 24 (100) 123 (87)
Fever 53 (77) 38 (79) 18 (75) 109 (77)
Shortness of breath 17 (25) 48 (100) 0 (0) 65 (46)
Changes to or no smell or taste 21 (30) 19 (40) 2 (8) 42 (30)
Sore throat 19 (28) 8 (17) 7 (29) 34 (24)
Headache 19 (28) 6 (13) 7 (29) 32 (23)
Runny nose/clear rhinorrhoea 16 (23) 8 (17) 4 (17) 28 (20)
Sinus congestion 10 (15) 9 (19) 4 (17) 23 (16)
Diarrhoea/vomiting 1 (2) 5 (10) 1 (4) 7 (5)
Cold symptoms 31 (45) 16 (33) 12 (50) 59 (42)
Feels sick 40 (58) 38 (79) 17 (71) 95 (67)
Weakness 44 (64) 22 (46) 11 (46) 77 (55)
Low back pain 3 (4) 0 (0) 1 (4) 4 (3)
COVID-19, coronavirus disease 2019.
4
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
Fig. 1. Study population. N = 141 COVID-19 patients, all with a laboratory-confirmed SARS-CoV-2 infection, were included in the analysis as the treated group. N = 377
positively tested COVID-19 patients of the public reference were included in the analysis as the untreated group. COVID-19, coronavirus disease 2019; SARS-CoV-2, severe
acute respiratory syndrome coronavirus 2.
5
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
Table 5
Physical examination: vital signs of patients in the treatment group
Parameter Median (IQR) Patients with available
parameters [n (%) of
N = 141]
Heart rate (beats/min) 86 (80–94) 89 (63)
Respiratory rate (breaths/min) 16 (15–18) 43 (31)
Systolic blood pressure (mmHg) 126 (120–139) 66 (47)
Diastolic blood pressure (mmHg) 80 (74–85.5) 66 (47)
Body temperature (°C) 37.2 (37–37.8) 79 (56)
Pulse oximetry (O2 %) 97 (96–98) 85 (60)
Body weight (kg) 88 (72.6–98.4) 43 (31)
BMI (kg/m2) 32.2 (28.5–36.3) 30 (21)
IQR, interquartile range; BMI, body mass index.
Table 6

Co-medications of patients in the treatment group
Drug class Patients [n (%) of N = 141]
Beta-blockers 17 (12)
Angiotensin-converting enzyme inhibitors 8 (6)
Angiotensin 2 antagonists 13 (9)
Calcium channel blockers 8 (6)
Hydrochlorothiazide 6 (4)
Statins 28 (20)
Bronchodilators 10 (7)
Antidiabetics 11 (8)
Insulin 26 (18)
Oral corticosteroids 13 (9)
Antibiotics 3 (2)
via telehealth so vital signs were not available for all of these patients. The highest proportion of patients had available measurements for heart rate (63%) and pulse oximetry (60%). Vital signs were not significantly different between risk stratification groups ($P > 0.05$) except for systolic blood pressure of groups A and B ($P < 0.05$).

Table 6 summarises the most important co-medications. Of the patients, 16% were taking angiotensin-converting enzyme inhibitors, angiotensin 2 antagonists, hydrochlorothiazide or a combination thereof. The most common long-term therapies at the time of COVID-19 diagnosis were statins (20%), beta-blockers (12%) and insulin (18%). A few patients had chronic prescriptions for oral corticosteroids (9%) for co-morbidities such as asthma or autoimmune diseases, and 3 patients (2.1%) received an additional antibiotic (levofloxacin) because of superinfections.

3.3. Hospitalisations and all-cause death

In the treatment group, 4 (2.8%) of 141 patients were hospitalised, which was significantly fewer than the 58 (15.4%) of 377 patients in the untreated group (Fig. 2) [OR = 0.16, 95% confidence interval (CI) 0.06–0.5; $P < 0.001$] (Table 7; Fig. 3). Therefore, the odds of hospitalisation of treated patients was 84% less than in the untreated patients. All hospitalised patients were male, with one in his twenties, two in their forties and one in his seventies. Three (75%) of the four hospitalised patients belonged to risk stratification group B and one (25%) to group A. All patients (100%) reported SOB at the time of consultation. The median time from onset of symptoms to consultation was 4 days. In the treatment group, one patient had to stay only 1 day in hospital, two other patients were discharged as cured and one patient died (see below). No patient was on a ventilator.

Of the 141 patients, 1 (0.7%) in treatment group A died after being hospitalised. This patient had a history of cancer and only

