How I would treat COVID

By Steve Kirsch
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After I appeared on the *60 Minutes* story about fluvoxamine, I've received a lot of questions from people about how to treat COVID. Here are my answers.

***Disclaimer**: The views expressed in this article are my own personal opinion based on my 1,000+ hour study of cutting edge research. The science is rapidly developing and this document may change over time. Please do not take any drugs or other actions mentioned in this article without seeking advice from a qualified medical professional.*

Executive summary

1. **The earlier the treatment, the better the result**. Please take this virus very seriously, like your life depends on treating it successfully. Do not make the mistake of thinking "I'm healthy, I don't need treatment." Early treatment reduces risk of serious disease and organ damage (including brain damage).
2. **It's ideal to treat the disease with multiple drugs that target different mechanisms.** Treating with single agents and single mechanisms results in good outcomes, but as we learned in HIV, using a cocktail of drugs will give better outcomes.
3. **Fluvoxamine is a highly effective anti-inflammatory treatment option which can dramatically lower hospitalization and fatality rates as well as lowering the risk of long-haul COVID and organ damage.** It is an inexpensive, safe drug that has been around for 37 years, has been shown to be highly effective in reducing hospitalization and long-haul COVID symptoms. It can be prescribed by any doctor today for COVID. It was recommended for a "shared decision making process" by more than a 2:1 margin by a key opinion leader panel of experts from the NIH, CDC, and leading academic institutions. Note: Fluoxetine (Prozac) can be substituted if fluvoxamine isn't available; it acts in a very similar way as fluvoxamine, but the dosing is much lower.
4. **Fluvoxamine has been very effective in treating long-haul COVID cases.** Given early, fluvoxamine prevents long-haul COVID (e.g., 100% success rate in 77 patients in the Seftel trial). Given late, after someone has long-haul, it takes much longer and will not completely reverse the damage.
5. **You can help stop COVID** and save lives by enrolling in the fluvoxamine clinical trial. You must have tested positive, be over 30 years old and be within 6 day of first symptoms. If you don't want to do this, you can talk to your doctor about getting a prescription today. There is no need to wait for the clinical trial.
6. **There are other drugs that have strong evidence of efficacy against COVIDthat are not listed on the NIH Guidelines or approved by the FDA under an EUA. Using these drugs can dramatically reduce the hospitalization and fatality rates.** I've

listed what I believe are the best options in this document. Most of these recommendations (and all of the "top" recommendations) have shown efficacy in double-blind randomized controlled trials, the gold standard of evidence.

What treatment do you recommend for COVID?

I recommend taking two drugs from different rows from the table below. At least one of those drugs should be fluvoxamine since it prevents long-haul COVID which affects 27% of COVID patients.

As of March 14, the top two drugs that are also cheap and widely available worldwide are fluvoxamine + ivermectin.

Drug mechanism of action	Drug options
Anti-inflammatory	Fluvoxamine 50mg BID Fluoxetine (Prozac) 10mg QD Budesonide .8mg BID Doxazosin 4mg QD Fenofibrate 145mg QD Statins Metformin 500mg qam; 1g qpm aspirin
Multi-purpose	Ivermectin 0.2mg/kg QD x 3 to 5 days
Antiviral (Interferon re-activator)	Interferon lambda (clinical trial only)
Antiviral (COVID-specific)	VIR-7831 (GSK) Banlanivimab (Lilly) Casirivimab and Imdevimab (Regeneron)
Antiviral (general purpose for RNA viruses)	Molnupiravir (EIDD-2801/MK-4482; clinical trial only) GS-441524 (not generally available yet)
Antiviral (TMPRSS2 inhibitor)	Proxalutamide 200mg QD (clinical trial only) Camostat 600mg QID + bicalutamide 150mg Bromhexine 8mg TID
Antiviral (cathepsin inhibitor)	SLV213 (Selva)

Notes:

1. Generally, both Fluvoxamine and Ivermectin are widely available options in the US and if you just took both, you have a tremendous advantage against COVID (estimated 95% or more risk reduction).

