COVID FAQ

By Steve Kirsch
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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.*

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About this document

This document contains answers to more detailed questions than I covered in the [How I would treat COVID](#) document. You should definitely read that document first.

Who are you?

I'm a serial entrepreneur and philanthropist and founder of [COVID-19 Early Treatment Fund](#).

I'm a serial high-tech entrepreneur and philanthropist. I have started 7 high tech companies, two with billion dollar market caps. I have a SM and SB in EECS from MIT. More about me on [Wikipedia](#) and [LinkedIn](#).

When the lockdown began, I took a leave of absence and started an organization, [COVID-19 Early Treatment Fund](#), that funds research on using repurposed drugs to treat COVID. We raised $5M, received 60 grant applications and funded 14. I chose that approach because I thought it would be the fastest and cheapest way to end the pandemic. Once we find a drug combination that reduces the hospitalization/death rate from COVID by more than a factor of 10, we should be able much more rapidly get back to normal.

For more, watch this 3 minute intro: [COVID-19 Early Treatment Fund (CETF) Introduction - YouTube](#).

I was featured in a [front page story in the LA Times](#) about COVID and the drug CETF funded, fluvoxamine, has been the subject of [many stories](#).

For more information, see [Fluvoxamine - The backstory](#) which answers questions about the history of the research, why we funded it, and suggestions on how we can improve the system so that it works better in the future.

Why are you writing this?

To save lives.

This document is written to help anyone who has an active COVID infection or long-haul COVID symptoms, regardless of whether you've been vaccinated or received one of the monoclonal antibodies. This document applies to all stages of COVID, from just infected to intubated in the ICU.

What do you recommend if I have COVID?

See [How I would treat COVID](#).

I'm in the ICU with COVID. What should I do?

It depends, but consider cyproheptadine and/or fluvoxamine and ivermectin.

The fastest, proven treatment is 8mg TID cyproheptadine for patients who are at WHO severity scale 5, 6, or 7. The evidence on this is so convincing that they are about to add it as an arm in a well respected large clinical trial.

One physician reports "we have 24-year olds with respiratory rates of 55 per minutes and impending doom completely resolve their tachypnea to normal respiratory rate within one day and get discharged within 48 hours with simple blockage of liberated serotonin." Physicians have been using this for 6 months because it works.

Cyproheptadine works quite fast when it works (e.g,. discharge in 48 hours).

If a patient isn't responsive to cyproheptadine within 48 yours, then using fluvoxamine and ivermectin in parallel is prudent since these drugs are complementary and both extremely effective:

1) 100mg TID of fluvoxamine until the CRP stabilizes, then reduce dosage and check for slippage of CRP upwards. See the image at the end of the evidence section below.
2) 0.3 mg/kg day of ivermectin orally for 5 days. Note this is a higher dosage than for outpatients. Ivermectin is complementary to the drugs above so can be added if the patient isn't responding. There is no downside to adding the ivermectin. The evidence is compelling.

NOTE: ICU doctors are in control of what you get. But most ICU docs will agree to your requests if there is evidence of both efficacy and safety. If not, you have the right to switch hospitals.

What are the most important points to note other than the drugs and dosing?

There are many points, 14 of which I've summarized below.

1. **If taking fluvoxamine, avoid caffeine.** If you must, take just 20% of what you normally would take. This is because the drugs amplify the effect and duration of caffeine. So

think 5x more effect than normal and the caffeine will buzz you for 31 hours or more. If you can't live with coffee, consider using fluoxetine instead.

2. **Make sure you don't have any dangerous drug interactions.** Be sure to tell your doctor about any meds you are currently taking such as MAOIs. Check this list of drug interactions.

3. **Start immediately.** The sooner you treat, the better. If you treat late, the drugs won't guarantee you won't be hospitalized. As soon as you get a positive COVID diagnosis, get on the drug. People who do that early basically have zero symptoms… like they never would have known they had COVID. Hours matter because the virus doubles in size every 10-12 hours.

4. **It works even if you start late.** It has worked for hospitalized patients, patients on high flow oxygen, intubated patients, and long-haul COVID patients. It will just take longer to reverse symptoms (and may require higher dose).

5. **The dosing is just a guideline.** 50mg BID dosing was both sufficient to avoid any negative outcome, but it was also low enough to avoid side effects. Fewer than 2% of patients will have mild nausea at this dose and it will go away when you stop the drug. We have seen people with relatively mild symptoms do well on just 50mg QD. You can tell when you have an effective dose because your symptoms will start to resolve in 24 hours and get better every day. So if you can't tolerate 50mg twice a day, reduce the dosage and monitor your symptoms. If you are declining, increase the dose.