took one daily dose of the triple therapy before hospital admission.
More patients (13/377; 3.4%) died in the untreated group (Fig. 4)
Fig. 2. Hospitalisation. Treatment with triple therapy of zinc, low-dose hydroxychloroquine and azithromycin was associated with significantly fewer hospitalisations compared with untreated patients of the public reference data. χ 2 (1, N = 518) = 14.17; $*$ P < 0.001.
Fig. 3. All-cause deaths. Treatment with triple therapy of zinc, low-dose hydroxychloroquine and azithromycin was associated with numerically fewer all-cause deaths compared with untreated patients of the public reference data. n.s., not significant. χ 2 (1, N = 518) = 1.98; P = 0.12.
6
Fig.2Fig.3Fig.4
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
Table 7
Clinical outcomes in the treated patient group versus the untreated patient group
Outcome Treated group [n (%) of N = 141]
Untreated group [n (%) of N = 377]
OR (95% CI) P-value
Hospitalisation 4 (2.8) 58 (15.4) 0.16 (0.06–0.5) <0.001
All-cause death 1 (0.71) 13 (3.5) 0.2 (0.03–1.5) 0.12
OR, odds ratio; CI, confidence interval.
Fig. 4. Odds ratios (ORs). The odds of hospitalisation in the treated patient group was 84% less than in the untreated patient group and was statistically significant (P < 0.001). The odds of all-cause death in the treated patient group was 80% less than in the untreated patient group but did not reach statistical significance (P = 0.12).
COVID-19, coronavirus disease 2019; CI, confidence interval.
Table 8
Summary of adverse events in the treatment group
Event Patients [n (%) of N = 141]
Any adverse event 67 (48)
Weakness 30 (21)
Nausea 20 (14)
Diarrhoea 15 (11)
Rash 2 (1)
(OR = 0.2, 95% CI 0.03–1.5) (Table 7; Fig. 3). Although the odds of all-cause death of treated patients was 80% less than in the untreated group, this difference did not reach statistical significance (P = 0.12).
All patients in the treatment group with the clinical outcome of hospitalisation or all-cause death received a prescription for the complete triple therapy including zinc, low-dose HCQ and azithromycin.
The outcome of the three different risk-stratified groups (A, B and C) was not significantly different.
The 208 patients presenting at the general practice who did not meet the risk stratification requirements and who were not treated with the triple therapy recovered at home and no hospital admis-

sions or deaths were reported.

3.4. Safety

In general, triple therapy with zinc, low-dose HCQ and azithromycin was well tolerated. After initiation of treatment in the 141 patients, 30 (21.3%) reported weakness, 20 (14.2%) nausea, 15 (10.6%) diarrhoea and 2 (1.4%) rash (Table 8). No patient reported palpitations or any cardiac side effects.

4. Discussion

This first retrospective case series study of COVID-19 outpatients in a primary care setting showed that risk-stratified treatment early after onset of clinical symptoms with triple therapy of zinc, low-dose HCQ and azithromycin was associated with significantly fewer hospitalisations (OR = 0.16; P < 0.001) in comparison with untreated patients (public reference data) of the same community. Based on the performed risk stratification, the prevalences of the co-morbidities hypertension, hyperlipidaemia and diabetes mellitus were the highest in group A (>60 years and clinical symptoms), asthma and other lung diseases were the highest in group B (≤60 years and SOB), and obesity and autoimmune disease were the highest in group C (<60 years, clinical symptoms and defined co-morbidities). The most frequent symptoms of these COVID-19 patients were cough followed by fever while available median body temperature measurements were in a normal range. Almost 50% of risk-stratified and treated patients were suffering from SOB while breaths per minute and blood oxygen saturation were still in the normal range. The median time from onset of symptoms to first medical consultation was 4 days (IQR 3–6 days). Approximately 16% of patients received co-medications known to be associated with zinc deficiency, such as antihypertensive drugs. No patient experienced any known severe adverse events that were considered drug-related during treatment or follow-up.

A growing number of reports provide evidence for the effectiveness or otherwise of a range of COVID-19 drug treatments. Therefore, a living systematic review and network meta-analysis was published to assess how trustworthy the evidence is using the Grading of Recommendations Assessment, Development, and Evaluation (GRADE) approach [24]. Based on their most recent update from 21 July 2020, the authors conclude that glucocorticoids probably reduce mortality and mechanical ventilation in patients with COVID-19 compared with standard care. However, the effectiveness of most interventions is uncertain because most of the randomised controlled trials so far have been small and have important study limitations [24].

Another meta-analysis focused on the effectiveness of chloroquine derivatives in COVID-19 therapy [25]. The authors concluded that chloroquine derivatives are effective in improving clinical and

virological outcomes and may reduce mortality by a factor of 3 in patients affected with COVID-19. They further conclude that big data are lacking basic treatment definitions and are the subject of
7
Fig.3[24][24][25]
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
conflict of interest [25]. At the time of this manuscript submission, only one peer-reviewed study had analysed the key health outcomes of COVID-19 patients diagnosed in a primary care setting [3]. Because of this gap in the data, the value of this study is multifold. It provides much needed recommendations for risk stratification and a treatment regimen to prevent hospitalisation and death of COVID-19 patients. The diagnosis of COVID-19 for all patients in this analysis was confirmed by PCR or IgG tests compared with a recent study in which <3% had a diagnosis confirmed by laboratory tests [26]. Starting triple therapy as early as possible after symptom onset is critical for treatment success because SARS-CoV-2 viral load appears to peak at Days 5–6 after symptom onset [27–29] and severe cases progress to ARDS after only 8–9 days [30,31]. Early antiviral treatment is an established protocol to manage severe disease progression, as was shown, for example, by a cumulative case–control study during the 2009 H1N1 influenza pandemic in Canada [32]. For patients at high risk for severe viral disease progression, it is recommended to start antiviral therapy as early as possible [33,34]. Early treatment might be also critically important to effectively reduce the SARS-CoV-2 viral load [5] and this underscores the role of early intervention by primary care physicians as reported herein.