2. All of the above are either a single injection (lambda) or taken for 14 days, with the exception of ivermectin which can be stopped at 5 days or when symptoms resolve.
3. While on fluvoxamine, you should avoid drinking caffeine because the drug will greatly magnify the effects. If you can't give up coffee, switch to fluoxetine.
4. Another option is to help science by enrolling in the clinical trial at www.stopcovidtrial.com. It is free and you can enroll in the comfort of your home. But you must be in the US, be at least 30 years of age and you must enroll within 7 days of first symptoms. This is a great option for people who don't have a doctor, can't afford a doctor or the medication, people who simply aren't sure if the drug will work or not, or people who want to help medical science move forward.
5. If things don't get better within a few days, try adding another drug.
6. If you cannot tolerate a drug, switch to the next one. None of the drugs require a waiting period for switching.
7. For anti-inflammatories, because they work via different mechanisms, you can use more than one anti-inflammatory but more than 2 is likely overkill
8. The prioritization is subjective and takes into account the effect size, quality of evidence, and availability. Only drugs where I believe that the evidence of efficacy strongly outweighs the risks are listed.
9. Bromhexine was ranked last in the TMPRSS2 inhibitor category because even though there was a very favorable randomized trial, Matt Hall's group at NIH couldn't replicate the TMPRSS2 inhibition (but were able to replicate it for camostat). So that's a real mystery.
10. Interferon lambda works orders of magnitude better than the monoclonals in terms of reduction of viral load, but is available in clinical trial only.
11. We may be sponsoring a trial in Brazil comparing two arms:
    a. ivermectin + bromhexine vs.
    b. camostat + bicalutamide+fluvoxamine.

    This will be an interesting test since both arms should perform extremely well.

What are the precautions I should be aware of before taking fluvoxamine?

See this article from the NAMI website for important precautions.

Aren't there lots of side effects from fluvoxamine?

For the relatively small dose and short duration used for COVID, most people experience no side effects whatsoever at the 50mg BID recommended dosing.

About 1% of people on the dosing recommended will have a side effect. The most frequent side effects are mild-nausea or insomnia. If you drink coffee while on fluvoxamine, the caffeine effects are greatly enhanced, so it is recommended to avoid coffee.

Side effects resolve when the drug is stopped.

Can I take these drugs if I am pregnant?

Talk to your doctor. Fluvoxamine is not recommended if you are pregnant, unless you have a serious condition such as COVID.

Ivermectin is not approved for newly pregnant women, but there is no evidence to suggest that this is a problem (it's just that it wasn't tested).

Are there any age restrictions on these drugs?

The dosages should be lowered for kids and for kids under 9 consult your doctor.

While the chances of kids getting sick with COVID are low, I would not want to lose my child to MIS-C which is related to the COVID infection.

Check with your doctor on her dosing recommendations. For kids, consider lower dosings of the SSRIs.

The dose for ivermectin is based on weight so the recommendation scales with age..

If my doctor isn't comfortable prescribing these drugs, what should I do?

A good doctor will ask you "do you have any evidence for that?"

Refer them to the JAMA study and the OFID paper by Dr. Seftel that confirmed the study.

You can also refer them to the *60 Minutes* story on fluvoxamine.

If you have a good doctor, they will be convinced by that.

If that doesn't work, another option is if you are feeling depressed because of the lockdowns, lack of human contact, etc. or you are exhibiting OCD symptoms such as obsessive hand washing, you can simply ask your doctor for a prescription for an antidepressant such as

fluvoxamine (generic brand or Luvox) or fluoxetine (Prozac). These are common medications for these conditions and the bonus benefit for you is that it will address your COVID as well. You can call any telehealth provider in this case (such as Teladoc) and it should be easy to get this medication since you are asking for a medication for an FDA-approved use.

Here are the rules for telehealth providers so check your state laws.