6. **You can benefit even if you don't have symptoms.** We haven't seen any evidence that waiting until you have symptoms before taking the drugs gives better outcomes. You really don't want to take your chances. If you wait, the drugs may be too late to help you. So I advise starting as soon as possible even if you don't have symptoms because the whole idea is to keep you COVID-symptom free.

7. **If you find a treatment with better evidence than fluvoxamine, take it.** Otherwise this may be the best you have. See the table in the Seftel paper for the statistics. If you know of something with two studies published in top peer reviewed journals with an effect size greater than 100% protection from hospitalization and long-term COVID effects, go ahead and take that. We haven't found anything close to that.

8. **Even if you've been vaccinated or got one of the monoclonals,** if you get infected with a COVID variant that is not covered by your vaccine or monoclonal, this information may come in handy if you get any symptoms. It is also useful for your friends.

9. **There is no withdrawal phase.** You can stop both drugs cold-turkey at the end.

10. **The duration is 14 days.** We haven't tested shorter durations of treatment so 14 days may be overkill. The 14 days was picked since that covers the time the virus is likely active in your body.

11. **The dose I recommend is a third of the max dose used in the Lenze trial.** The WashU fluvoxamine study used dosing that went up to 3 times higher than what we suggest here. The dosing suggested here mimics the later study (Seftel) which showed that ⅓ of the maximum dose used in Lenze gives 100% protection against actual hospitalization, rapidly resolved symptoms, eliminates any long-term COVID, and avoided any drug side effects. So you can take a higher dose, but you may just be adding side effects, and not increasing benefit (and possibly reducing benefits). We don't

have enough data on the dose vs. efficacy so for now we are simply suggesting the lowest dose with a 100% protection.

12. **Any psych effects are minimal.** These normally take [4 to 6 weeks to kick in](). So the dose is short enough so people won't really notice much of a difference other than you are back to your old self, your brain fog is gone, etc.

13. **Any side effects are temporary and will go away when you stop the drug.** Fluvoxamine alone can cause mild nausea. It goes away as soon as you stop using the drug.

14. **It may be helpful even if you are 14 days into COVID and have apparently recovered.** There are cases where people apparently recover and later relapse. Treating for 14 days may reduce the likelihood of this happening, which intuitively makes sense but we have no studies on this yet.

What do you recommend if I've had COVID, but still have symptoms, i.e, long-haul COVID?

If you've had COVID, but still have symptoms then to use a fire analogy, the fire is out, but we are now dealing with some "hot coals" that are still smoldering. We could also be dealing with permanent organ damage, but let's hope that isn't the case.

The general method is low dose, but relatively long duration. Ideally, if the drug works, you will see very solid results within a day or two, and be close to back to normal after about 10-12 days. At that point, you want to cut the dose in half to ensure that you keep your "gains." If you don't, you will need more time at the higher dose.

Here are drugs that have worked for long-haulers to relieve their long-term COVID symptoms such as fatigue, brain fog, etc.Try one, if it doesn't work, take a week or two to recover to baseline before switching otherwise you can get serotonin syndrome (which can be life threatening). Everyone is different and there are no set protocols.

Never assume that higher dosing is better; higher dosing could be worse. For example, a little alcohol is fine, too much alcohol leads to hangovers. Same thing applies to drugs. Find your sweet spot through experimentation within limits set by your doctor. We know that meds that **work for some long-haul COVID cases will actually make things worse in others** so it is important to methodically try the list and see what works for you.

Drugs that have worked for people that are available today off-label:
1) 50 mg BID of fluvoxamine - If it works for you, you can start to feel much better in 24 hours and get progressively better over days. Dr. Drew for example, was suffering from brain fog and fatigue after recovering from COVID and it persisted. After he started on fluvoxamine, he got 80% better on the first day (and his fans all noticed the difference exclaiming "he's back"), but it took 12 days at 50mg BID till he felt he was back at 100%

and at that time he cut the dose in half and did not regress for 4 days, but then had to up the dose to 50mg BID. If you have any side effects, **try changing the timing of the drug**, e.g., Dr Drew found it was better to take the drug at night rather than in the morning. You need to find **the dose and timing that works best for you**. It might be half that dose taken at bedtime, for example. Once you are 100% recovered, then you can try halving that dose and seeing if you maintain your recovery. Eventually, you'll be able to stop the drug entirely. Note that Dr. Drew used 50mg BID dosing which was too much (he got a response very quickly, but a PK analysis of the drug shows that takes 7 days to reach a steady state concentration in your body so if you get a very quick response like that, it's being overdosed).