A further strength of this approach was the simple risk stratification of symptomatic outpatients to determine the need for therapy, a strategy not yet applied in COVID-19 primary care [35] but routinely implemented in primary care for other diseases [36]. Underlying assumptions of the risk stratification used in this setting are different to other recommendations [37]. Here, age-stratified high risk was defined as >60 years (typically defined as >65 years) to encompass the common increase of co-morbidity incidences in this age group [38]. Patients ≤60 years with SOB, even without reduced pulse oximetry values, were treated because it was assumed the virus will likely spread from the upper to lower respiratory tract [39]. Also treated were patients ≤60 years with clinical symptoms and prognostically relevant co-morbidities [37]. By applying this risk stratification approach, respective care was tailored to patients with a higher likelihood for hospitalisation or fatality, which ensured that the medical principles of ‘patient first’ and ‘doing no harm’ were maintained [40]. As a result, 61.8% of COVID-19 patients were treated with standard of care only and recovered at home, and only 37.9% needed treatment with the triple therapy. The antiviral potential of HCQ has been broadly described in

vitro and in vivo [41–43]. HCQ has a long terminal elimination half-life of 32 days in plasma and 50 days in blood [44]. Therefore, the treatment approach was conservative, with the starting dose being the same as the maintenance dose and with a short treatment duration of only 5 days, being even more conservative than other recommendations [42]. HCQ-dependent intracellular increases in pH might directly interfere with pH-dependent SARS-CoV-2 replication [19]. Also, chloroquine and probably HCQ have characteristics of a zinc ionophore resulting in increasing intracellular zinc concentrations [20]. The dose of elementary zinc in this study was similar to doses previously studied to successfully prevent infections in the elderly [45]. The antiviral effects of zinc against a variety of viruses have been demonstrated during the last decades [46]. Zinc, in addition to its role as a general stimulant of antiviral immunity, is known to specifically inhibit coronavirus RNA-dependent RNA polymerase (RdRp) [21]. Based on the ionophore properties of HCQ, it has been hypothesised that zinc may enhance the efficacy of HCQ in treating COVID-19 patients [22]. In addition, zinc might inhibit the serine protease furin [47]. Furin is expressed on endothelial cells, monocytes/macrophages and smooth muscle cells in human atherosclerotic plaques [48] and therefore might play a critical role for the severe cardiovascular complications of COVID-19. As furin might be responsible to favour SARS-CoV-2 spread compared with other Betacoronaviruses [49,50] and as furin inhibition protects from certain viral-dependent infections [51], it may be important to evaluate the potential role of zinc in inhibiting this pathway.

Azithromycin was added to the treatment regimen as preliminary data provided evidence for more efficient or synergic virus elimination in conjunction with bacterial superinfection [13,52]. Although there is a synergistic antiviral effect between zinc, HCQ and azithromycin, zinc supplementation may be instrumental for the outcome of patient populations with severe clinical courses. Zinc deficiency was confirmed in a large number of healthy elderly [53] and in diabetic patients [54]. In addition, it has been documented that the antihypertensive drugs hydrochlorothiazide, angiotensin-converting enzyme inhibitors and angiotensin 2 receptor antagonists can result in increased urinary excretion of zinc with subsequent systemic zinc deficiency [55]. Age, co-morbidities and relevant co-medications align well with the majority of described COVID-19 patients at high risk, including the risk-stratified population of this analysis. Zinc deficiency might explain why certain patient groups seem not to benefit from HCQ monotherapy. During the 5-day treatment with the triple therapy and during follow-up, no severe adverse events were observed and no cases of cardiac arrhythmia were reported in this general practice, which is in accordance with available safety data for more than 300 000 patients [56].

Inherent to all retrospective analyses, our study has certain limitations, such as non-randomisation and blinding of treatment. Also, only the outcome data of the untreated control group based on the public reference were available; because no other data on patient characteristics or clinical symptoms were available, no risk adjustment was possible. Therefore, confounding factors and selection bias, among other issues, might exist. The demographic composition of the treatment group might also have had an influence on our findings. Because many physician appointments had to be managed by telehealth, vital parameters were not available for the majority of patients. Viral load and electrocardiogram (ECG) data were not analysed. Treatment with the triple therapy resulted in a numerically lower rate of all-cause death. In the absence of clinical details about the untreated patient group, the lower rate of all-cause death in the treated group was not statistically significant. However, patients in the treated group were all positively risk-stratified while the risk of the untreated group was obviously lower as this group included high- and low-risk patients. When we compared the outcome of all risk-stratified patients in the study group (treated and non-treated) with the control patients (not stratified, treated with standard therapy), hospitalisation and all-cause death were significantly less in the study group ($P < 0.0001$ and $P = 0.0154$, respectively). These data were not shown in the results section because relevant clinical information was not completely available for all patients in the control group to allow risk adjustment between groups.