If you have confirmed COVID and either are 65 or older **or** have at least one risk factor such as cancer, diabetes, hypertension, heart, or lung disease, BMI>35, then you can get a prescription for fluvoxamine at CityHealth via telehealth. There is no charge if you have insurance. See their excellent information sheet on fluvoxamine.

If you don't qualify for CityHealth, here is a list of other telemedicine docs that that prescribe treatments for COVID (including fluvoxamine) based on the latest scientific evidence:

1. Dr. Syed Haider. He charges a flat $115 for the consultation/prescription and all follow-up and does not take insurance. I highly recommend him. However, he's only licensed in these states: Arizona, Colorado, Delaware, Florida, Hawaii, Idaho, Illinois, Indiana, Iowa, Kansas, Massachusetts, Michigan, Missouri, Nebraska, New Jersey, New York, North Carolina, Pennsylvania, South, Carolina, Texas, Vermont, Virginia, Wisconsin, Wyoming.
2. Text2md Covers: AL, AZ, CO, FL, GA, IA, ID, IL, KS, KY, MD, ME, MI, MN, ND, NE, NJ, NV, OK, SC, SD, TN, UT, VT, WA, WI.
3. Myfreedoctor.com but ask for Robert Apter, MD in Sedona, AZ. **He can prescribe in all 50 states!**
4. Dr. Tom Yarema: California (CA)only
5. Sandra Arce-Garzon, MD: Wisconsin (WI) only
6. Margaret Aranda, MD. West Hills, California 91307-4011 O: (800) 992-9280
7. The doctors listed on this site: Doctors that prescribe ivermectin.

If they don't prescribe fluvoxamine today, suggest they contact one of the doctors above.

Why should I take any drugs at all to treat COVID? Isn't that dangerous?

Because we have evidence that shows it can substantially improve your odds of recovering from COVID without any long-term or permanent damage.

In general, using drugs as soon as ASAP after you learn you are infected is more likely than not to:

1) Reduce the chance that you will be hospitalized or die

2) Allow you to recover from COVID much faster
3) Reduce the chance you will have symptoms
4) Reverse the COVID symptoms you may be having within an average of 3 days (if given early)
5) Reduce the chance that you will suffer from any long term effects, such as long-haul COVID (aka PACS), permanent organ damage (including lung and brain damage), or death from COVID or MIS-C.

For more information, see Eric Feigl-Ding's tweet especially point 7).

Why isn't my doctor telling me about these drugs?

Because she likely doesn't have time to read the papers and all the studies and all the evidence in the Fluvoxamine public data repository and then make a determination.

Evidence is never perfect. Evaluating all the evidence and getting the right answer is hard. Could there be confounding factors that explain the result?

Instead of analysis with the risk of making a mistake, most doctors typically wait for either a Phase 3 study, an EUA, or the drug to be added to the NIH Guidelines. In other words, they wait for some other recognized authority to make the determination.

The problem is in a pandemic, that takes too long to wait for one of these events that can change clinical practice.

Why do I need to take more than one drug?

We know today that no individual drug is likely to be more than 90% effective on its own. By combining very safe drugs with different and complementary mechanisms of action, we should be able to achieve very high efficacy rates against the virus while still having a very low side-effect profile..

There are several reasons why individual drugs are typically not more than 90% effective:

1) **Specificity.** Most of these drugs are repurposed drugs not specifically designed for the virus, therefore they are not super selective. To achieve a 100% kill rate for the virus, the dosage would have to be so high it would either kill you or cause massive side effects
2) **Variability of the host and the disease**. Everyone is different and reacts differently to both the virus and drugs. So if we just used an anti-inflammatory, but the person has a high viral load, we'd have made the wrong choice. (because in general, to get to 100%, most drugs would have to be dosed so high as to kill you along with the virus),
3) **Single action**. The drugs themselves are typically primarily single mechanisms. But a virus is best fought on multiple fronts. It's the same way you'd battle a fire. For a fire,

you'd try to reduce the heat, the fuel, and the oxygen since these are the three pillars of a fire. A virus has a similar set of attack points.