2) 10mg QHS of a TCA like Elavil - Tricyclic antidepressant (TCA) have been game changing for some long-haul COVID patients.TCAs works differently than an SSRI so if the SSRI made things worse, a TCA may make things better. **Be sure to wait at least 4 days before switching between drugs, as mixing an SSRI with a TCA can be life threatening** (which your doctor should tell you). One 11 month long hauler wrote: ""Elavil almost resets me in the morning. I go to bed feeling like the crash is coming, take Elavil as scheduled at night, and wake up the next no longer with the crash present. Whereas before, the crash would linger on the day after and I'd be in bed all day exhausted."

3) 7.5mg QHS of Mirtazapine - Mirtazapine affects different receptors than the other drugs and may be effective.

4) Try combining the fluvoxamine and the mirtazapine. This will block all the interesting inflammation receptors. When used with fluvoxamine, it magnifies the effect by 3X to 4X so don't exceed 7.5mg.

What is the fluvoxamine prescribing information?

There are no specific COVID-specific cautions with regards to prescribing.

The standard FDA cautions apply. See [Fluvoxamine Prescribing Information](#).

What are fluvoxamine drug interactions?

Use with any monoamine oxidase (MAO) inhibitor is contraindicated.

WashU noted [three important issues on their fluvoxamine page](#):

- First, patients should **avoid caffeine** while taking fluvoxamine. It prevents the body from properly metabolizing caffeine, making it stay in the system 5 times as long as expected. This is not dangerous but can cause insomnia and jitteriness. If they must have caffeine, they should limit their intake to ½ of a small cup of coffee, or one can of soda, or one tea, in the morning. They can return to their regular caffeine intake once they have stopped taking fluvoxamine.
- Second, fluvoxamine affects the metabolism of some drugs. In particular, people taking theophylline, clozapine, olanzapine, or tizanidine should either avoid fluvoxamine or talk to their doctor about how to avoid a dangerous drug interaction (e.g., by reducing or minimizing the drug).
- Finally, fluvoxamine is an antidepressant. People with psychiatric conditions such as bipolar disorder or who already take psychiatric medications such as antidepressants or mood stabilizers should talk with their doctor before taking fluvoxamine.

When should I start the drugs?

As soon as possible after you know you are infected with COVID.

A virus is like a fire: treat early to minimize risk of irreparable damage.

The later you start the drugs, the longer it will take to recover and the higher dose you may need to take.

If I take the drugs, can I still die from COVID?

Yes. While the drugs are highly effective, people on the drugs do die, so it's not a 100% guarantee.

The best way to avoid death is to use both an antiviral and anti-inflammatory. How many people who have used both have died? I'm not aware of a single one.

For example, in the Hoertel paper, there was 1 person of the 9,500 in the study who was on fluvoxamine who got COVID and died.

I have low risk factors. Do I need to take any drugs?

Maybe.

The problem is that we cannot predict ahead of time who needs to be treated.

If you ignore treatment, you can end up with long-term COVID or dead.

I know a 22 year old in great health who has been completely sidelined (mostly in bed) for 10 months now from COVID and nothing works. I also know that there are kids who recover from COVID and then die from [MIS-C which is related to the COVID infection](#).

Please take this disease seriously. In my view, everyone should treat this disease as if it will kill you if not treated, because you may be right.

Is it legal for you to talk about using drugs for indications that aren't approved by the FDA?

Yes and innovation requires open discussion about cutting edge treatments.

Companies are not allowed to market or promote an FDA-approved medication beyond its approved indications. This is why you will not see any doctor or medical institution advertise fluvoxamine or ivermectin as a COVID treatment.

But if you aren't doing so for commercial purposes, then freedom of speech still applies, so this document is legal because I don't make any money when someone takes the drugs.

Similarly, CETF can run newspaper ads letting people know about the drug and its efficacy and encourage people to talk to their doctors.

However, any doctor who is willing to prescribe fluvoxamine is not allowed to market or advertise that fact if the drug isn't FDA approved for that use.

What this means is that even if the drug is 100% effective and is on the top of the NIH Guidelines as the most important drug to get, physicians and medical groups are not allowed to

let you know that they will prescribe the drug until the FDA approves its use (with an EUA or straight approval). So it will be months before you see anyone advertising about the drug.