In this study, the ratio of males and average age was comparable with a relevant number of other studies, but the distribution of co-morbidities was not [57]. The latter was expected because outpatients usually have a different distribution of age and especially of co-morbidities than critically ill inpatients. As expected, the prevalence of hypertension, hyperlipidaemia and cardiovascular disease correlated positively with age, while asthma correlated negatively. Approximately 50% of risk-stratified and treated patients presented with SOB, while the parameters breaths per minute and blood oxygen saturation were still within the normal range. These patients would usually not be considered for hospital admission, although SOB might be considered an alarming early sign of disease progression.
Based on the implemented risk strati-fication, these patients were identified and treated immediately. Indeed, three of four hospitalised patients were in risk stratification group B including patients especially with SOB, and also the
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[25][3][26][27–29]3031[32]3334[5][35][36][37][38][39][37][40][41–43][44][42][19][20][45][46][21][22][47][48]4950[51]1352[53][54][55][56][57]
R. Derwand, M. Scholz and V. Zelenko International Journal of Antimicrobial Agents 56 (2020) 106214
hospitalised patient of group A reported SOB at the time of consultation. This supports the assumption that COVID-19 patients with

SOB are at much higher risk for disease progression and need to be monitored closely.

In contrast to many other studies, the most frequent symptom was cough and not fever [58,59]. Changes in smell or taste in one-third of patients and a negative correlation with age were similar to findings from other groups [60]. While mean time from onset of symptoms to treatment was only 4.8 days (median 4 days), previously reported time spans range from 6.3 days [61] to 8 days [16], up to 16.6 days [14], or it was often even not reported [62]. In most of these studies, COVID-19 disease had most likely already progressed at the time of presentation to stages II or even stage III of the disease [6]. In many studies, often only limited information is provided about co-medications and specifically about clinical symptoms at admission [62]. The latter would be very important to better understand the differences in clinical presentation between inpatients and outpatients and thus the urgency for early anti-COVID-19 treatment in the outpatient setting [63]. The potential of zinc to enhance the antiviral efficacy of HCQ was already described in detail elsewhere [22]. This hypothesis was recently confirmed in a study using a similar triple therapy and treatment duration [23]. Zinc added to HCQ and azithromycin resulted in a significantly increased number of patients being discharged, a reduction in mortality, or transfer to hospice. In another study, when a lower dose of 200 mg of HCQ twice daily was added to basic treatment, mortality of even critically ill patients was significantly reduced [64]. These and our findings indicate that proper dosing of HCQ with its long half-life might be key for a favourable outcome of COVID-19 patients. In critical care, drugs with short half-lives are usually preferred. Especially in critically ill COVID-19 patients, higher doses of HCQ may have unforeseeable effects, e.g. on insulin sensitivity in obese patients [65] and on glucose levels in diabetics [66,67]. Besides glucose levels, it is important to closely monitor renal function, which is increasingly affected during progression of COVID-19 [68]. Because HCQ is substantially excreted by the kidneys, the risk of toxic reactions is greater in patients with impaired renal function [69].

4.1. Potential implications for clinicians and policy-makers

Clinical experience from severely ill inpatients with pneumonia who were treated with high-dose HCQ is not readily transferable to the outpatient setting with upper respiratory tract disease only**. For outpatients with a median of only 4 days after onset of symptoms, COVID-19 represents a totally different disease and needs to be managed and treated differently [63].** A simple-to-perform outpatient risk stratification, as shown here, allows for rapid treatment decisions and treatment with the triple therapy of zinc, low-dose HCQ and azithromycin and may prevent a large number of hospitalisations and probably deaths during the SARS-CoV-2 pandemic.

This might also help to avoid overwhelming of healthcare systems.

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Competing interests

RD is/was at the time of writing an employee of Alexion Pharma Germany GmbH, and his engagement and contribution to this study and publication was private and independent from his employer; MS is/was at the time of writing External Senior Advisor for the company LEUKOCARE (Munich, Germany) and is/was Manging Director at Starts- and -Ups Consulting (Frankfurt, Germany); VZ is/was a general practitioner in New York State (USA).

Ethical approval

This study was approved by the Western Institutional Review Board and was exempt under 45 CFR § 46.104(d)(4). Ref. Number: D4-Exemption-Zelenko (06-16-2020).

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Full article in .PDF:
https://www.sciencedirect.com/science/article/pii/S0924857920304258

COVID Tests Gone Wild—An Epidemic of COVID Positive Tests

By John Hunt, MD International Man January 11, 2021

1. Editor's Note: In the setting of COVID-19, almost every country in the world closed its borders, locked down its citizens, and forced businesses to close. Today, most governments still restrict travel, economic activity, and social gatherings.
The justification for these unprecedented measures has been a growing number of COVID-19 cases. This has unleashed an epidemic of COVID testing—with PCR and rapid antigen tests as the means of identifying positive COVID cases. Our very own Dr. John Hunt examines the science behind COVID testing, whether the testing paradigms are effective, and the rationality behind government response to the virus.

2. What COVID tests mean and don't mean
RT-PCR tests can be designed to be highly sensitive to the presence of the original viral RNA in a clinical sample. But a highly sensitive test risks poor specificity for actual infectious disease.
Rapid antigen tests are different. They measure viral protein. They do so by reacting a clinical sample with one or two lab-created antibodies that are labeled with a measurable marker. These antigen tests are often poorly specific, meaning they can show as positive in the absence of any actual viral protein or any COVID disease.

3. For a lab test, what does it mean to be sensitive? What does it mean to be specific?
I'll use COVID to help explain these terms. In order to do this correctly, we need to avoid using the language of the media and government because those institutions tend to mislead us via language manipulation. For example, they've wrongly taught us that a COVID-positive test is synonymous with COVID- disease. It isn't, as you will soon see.