Why is your method better than other combinations?

We have a very clear understanding of the possible attack points to disable the virus without killing you in the process.

The method I describe above is basically an enumeration of those attack points, and lists the drug which has the best result for that attack point.

This approach may ultimately not prove to be the most effective way to treat the virus, but it is the most logical starting point for an effective treatment. So it's logically the very first thing you would try.

Is there sufficient evidence for these treatments?

I certainly believe so. Every one of the top recommendations above (individual drugs) are supported by the gold standard of scientific evidence, double-blind, placebo controlled randomized clinical trials.

The problem is that doctors and the medical community in general won't touch these treatments because they believe there isn't enough evidence to justify widespread use. They take a one size fits all approach (waiting for a definitive phase 3 trial) rather than looking at the data on the table and seeing if the data is strong enough to overcome the common confounding factors.

What doctors should be doing is calling for an immediate systematic review of the evidence we have now for the top drugs, rather than saying "let's wait months for more data." You'd only wait for phase 3 trial if the systematic review fails to confirm we have enough data. Instead, doctors skip that part and simply make the assumption that the systematic review would fail. That's a bad mistake that costs lives.

For example, let's look at fluvoxamine.

For fluvoxamine, the chance of the symptom data happening by chance is less than 1 in 10^{14} since none of the 77 treated patients had long-term COVID vs. 60% of the no treatment group. Nobody disagrees with this number (it's a straight forward math calculation that anyone can do in seconds using Fisher exact test). No confounders, other than the doctor fabricating all the data, are strong enough to overcome that p-value.

But it's unlikely Seftel is fabricating the data:

1. There are over a dozen confirmatory data points that are independent of each other (all positive)
2. He has no history of scientific fraud
3. He has no motivation to fabricate the data (it isn't his drug and he doesn't profit from it)
4. It would wreck his career
5. It doesn't fit with his personality type
6. It would be easily be discovered (by interviewing his patients or looking at hospital records)
7. If he was faking the data, he wouldn't have made such a perfect result because the more perfect the data, the more suspicion
8. 100% of the patients demanded the drug after 3 weeks into the trial (they are independent observers of how their peers reacted to the drug)
9. David Wiseman, who is the only guy to find discrepancies in Boulware's HCQ data, couldn't find any fault in Seftel's data

Any confounders (placebo effect, healthy patient bias, observer bias, trial protocol not registered with IRB in advance) are insufficient to overcome a p value of 1e-14 (they hardly move the needle). I point this out to everyone who claims that "We need to wait for phase 3 data" and then they either 1) change the topic or 2) say they don't have time to discuss. So I'd be grateful to anyone who does have the time to answer my question with actual data.

Secondly, those confounders cannot explain the hospitalization data either.

A few people have pointed out the fluvoxamine reduces anxiety. However, this doesn't change any of the overall statistics of either trial (see the evidence for details)

So there is a bias to simply excluding anything not DB-RCT as anecdotal, even though nobody can explain the outcomes in this case. We never look inside the box to see if it merits "special treatment."

Reality: **the people who claim we need more data don't have any data to back up their claim** that we **need** more data. They simply cite "there are lots of failed phase 3 trials" but **none of them have an actual number** we can apply to a p-value to test for significance in studies that are not DB-RCT. This means they completely ignore anything that is not a DB-RCT. So in the Seftel study, if half the people in the no treatment group died, and nobody in the treatment group died, they would totally ignore that and say, "we need more data" since they have no criteria to adjust the p-value.

Key opinion leaders (KOLs) from the CDC, NIH, and leading academic institutions met on January 22, 2021. See this Washington Post op-ed for more information about that meeting. They strongly recommended by more than a 2:1 margin that patients talk to their doctors about fluvoxamine. In Croatia, they did a debate with 150 people in the medical community voting. Dr.

Robert Likić presented the argument for using now. The vote was 54% to prescribe it now vs. vs. 34% to wait for the phase 3 data (the others didn't have an opinion).