If this is such a great treatment, why haven't Stanford, Harvard, UCSF, and other institutions added this to their clinical practice guidelines?

They are waiting for the Phase 3 trial to complete.

Even if we can prove the probability of that trial being successful (based on the evidence on the table) is 99% or better, it doesn't matter: they will wait. We can't even get on the clinical practice committee agenda in any institution without a phase 3 trial (even in a pandemic).

But privately, we know the Deans are recommending the drug to their friends.

The reasoning is a combination of:

1) Genuine discomfort over whether the evidence is sufficient to warrant an "all in" commitment (and note that as of February 17, 2021, only one academic institution has reached out to any of the principal researchers to request a briefing and Q&A which means the evidence is judged without an opportunity to argue the case)
2) Uncertainty about what constitutes, in any case, the amount of evidence that would warrant an "all in" commitment under these very unique circumstances;
3) Some gun-shyness following the hydroxychloroquine debacle;
4) Skittishness about sticking out necks (the reason that the one academic institution that reviewed all the evidence had)

If this works so well, why haven't I read extensively about it in the press? Shouldn't everyone know?

The press doesn't want to report on anything that could raise false hopes.

Until the mainstream medical community embraces the results, you'll see little about it in the press.

The reason here is two-fold:

1) The press assumes that the medical community always gets it right (promptly)
2) The medical community is seriously dragging their heels on overwhelmingly positive evidence (and no negative evidence) because they fear if they make another HCQ mistake, it's over for their credibility. So they are going to wait for Phase 3 data before

making a move. People will die but their credibility will be intact. Even if the chance of a phase 3 failure is <0.01%, they will wait.

What is the evidence supporting fluvoxamine as a COVID treatment?

These studies in the table below all support the hypothesis that fluvoxamine, a closely related drug in mechanism fluoxetine, and/or SSRIs/antidepressants can improve COVID outcomes.

Direct evidence

Study	Type	Effect size (larger is better)	N	p-value
Lenze	Double-blind RCT	100%	152	.009
Seftel	Prospective quasi-randomized RWE	100%	125	.002

The combined p-value for the two randomized studies on fluvoxamine is <1e-4 with a 95% chance that the effect size is 75% or more. The studies are all supportive of the mechanism of action.

The most compelling endpoint is having one or more COVID symptoms after 2 weeks on the drug. In the no treatment group, there was a 60% chance that someone has at least one long-term COVID symptom among the racetrack workers after 2 weeks. In the treatment group, there were no cases. If you do a Fisher exact test on the symptoms, you find that the effect size is 96% or more with 95% confidence, and the **p-value is $< 10^{-14}$**. Nobody has postulated a confounder that can negate that, but if you have one please let me know. It cannot be "healthy patient bias" or placebo effect because none of those effects are strong enough to cause a **p-value of $< 10^{-14}$**.

Anyone who enrolls in clinical trials would be considered a "healthy patient" yet the hospitalization rates for clinical trials mimic the community the people are drawn from. But more importantly, Dr. Seftel reports that the healthy patients uniformly chose "no treatment" because they believed their healthy habits meant they would not get sick and therefore had no need for a psych drug.

It can't be the placebo effect because in the Lenze trial, there should have been no difference in outcomes. So if there is a placebo effect, it is small.

One of the long haul COVID symptoms, anxiety, is directly addressed by fluvoxamine which is an anxiolytic. Therefore, in looking at the effect on long haul COVID symptoms, we need to ignore the column for anxiety. When we do that, it does NOT change the result… 60% still had one or more of the 14 remaining COVID symptoms.

So you have to conclude the doctor fabricated the health records. But that would be easy to validate. And you'd have to believe there was a mass conspiracy to publish only positive evidence on the effects of sigma1, fluvoxamine, SSRIs, etc. for all the other evidence below.

Actual experience of doctors prescribing ivermectin who added FLV as a second drug

Doctor	Number of FLV patients	On IVM+ supplements only	After added FLV
Syed Haider	100	20 patients: 10% hospitalization rate; 20% got worse after starting IVM	0% hospitalizations; 0% got worse after starting the combo
Miguel Antonatos	80	0% hospitalization rate on IVM + supplements (Vit C, D, zinc, quercetin, full dose aspirin)	0% hospitalization rate. "I have seen improved symptoms with fluvoxamine treatment and it works well in long haulers as well. I will continue recommending fluvoxamine as first line treatment along with ivermectin for early outpatient treatment and for selected long haulers."