4. So for this article, I will use the term "Relevant Infectious COVID Disease" to mean a condition, caused by COVID-19, in which a patient is sickened by the virus or has (in their airways) living replicating virus capable of being transmitted to others. This seems a fair definition of what we should be caring about in this disease. If the patient isn't sick and isn't capable of transmitting the disease, then any COVID RNA or protein that may appear in a test is not relevant, nor infectious, and therefore of little to no consequence.

5. You can think of a test's sensitivity like this: In a group of 100 people who absolutely have Relevant Infectious COVID Disease, how many people does the test actually report as "positive?" For a test that is 95% sensitive, 95 of these 100 patients with the true disease will be reported by the test as COVID positive and 5 will be missed.

6. Specificity: In a group of 100 people who absolutely do not have Relevant Infectious COVID Disease, how many will be reported by the test as "negative?" For a test that is 95% specific, 95 of these healthy people will be reported as COVID-negative and 5 will be incorrectly reported as COVID-positive

7. Sensitivity and Specificity are inherent characteristics of a test, not of a patient, not of a disease, and not of a population. These terms are very different than Positive Predictive Value (PPV) and Negative Predictive Value (NPV). PPV and NPV are affected not only by the test's sensitivity and specificity but also by the characteristics of the people chosen to be tested and, particularly, the patients' underlying likelihood of actually having true Relevant Infectious COVID Disease. The Positive Predictive Value—the chance a positive test actually indicates a true disease—is greatly improved if you test people who are likely to have COVID, and, importantly, avoid testing people unlikely to have COVID.

8. If you do a COVID test with 95% sensitivity and 95% specificity in 1,000 patients who are feverish, have snot pouring out of their noses, are coughing profusely, and are short of breath, then you are using that test as a diagnostic test in people who currently have a reasonable up-front chance of having Relevant Infectious COVID Disease. Let's say 500 of them do actually have Relevant Infectious COVID Disease, and the others have a common cold. This 95% sensitive test will correctly identify 475 of these people who are truly ill with COVID as being COVID-positive, and it will miss 25 of them. This same test is also 95% specific, which means it will falsely label 25 of the 500 non-COVID patients as COVID-positive. Although the test isn't perfect it has a Positive Predictive Value of 95% in this group of people, and is a pretty good test overall.

9. But what if you run this very same COVID test on everyone in the population? Let's guesstimate that the up-front chance of having Relevant Infectious COVID in the US at this moment is about 0.5% (suggesting that 5 out of 1000 people currently have the actual transmittable disease right now, which is a high estimate). How does this same 95% sensitive/95% specific test work in this screening setting? The good news is that this test will likely identify the 5 people out of every 1000 with Relevant Infectious COVID! Yay! The bad news is that, out of every 1000 people, it will also falsely label 50 people as COVID-positive who <u>don't</u> have Relevant Infectious COVID. Out of 55 people with positive tests in each group of 1000 people, 5 actually have the disease. 50 of the tests are false positives. With a Positive Predictive Value of only 9%, one could say that's a pretty lousy test. It's far lousier if you test only people with no symptoms (such as screening a school, jobsite, or college), in whom the up-front likelihood of having Relevant Infectious COVID Disease is substantially lower.

10. The very same test that is pretty good when testing people who are actually ill or at risk is lousy when screening people who aren't.

11. In the first scenario (with symptoms), the test is being used correctly for diagnosis. In the second scenario (no symptoms), the test is being used wrongly for screening.
A diagnostic test is used to diagnose a patient the doctor thinks has a reasonable chance of having the disease (having symptoms like fever, cough, a snotty nose, and shortness of breath during a viral season).

12. A screening test is used to check for the presence of a disease in a person without symptoms and no heightened risk of having the disease.

13. A screening test may be appropriate to use when it has very high specificity (99% or more), when the prevalence of the disease in the population is pretty high, and when there is something we can do about the disease if we identify it. However, if the prevalence of a disease is low (as is the case for Relevant Infectious COVID) and the test isn't adequately specific (as is the case with PCR and rapid antigen tests for the COVID virus), then using such a test as a screening measure in healthy people is forcing the test to be lousy. The more it is used wrongly, the more misinformation ensues.

14. Our health authorities are recommending more testing of asymptomatic people. In other words, they are encouraging the wrong and lousy application of these tests. Our health officials are doing what a first-year medical student should know better than to do. It's enough of a concerning error that it leaves two likely conclusions: 1) that our leading government health officials are truly incompetent and/or 2) that we, as a nation, are being intentionally gaslighted/manipulated. Or it could be both. (Another conclusion you should consider is that my analysis of these tests is incorrect. I'm open to a challenge.)

15. So what if you, as an individual, get a positive PCR test result (one that has 95% specificity) without having symptoms of COVID-19 or recent exposure to a true Relevant Infectious COVID Disease patient? What do you do? Well, with that positive test, your risk of having COVID has just increased from less than 5 in 1,000 (the general population risk) to about somewhere perhaps 5 in 55 (the risk of actual Relevant Infectious COVID Disease in asymptomatic people with a COVID-19-positive test). That's an 18-fold increase in risk, amounting to a 9% risk of you having Relevant Infectious COVID Disease (or a 91% chance of you being totally healthy). That may be a relevant increase in risk in your mind, enough that you choose to avoid exposing your friends and family to your higher risk compared to the general population. But if the government spends resources to contact-trace you, then they are contact-tracing 91% of people uselessly. And they are deciding whether to lock us down based on the wrong notion that COVID-positive tests in healthy people are epidemiologically accurate when indeed they are mostly wrong.