People are welcome to analyze that data, but as of March 9, 2021, nobody (except for David Wiseman) has asked to see the patient data. They simply claim we need more data without looking at the data we have. Wiseman did look at the data and concluded that we did in fact have more than sufficient data to get an EUA; it was obvious to him.

When I try to get people to look at the data on the table, they simply say, "I'll wait for the phase 3 data."

This is a pandemic. We should be making decisions based on the best evidence available.

Our decision making should be based on which decision is likely to save the most lives based on a probabilistic analysis based on the evidence we have at hand. The decision should not be based on minimizing reputational loss if the decision is wrong. In short, we should be more concerned about lives than reputational risk of the NIH or FDA.

I strongly disagree with people who believe that we should wait for more evidence for fluvoxamine, i.e., the Phase 3 trial to complete.

The fact is that the Phase 3 trial is recruiting very slowly. The day after the *60 Minutes* interview aired, we could attribute only 5 additional new enrollments from that interview. So people are simply waiting for more data, just like Francis Collins told them, instead of enrolling in the trial (which was put in the Overtime section of *60 Minutes* that fewer people see). So this is a bit like the famous Samuel Beckett play *Waiting for Godot* where everyone is waiting for Godot who never comes. In our case, we will eventually complete enrollment in the Phase 3 trial, but as of March 10, it will take another 4 months to execute since we need to enroll over 1,000 patients and we are now enrolling at a rate of 50 a week. The NIH and public health officials are doing nothing to help publicize the trial and companies like facebook block our ads. It's frustrating. And even more frustrating when you consider how compelling the data on the table is.

The probability fluvoxamine works is basically nearly 1, the effect size is 75% or more.

So deploying fluvoxamine we are likely to save 75,000 lives a month compared to the status quo treatment. Deploying option B (the current strategy) is 100,000 lives lost.

The people who claim we need data say that the Seftel result was simply because patients got to choose and every healthy patient chose to take the drug. So we'd have to believe that patients who opt for treatment never get sick with COVID. That's ridiculous. If that were true, the Lenze clinical trial (and all other COVID trials) would have no patients who got sick at all (or at least two orders of magnitude lower than the general population) since all of the people who enrolled in a clinical trial "opted for treatment" so none of them should get sick.

So the argument we need more evidence falls apart if you examine it closely. However, the people who make that argument are not held accountable to defend it.

So that's the real reason why we need more data: because doctors don't want to face the fact that they can't explain how the data could be wrong and refuse to accept the evidence in front of them.

There's a choice here:

1. We can keep ignoring the data, tell people to do nothing, and lose 100,000 lives a month for sure
2. We can try fluvoxamine now and have well more than a 60% chance to save 100,000 lives (since the overall success rate of phase 2 trials in phase 3 is 60%)

What do you think we should do?

Note: As far as I know, there has never been a phase 2 trial with the level of evidence that fluvoxamine has today that then later failed in a properly executed phase 3 trial with the same endpoints as the phase 2 trial. I've asked dozens of people and nobody has a single example. So since there have been over 40,000 phase 3 trials, we are looking at a 1/40,000 chance that the fluvoxamine phase 3 trial fails. It is stupid to wait.

If you are drowning and I throw you a life preserver that was only tested 150 times, worked every time, and the chance of failure is 1e-14 based on the most recent quasi-randomized trial (since a DB-RCT would be unethical to do), do you take it? Or do you ask for a phase 3 trial first??

Interestingly, **there seems to be a double standard on evidence**. There was only one RCT on mask wearing, which showed a very small protective effect that was not statistically significant, and it was done in Nov 2020, whereas the CDC mask policy was 7 months before the first and only RCT.

No RCT for Masks? No Problem says it's fine to not have an RCT for mask wearing because we can observe the effect in populations with and without masks.