Additional supporting evidence

Study	Type	Effect size (larger is better)	N	p-value
Hoertel	Retrospective observational	74% for fluoxetine (too few hospitalizations of fluvoxamine to measure)	7,230	.013
Fröhlich	Retrospective observational showing that the only comorbidity that is protective against COVID is depression	36% (but this is effect of ALL antidepressants as a group, not fluvoxamine individually)	6,637	.003
UCSF/Stanford	Retrospective observational using the CERNER database **	18% (antidepressant vs. no antidepressant)	89,034	<.005
TriNetX	Retrospective observational	26.7% reduction in	25,616	n/a

		hospitalization rate for 80 year olds on fluoxetine compared to all 80 year olds*		

*TriNetX data was not compared against a matched placebo group, but against all 80 year olds. But 80 year olds on the drug are much more likely to have worse outcomes than those not on antidepressants typically by around 50%.

**This study is available in the private area of the fluvoxamine repository but is not yet published.

Here is a summary of data supporting the hypothesis that fluvoxamine and fluoxetine are both effective in improving COVID clinical outcomes:

1. Alban Gaultier was the first person to discover that the Sigma1 receptor was important in reducing inflammation in his 2019 paper, "Modulation of the Sigma-1 Receptor-IRE1 pathway is beneficial in preclinical models of inflammation and sepsis." The paper concludes, "Further, we show that the S1R ligand fluvoxamine can enhance survival in mouse models of inflammation and sepsis and can inhibit the inflammatory response in human peripheral blood cells. Collectively, our data show that S1R is uniquely poised to sensitively control IRE1 activity during inflammation." It was this paper which inspired Dr. Angela Reiersen to float the idea of fluvoxamine for COVID on March 25, 2020 in an email to Dr. Eric Lenze leading to the WashU fluvoxamine Phase 2 trial.
2. Randomized controlled trial (RCT), published in JAMA done by Lenze and Reiersen at WashU showing 100% protection from hospitalization due to respiratory distress due to COVID vs. 8.3% rate with placebo. 80 patients got the drug. 72 got the placebo. p=.009. The study basically confirmed the hypothesis generated by the Gaultier lab that fluvoxamine would reduce an "out of control" inflammatory response.
3. Quasi-randomized real-world evidence (RWE) trial published in OFID showing 100% protection from actual hospitalization (vs. 12.5% hospitalization in no treatment group). Also 100% protection from long-haul COVID vs. 60% of the no treatment group had 1 or more symptoms at 2 weeks. 77 employees got the drug; 48 refused the drug. p=.005.
4. If you combine the data of just those two studies, you get a p-value of <.0001 and a 95% confidence of an effect size of 75% using Fisher Exact Test. This is a conservative estimate of the true value since the quasi-randomization handicapped the treatment group.
5. Observational study in a Paris psych hospital showed that the psych patients had 3.5x lower rate of COVID than the staff. Too large to ignore.
6. Observational study published in *Mol Psychiatry* by Nicholas Hoertel showing the protection of SSRIs ranged from a 50% reduction to 75% reduction. All the SSRIs were ordered based on their Sigma1 activation: the higher the sigma1 agonist property of the drug, the greater the protection. Fluvoxamine wasn't included due to too few datapoints, but since it has higher sigma1 activation than the other SSRIs (which is why it was

chosen for the University of Virginia study), it's likely had the patient counts been higher it would have seen to be more protective.

7. German study showing that the only comorbidity that reduces COVID risk is depression (which means effectively, SSRI users). This is a large effect and statistically significant (36% reduction, p=.003).

8. Study at Stanford and UCSF (David K. Stevenson, Marina Sirota, Tomiko Oskotsky, and others) using the CERNER database showed that SSRIs, in particular fluoxetine, was effective in lowering mortality risk by 10% to 30% (depending on the comparison group). In all cases, it was protective. The results for SSRIs as a class were statistically significant [not yet published]. For example, they found that SSRIs vs. non-SSRIs had a highly significant protective effect for mortality (16%, p<.0084). They also found that people on antidepressants in general were more likely to survive than people not on anti-depressants, a truly stunning result that matches what was found the German study. This is stunning because people on antidepressants are much older, have more comorbidities, and in general have 50% worse outcomes than the general public. This result was very statistically significant (<.005). The numbers for fluvoxamine were too small to estimate.

9. TriNetX did a retrospective study on over 700,00 US patients on SSRIs and found that fluoxetine had the greatest protection from hospitalization compared to other SSRIs (confirming the Hoertel study results). The numbers for fluvoxamine were too small to estimate.