16. For the 50 asymptomatic low-risk people falsely popping positive out of each group of 1,000, what makes them pop positive? For a rapid antigen test, it is because the test is never meant for use as a screening test in healthy asymptomatic people because it's not specific enough. For a PCR test, positivity confidently means that there was COVID RNA in that sample, sure, but your nose or mouth very likely just filtered some dead bits of viral debris from the dust particles in the air as you walked through CVS to get the test before you learned you were supposed to use the drive-through. PCR can be way too sensitive.

17. A few strands of RNA are irrelevant. Even a few hundred fully intact viral particles are not likely to infect or cause disease. Humans aren't that wimpy. But keep in mind that there is a very small chance that the test popped positive because you are about to get sick with COVID-19, and the test caught you, by pure luck, just before you are to become sick.

18. On top of this wrong use of diagnostic tests as screening tests, the government has been subsidizing hospitals for taking care of COVID-19-positive patients. Let's say a hospital performs a COVID test 4 times during a hospital stay as a screening test in a patient who has no symptoms of COVID. If that test pops positive once and negative three times, the hospital will report that patient as having COVID-19, even though the one positive result is

highly likely to have been a false positive. Why do hospitals do this testing so much? In part, because they'll get $14,000 more from the government for each patient they declare has COVID-19.

19. When we see statistics of COVID-19 deaths, we should recognize that some substantial percentage of them should be called "Deaths with a COVID-19-positive test." When we see reports of case numbers rising, we should know that they are defining "case" as anyone with a COVID-19-positive test, which, as you might now realize, is really a garbage number.

20. Summary:

1. We have an epidemic of COVID-positive tests that is substantially larger than the epidemic of identified Relevant Infectious COVID Disease. In contrast, people with actual, mild cases of COVID-disease aren't all getting tested. So the data, on which lockdowns are supposedly justified, are lousy.
2. The data on COVID hospitalizations and deaths in the US are exaggerated by a government subsidization scheme that incentivizes the improper use of tests in people without particular risk of the disease.
3. Avoid getting tested for COVID unless you are symptomatic yourself, have had exposure to someone who was both symptomatic and tested positive for COVID, or have some other personal reason that makes sense.
4. Know that getting tested before traveling abroad puts you at a modest risk of getting a false-positive test result, which will assuredly screw up your trip. It's a new political risk of travel.
5. There is a lot more to this viral testing game, and there are a lot of weird incentives. There are gray areas and room for debate.
6. Yes, the COVID disease can kill people. But a positive test won't kill anybody. Sadly, every COVID-positive test empowers those politicians and bureaucrats who have a natural bent to control people—the sociopaths and their ilk.

21. John Hunt, MD is a pediatric pulmonologist/allergist/immunologist, a former tenured Associate Professor and academic medical researcher, who has extensive experience and publications involving PCR, antigen testing, and analysis of respiratory fluid. He is internationally recognized as an expert in aerosol/respiratory droplet collection and analysis. He's also Doug Casey's coauthor for the High Ground novels Speculator, Drug Lord, and the just-released Assassin, and he is a founding member of the LLC that owns International Man.

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https://www.lewrockwell.com/2021/01/no_author/covid-tests-gone-wild-an-epidemic-of-covid-positive-tests/

By way of full disclosure, neither Charity Navigator nor GuideStar lists America's Frontline Doctors as an operating charity, and its founder, Dr. Simone Gold's has received recent, negative publicity.

America's Frontline Doctors on the COVID vaccines

By H.P. Smith

American Thinker

February 3, 2021

(emphasis added)

America's Frontline Doctors (AFLDS) recently published a white paper that is the first objective look at the experimental vaccines that are now being enthusiastically offered to the public on an ever-increasing scale.

They are the group that advocated for the safety of the very well-known, long-studied, and widely-used drug hydroxychloroquine as a treatment for COVID-19 during the summer of 2020, holding a well-publicized press conference in Washington, D.C. They were, of course, immediately attacked and almost entirely silenced for their efforts because hydroxychloroquine had been touted by President Trump as an effective treatment against Covid-19.

A few key highlights from the paper: First, **all three vaccine candidates -- from Pfizer, AstraZeneca, and Moderna -- are still experimental and investigational**. Pfizer, in its own executive summary to the FDA on December 10, 2020 calls it "*...an investigational Covid-19 vaccine*."

The experimental vaccines from both Pfizer and Moderna utilize mRNA (messenger ribonucleic acid) which instructs the body's cells on manufacturing proteins. It is a **technology that has not "*ever been approved for any disease, or even entered final-stage trials until now.*"** There have been no independently published animal studies on any of the vaccines, and it is not yet known what effects they will have on the elderly, the very young, or women who are pregnant or might become pregnant in the near future. I'd say those are important groups whose safety is critical.