But they actually did an RCT in Denmark with 4862 participants in November 2020. It's the only RCT on mask wearing. And of course the medical community values RCTs above all other forms of evidence, right?. Guess what? It made **no statistically significant difference** with a p value=.33 (and the range from the study shows it could even be harmful). The mask wearers in the study were on average just 15% less likely to get infected, but the benefit was small and not statistically significant. The study was criticized because 7% of people in the mask group didn't comply. But that leaves 93% who did.

What's bizarre is that for fluvoxamine RCT we have a p value of .009 and effect size of 100% and we need more DB-RCT data!?!?!

Note that one paper, A rapid systematic review of the efficacy of face masks and respirators against coronaviruses and other respiratory transmissible viruses for the community, healthcare workers and sick patients concluded mask wearing was beneficial, but there is just one tiny little problem: **all the trials that were cited were done 8 years before COVID**!!!

There is a study that was just published in March 2021 (this is almost a year after face masks were recommended by the CDC) using observational data that concluded mask wearing was effective. **So the CDC basically issued this recommendation without any RCT or observational studies to rely on.**

The Danish RCT only looked at the protection of the wearer. There is no RCT on whether wearing a mask prevents someone with COVID from spreading the virus. There was a hair salon study that is used to prove that masking works, but there were no controls since everyone was wearing masks. The comments on the Brooks paper in JAMA are interesting to read.

It's the same story for hand washing. Where was the demand for an RCT on that one? I could only find this paper Does hand hygiene reduce SARS-CoV-2 transmission? which concluded it didn't work and it wasn't even a study.

The bottom line is that RCT data isn't necessary at all if the CDC says to do something.

Why isn't anyone at the NIH promoting the fluvoxamine trial?

They aren't allowed to. It's considered unethical because it would be promoting one trial over another.

If your method is so great, why isn't the WHO adopting it?

WHO has a SOLIDARITY trial, but it is only for in-patients. The drugs here are for early treatment (i.e., outpatients). Unfortunately, the WHO doesn't have an infrastructure for testing drugs that would prevent hospitalization. Now you would think they should and you'd be right about that.

Why isn’t fluvoxamine listed on the NIH Guidelines?

I have no idea and they aren’t allowed to tell me because their deliberations are all secret.

A panel of experts from the NIH and CDC that met on Jan 22 recommended it be put on the guidelines and that recommendation is being ignored.

I’m told that secrecy is in the best interest of the public. While I disagree with that policy, there is nothing you or I can do to change it.

Also, because fluvoxamine isn’t on the NIH guidelines, if you talk about it in a post on social media, you run the risk of having your post censored and your account suspended. Medium cancelled my account for life for posting one article about fluvoxamine, Facebook censored a post saying I would be talking about fluvoxamine on *60 Minutes*, and warned me if I violated their community standards again, my account would be suspended.

Will these drugs work on hospitalized patients?

More than likely.

For example, in every single case where people have tried fluvoxamine that I’m aware of the patient got better and got out of the hospital. There was one exception where the doctors were authorized only for 15 days of fluvoxamine on an intubated patient. That patient got better on the fluvoxamine but after the fluvoxamine was stopped, they regressed and died.

However, we have not been able to interest any hospital in opening a clinical trial on these agents (we have the money but no hospital is willing to open a trial), even for newly hospitalized patients, so no major hospital has added any of these drugs to their protocol.

Did you apply for an EUA?

As of March 10, our EUA application has been sitting at the FDA for almost 6 weeks.
The FDA should be required to explain how many lives are going to be saved with this drug approved vs. not approved for COVID.

The difference is likely 75,000 lives a month since there is a 100% chance that the effect is real, and 95% chance it is 75% protective at all. That’s what the statistics say.

If the FDA has better data, they should produce it now, otherwise, accept the data on the table if they cannot disprove it.

How can I help?

Simplest way to help us make progress is to make a donation to CETF.

I have many more questions…

I thought you would!

See my extensive COVID FAQ which covers a lot more questions including the evidence supporting fluvoxamine, the mechanisms of action, treating long-haul COVID, and more.

For more information on the backstory behind my efforts to let the world know about drugs mentioned in this article, see The fluvoxamine backstory.