10. Getting rid of excess serotonin is likely the primary mechanism of action for why SSRIs are so effective in this disease. Farid Jalali has produced several videos on the role of platelets, serotonin, and SSRIs in COVID (see COVID-19 Pathophysiology: Are Platelets and Serotonin Playing a Bigger Role than we Think? and COVID-19: Platelets, serotonin, SSRIs, and cyproheptadine). Farid presented to Steve Winston's COVID expert group and there wasn't a doctor on the call who wasn't convinced by this. I had comments after the video like, "I thought the evidence for fluvoxamine was strong before this, but this explanation really sold me on the mechanism."

11. Nicolas Hortel believes the FIASMA effect explains why fluvoxamine works so well. It could be all three are at play, explaining the efficacy of the drug.

Additional evidence, arguments, and data:

12. NIH Director Francis Collins has called out the importance of Sigma-1 in COVID. This drug is the top drug to activate Sigma-1: "COVID-19 patients taking ...drugs ... t**hat target SIGMAR1** were **half as likely** as those taking other types of ...drugs to require mechanical ventilation"

13. There are more than 35 papers documenting mechanisms in support of using fluvoxamine for COVID.

14. There are 8 plausible mechanisms of action that are triggered by fluvoxamine that may account for its effectiveness, but the Hoertel paper makes it pretty clear that the main mechanisms are most likely the modulation of serotonin (see Farid Jalali's video) and Sigma-1 activation (see the Gaultier paper) are the main mechanisms.

15. Dr Seftel characterized the drug use as "the most amazing effect he's seen in the 25 years he's been practicing medicine" [Drug Repurposing Research Leads to Potentially Game-Changing Treatment to Prevent Clinical Deterioration in Outpatients With COVID - The Doctor's Channel](#)

16. At the racetrack, the initial acceptance rate of the drug was less than 50%. After 3 weeks, it was 100% because the workers could see the disparity between the two cohorts. They did not need p-values or Phase 3 studies or to listen to their doctor. They could see the obvious difference between the two cohorts because it is a tight knit community. And people who had initially chosen the no treatment group switched over. So now we have an important datapoint: that both neutral and negatively-biased observers decided that treatment was the better route. That's pretty stunning. And we can also see that patient choice reflected in the enrollments: 77 vs. 48. It wasn't because patients believe the doctor since enrollments at the start were skewed against the doctor's advice. So something clearly happened to skew patient selection.

17. We don't know of a single case where the drug failed to return the patient to normal, although hospitalized patients take longer, e.g., an intubated patient took 15 days at 300mg/day of fluvoxamine to lower their CRP to normal levels.

18. Dr. Seftel observed that in most cases, people start to feel better in 24 hours and are pretty much back to normal in an average of 3 days. Nobody in the treatment group got anywhere close to hospitalization. This kind of consistency across 77 patients cannot be explained any other way than that the drug works. Nobody has asked to see this data as of Feb 27, 2021.

19. At the 50mg BID dosing, none of the patients in the Seftel study reported any drug-related side effects which further increases the benefit:risk tradeoff. However, I know of one person who experienced mild nausea while on that dose.

20. Unlike the Lenze study which was remote and had a lot of limitations including self-reported symptoms, the Seftel study was on local patients where the doctor could observe the patient's progress and symptoms directly.

21. Unlike the Lenze study, there was no exclusion criteria for either cohort. People at all stages of the disease were enrolled. This makes it MUCH MUCH tougher for the Seftel study to show a 100% success rate in preventing hospitalization.

22. I haven't heard of any case where taking the drug caused the patient to get worse

23. There were 77 patients in the Seftel treatment group (not 65 as listed in the table in the paper). This was due to an IRB rule because the 65 number was previously publicly disclosed. Most all the people who report on the paper think the number is 65, showing very few actually read the paper itself.

24. Nobody in the Seftel study (who were allowed to switch groups at any time) switched from the treatment group to the no treatment group whereas 8 people in the no treatment group got sick and decided to switch over to the treatment group because they were so sick. These cross-over patients were not counted twice, they were treated as if they enrolled in the treatment group originally. This means that the 77 patients in the treatment group were not randomly selected, but disadvantaged with 8 known sick patients.

25. In the Seftel study, examination of age, ethnicity, comorbidities, etc. shows the treatment group was much more likely to be hospitalized than the "no treatment group". Therefore the resulting p value (.005) is conservative since they got the short end of the randomized stick. Now when you add those 8 cross-over patients to the mix, it becomes extremely difficult to argue that Seftel simply got "lucky" with a healthier cohort.