The white paper speaks extensively about Antibody-Dependent Enhancement (ADE) as a well-known complication of vaccines that can result in making the disease worse if contracted. It also goes into somewhat frightening detail about the possibility that the mRNA vaccines "*may permanently interfere with a woman's ability to maintain a pregnancy.*"

A curious detail that the AFLDS paper mentions is the National Childhood Vaccine Act, a little-known law passed in 1986 that **"*provides immunity from liability to all vaccine manufacturing companies.*" None of them can be held responsible** for rushing an untested, potentially dangerous vaccine to fight a flu virus that has an incredibly low Infection Fatality Ratio (IFR). In this case, it's a very real possibility that the "*cure*" may be worse than the disease.

The point of the white paper is not to say that people shouldn't get the shots; it is merely to inform people of the reality of the situation, such as the possibility that the vaccines may cause a worse spread of the virus via asymptomatic carriers. Those vaccinated may think it is safe to be around

others when it actually isn't. People should be given all the facts before making a decision for themselves or their families. At this stage, do people getting a shot even know which shot they are getting?

America's Frontline Doctors are also circulating a petition against the possible future mandating of the vaccine. Rumors have been spreading that **there may come a time when employers, schools, airlines, and even concert venues may require proof of vaccination, which would be illegal on many levels,** not the least of which is the access by third parties to people's private medical data. What happened to "*my body, my choice*?"

It's interesting that the same "*scientific*" and media communities that rebranded HCQ -- a drug with decades of history of safe and widespread use -- as unsafe during mid-2020 is now aggressively pushing the distribution of vaccines with very little testing data of any kind and, by definition, zero data regarding any possible long-term complications. I ask why.

I don't know about you, but I am not someone who takes everyone at their word, especially these days. I have a scientific educational background that taught me to involve the scientific method as part of my critical thinking. **I am much more likely to listen to a group of respected practicing physicians brave enough to risk their careers in order to advocate caution in taking an experimental "*vaccine*" than I am to take the word of politicians, media talking heads, or the liability-immune pharmaceutical companies poised to make billions from its distribution.**

Duckduckgo.com search for Pfizer/BioNTech vaccine 17 January 2021 Heartsill

https://www.fda.gov/media/144414/download

Fact sheet – 6 pages. From Pfizer and Biontech in Germany – Dec 2020

"The Pfizer-BioNTech COVID-19 Vaccine has not undergone the same type of review as an FDA-approved or cleared product. FDA may issue an EUA when certain criteria are met, which includes that there are no adequate, approved, available alternatives. In addition, the FDA decision is based on the totality of scientific evidence available showing that the product may be effective to prevent COVID-19 during the COVID-19 pandemic and that the known and potential benefits of the product outweigh the known and potential risks of the product. All of these criteria must be met to allow for the product to be used in the treatment of patients during the COVID-19 pandemic" (p. 6).

https://www.thehealthsite.com/news/4400-adverse-events-reported-in-us-after-receiving-pfizer-biontech-vaccine-789801/

4,400 Adverse events – Jan 7, 2021 The Healthsite.com

"Nearly 4,400 adverse events were reported after people received the Pfizer-BioNTech Covid-19 vaccine in the US, with 21 cases determined to be anaphylaxis, according to a report by the Centers for Disease Control and Prevention (CDC)."

https://www.theguardian.com/world/2020/dec/02/how-does-the-pfizerbiontech-covid-vaccine-work-and-who-will-get-it

How does the Pfizer/BioNTech vaccine work and who will get it? 2 Dec 2020

"The Pfizer/BioNTech Covid jab is an mRNA vaccine – a cutting-edge technology. The vaccine works by introducing into the body genetic material, called mRNA, that contains the instructions to make the so-called "spike" protein of the coronavirus.

In response to these proteins, the body's immune pathways are activated – a response that offers protection should we encounter the virus itself."The Pfizer/BioNTech vaccine, while exciting, brings logistical challenges. Among them, the vaccine must be stored and transported at about -70C.

https://www.newscientist.com/article/2261805-everything-you-need-to-know-about-the-pfizer-biontech-covid-19-vaccine/

Everything you need to know about the Pfizer/BioNTech covid-19 vaccine

NewScientist 3 December 2020

How effective is the vaccine?

About 95 per cent. The phase 3 trials of the Pfizer/BioNTech vaccine involved 42,000 people, about half of whom got the experimental vaccine and the rest a placebo. In total, 170 people fell ill with covid-19. Only eight of them were in the vaccine group; 162 had received the placebo. So around 5 per cent of cases were in the vaccine group, which is where the 95 per cent figure comes from. That is a very healthy number: the World Health Organization (WHO) has said it would be happy with 50 per cent.

What is in the vaccine?

The active ingredient is messenger RNA that carries instructions for making the virus's spike protein, which it uses to gain entry to cells. The mRNA is synthetic, not extracted from actual viruses. It is delivered in a tiny sphere of inert fatty material called a lipid nanoparticle.

mRNA vaccines (from **messenger RNA**, above) **BioNTech vaccine 17 Jan 2021 Heartsill**

https://www.popularmechanics.com/science/health/a34787908/what-is-mrna-covid-19-vaccine-pfizer-moderna/

Everything You Need to Know About mRNA, the COVID-19 Vaccine's Secret Weapon - Pfizer and Moderna both use this tech in their breakthrough vaccines. But how does it work? And is there a catch?