26. The most common complaint in the treatment group in the Seftel study was "why won't you let me get back to work?"

27. Brain fog very quickly resolved in the treatment group in the Seftel study (within avg 3 days), again showing the drug works consistently and reliably.

28. The drug has been used on inpatients, patients on high flow oxygen, and intubated patients. In every case, I am aware of, the drug has reversed the COVID inflammatory response.

29. The effect size in both trials was 100%.

30. The Seftel study was quasi-randomized: it was based on the choice of the person. The people basically thought whether they would get sick or not or used their own algorithm. This is random because we couldn't predict in advance what each patient would choose, and the patient couldn't know in advance whether they would get very sick or not. Normally, there is a healthy patient bias where the healthy patients choose the drug and trust the doctor. In this case, that wasn't the case as the treatment group not only had higher comorbidities, but when people failed in the no treatment group, they switched over to the treatment group. Bottom line: the treatment group got a cohort that was, as far as anyone could tell, at higher risk for a poor COVID outcome. This makes the 0% vs. 12.5% disparity between the groups even more pronounced than if this had been a truly randomized study. However, the Seftel study was open label so there can be both patient and doctor bias. So that must be factored in as well. This is why there are multiple data points that we rely on to see an effect, not just a single study.

31. Fluvoxamine is a safe drug which has been marketed for over 37 years and with no reported deaths of overdose in the literature, making it far safer than over the counter drugs such as Tylenol which kills hundreds of people per year.

32. The Hoertel paper showed that the protection was ordered by Sigma1 activation. Even though fluvoxamine wasn't included in the analysis, one can logically conclude that higher sigma1=greater protection from the data in the study. Since fluvoxamine has higher sigma1 activation than any other SSRI, it's reasonable to assume that it has the highest protective effect. However, this doesn't necessarily mean that fluvoxamine is the best choice; it likely is, but we'd need further studies with other SSRIs to confirm this.

33. In France, Nicolas Hoertel reports that the percentage of patients with COVID who are on an SSRI is 5%, but the overall rate of SSRI use in France is around 20%. This is not due to an age skew either, because most SSRI users are older people (the older you are the more likely you are on SSRI). Therefore, we would have expected the rate of SSRI users in the hospital would be higher than 20%. So this implies a 75% effect size which is consistent with what they found when they analyzed the hospital data.

34. Dr. Drew tweeted that fluvoxamine changed his life as a COVID long-hauler.

35. None of these are "cherry picked" anecdotes. All the anecdotes are positive and there are many more that were not included, all positive including doctors I know who have

tried this off-label and reported the results to me. But I also had direct experience with 3 of my friends. So I didn't have to trust any of the studies myself, simply my own eyes. One of my friends, a very healthy Lt. Col. in the Army, was so sick with COVID he hadn't been able to get out of bed for 5 days with no energy. He started feeling better within 2 days of starting the drug and within 7 days, he was mentally back at 100% and physically was then able to walk and exercise, and eat (he had lost 20 lbs at that point). He says all of his COVID symptoms resolved before he finished the 14 day course for the drug. He thinks they should promote to other members of the Army and is frustrated the Army isn't allowed to do that.