From Popular Mechanics Nov 30, 2020

What Is mRNA?

So-called "messenger" genetics do just what they say on the tin: deliver genetic information to parts of your body, usually in order to overwrite or erase the genetic information that's already there. Your body naturally makes and uses mRNA already. It's one of three kinds of ribonucleic acid (RNA) that all work together to translate pure genetic deoxyribonucleic acid (DNA) information into proteins in your body. It's taken scientists decades to identify this specific mechanic and turn it into medicine.

What IS mRNA Used For?

"These drugs also require a delivery system such as a lipid nanoparticle to get the drug to specific target tissues. [T]his carrier molecule can trigger its own immune response. Therefore, before taking [the RNA drug], which is administered through an intravenous infusion, patients must take a steroid, acetaminophen, and antihistamines to decrease the chance of having immune reactions."

Both Pfizer and Moderna have mRNA-based vaccines nearing the very end of clinical development and testing. There's no reason to think mRNA is the only *kind* of vaccine option—it's just the fastest in this particular situation, with a dozen other options in advanced development around the world. And it's true that no mRNA vaccine has ever made it to market, but some have tried. These will be the first to be approved, if they are.

"Moderna and BioNTech each designed a tiny snip of genetic code that could be deployed into cells to stimulate a coronavirus immune response," *Stat* reports.*

https://www.statnews.com/2020/11/10/the-story-of-mrna-how-a-once-dismissed-idea-became-a-leading-technology-in-the-covid-vaccine-race/

***The story of mRNA: How a once-dismissed idea became a leading technology in the Covid vaccine race**
November 10, 2020 Boston Globe

See the 2:02 STAT video "mRNA stands for messenger RNA.

"On May 18, Moderna issued a press release trumpeting "positive interim clinical data." The firm said its vaccine had generated neutralizing antibodies in the first eight volunteers in the early-phase study, a tiny sample. But Moderna didn't provide any backup data, making it hard to assess how encouraging the results were. Nonetheless, Moderna's share price rose 20% that day.

Some top Moderna executives also drew criticism for selling shares worth millions, including Bancel and the firm's chief medical officer, Tal Zaks. In addition, some critics have said the government has given Moderna a sweetheart deal by bankrolling the costs for developing the vaccine and pledging to buy at least 100 million doses, all for $2.48 billion. That works out to roughly $25 a dose, which Moderna acknowledges includes a profit.

In contrast, the government has pledged more than $1 billion to Johnson & Johnson to manufacture and provide at least 100 million doses of its vaccine, which uses different technology than mRNA. But J&J, which collaborated with Beth Israel

Deaconess Medical Center's Center for Virology and Vaccine Research and is also in a late-stage trial, has promised not to profit off sales of the vaccine during the pandemic.

Over in Germany, Sahin, the head of BioNTech, said a Lancet article in January about the outbreak in Wuhan, an international hub, galvanized him.

"We understood that this would become a pandemic," he said.

This story below reads like a plan was in effect with the government and Moderna. So the question is who owns Moderna? Wonder how hard it would be to have that question answered?

of course, no. Of Course NO! ---- Yeah, right.
https://factcheck.thedispatch.com/p/does-anthony-fauci-own-half-the-patent

This site can't be reached:

https://geopolitics.co/2020/09/05/bill-gates-vaccine-company-moderna-has-a-big-problem/

5 Horrifying facts about the fda Vaccine approval process

FDA-Vaccine-Approval-Process-2019.pdf

https://sarasotavaccinationchoice.wordpress.com/2017/02/07/5-horrifying-facts-about-the-fda-vaccine-approval-process-free-report/

83a9c750-e187-495d-9895-2ade2754bf2

Bill Gates told us about the Corona virus in 2015

https://www.news18.com/news/buzz/bill-gates-warned-us-about-covid-19-in-2015-now-he-is-predicting-two-more-disasters-3394487.html

HCQ SUPPRESSION (from a previous 'Reset' email)

https://www.oom2.com/t71428-flu-detat-infiltration-not-invasion
Standing to gain trillions from the COVID vaccines, the medical industry is supporting the political and legal aspects of this financial reset with its suppression of hydroxychloroquine and its approval of the medical martial law now being imposed, with varying degrees of intensity and/or success from jurisdiction to jurisdiction.

A huge funder and orchestrator of the medical aspect of this operation is Bill Gates. He funds and controls both the Big Pharma companies and the regulators that own the patents for the methods of detection and treatment of COVID-19, in addition to the actual SARS-CoV-2 virus, itself.
At this point, it's important to recall that if the novel coronavirus is a natural virus that evolved spontaneously, it would be illegal to patent it. Conversely, if it's bioengineered, then it would violate biological and chemical weapons treaties and laws. Regardless, in SARS-CoV-2, we are dealing with a monstrous crime and fraud; one of malfeasance, cynicism and cruelty that boggles the mind.
Two films came out this week, which illustrate some of the mechanisms by which this stunning coordination of repressive action by governments and corporations around the world has been deployed.

[The first film is 'ShadowGate' by Millie Weaver and the second is 'Plandemic II' by Mikki Willis, both of which are superb and that everyone reading this must view as soon as possible.]

There are more pix of this guy…

I rest my f'ing case…

Revised 10 Feb 2022 - gh