36. ICU intubated patient anecdote: This shows a direct cause-effect relationship that is very unlikely due to luck… as soon as the drug was given the patient improved dramatically, as soon as the drug was stopped the patient very rapidly declined and died. This is not proof of efficacy, but just pointing out that it appears more likely than not that fluvoxamine is effective even after a patient has been hospitalized.
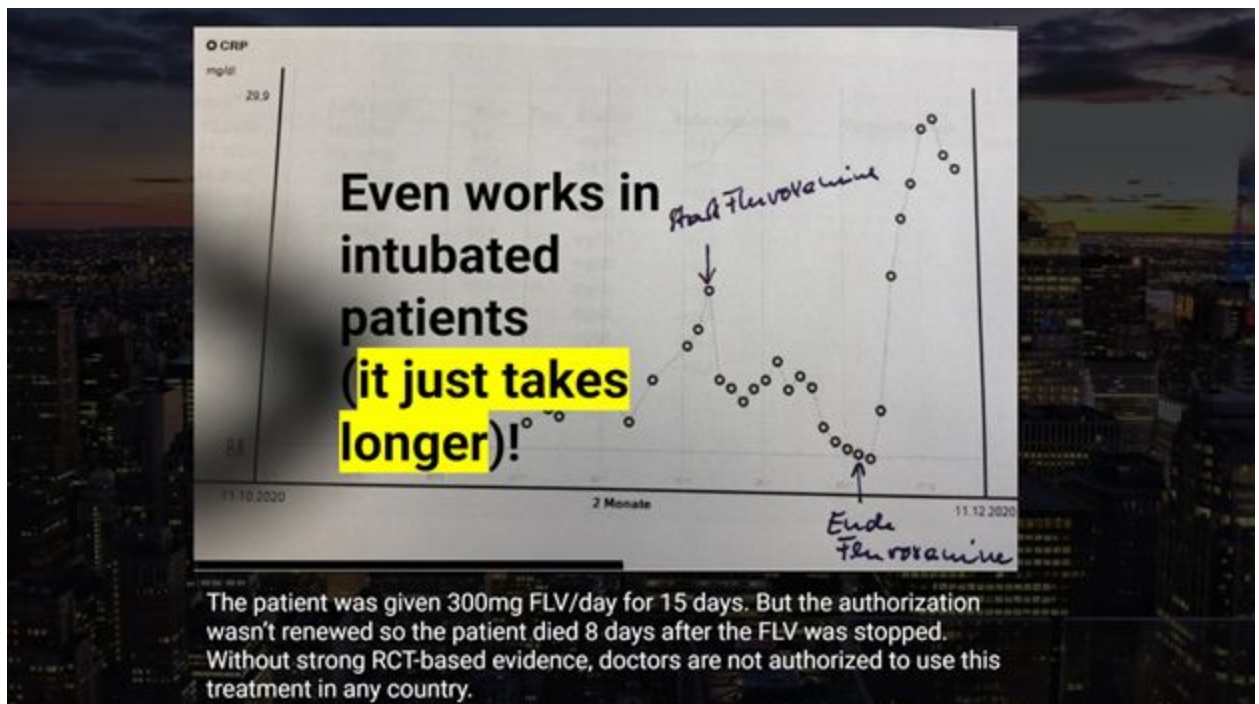
Notes:
1. In the Lenze Phase 2 trial, one person in the treatment group did get hospitalized for dehydration due to diarrhea. The drug did not cause the diarrhea, it was a pre-existing symptom. Therefore using the drug did not increase the hospitalization rate (and was given too late for this patient to avoid the hospitalization).
2. Nobody I talked to could come up with a single drug with comparable evidence where the Phase 3 trial failed to show the drug was effective (we already know it is safe since FDA approved). Since there have been well over 40,000 such Phase 3 trials (since there are 20,000 FDA approved drugs and half the phase 3 trials fail), the chance of a failure of the Phase 3 trial is very small (<0.005%).

3. [You can view the Seftel patient data yourself here](#). If you sort by patients who accepted treatment, you'll see what I'm talking about. There is a 60% chance that a patient has 1 or more COVID symptoms after 2 weeks. But nobody in the treatment group reported any COVID symptoms. The chance that this happened because we got "lucky" in our choice of patients is very small: less than 10^{-14},

In short,
1. there was no evidence of any type that would lead us to believe that the placebo/no treatment is superior to the treatment in any phase of the disease. Not even a single anecdote.
2. There was no evidence that showed that the drug didn't have a protective effect
3. There was no evidence that the drug caused harm
4. If there is truly no effect, it would be very hard to explain all the points above.
5. There was no inconsistent evidence.

Therefore, it comes as no surprise that fluvoxamine 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. They also recommended it be added to the NIH COVID-19 Treatment Guidelines.

Every individual piece of evidence has flaws, it is the sum total of all the evidence that provides a consistent sign of efficacy and safety.

See [Fluvoxamine public data repository](#) for all the evidence.

Shouldn't we wait for more evidence like the Phase 3 study results before doctors start prescribing this drug? Or wait for this to be on the NIH guidelines?

No. Mathematically, you can show that more lives will likely be saved by prescribing now than by waiting. There is no evidence that the drug could cause more lives to be lost.

Some people claim that there isn't enough evidence yet. But the problem with that statement is that **none of the people who make that claim have looked at all the evidence that is on the table today and none of them can explain it all away**; they make that statement based only on the very limited data they're aware of.

As of March 7, 2021 not a single scientist (or anyone else for that matter), has requested access to the data for the key Seftel study that confirmed the Phase 2 DB-RCT result. So the argument that "we need more data" is basically a hand-waving argument that 1) the only data worth considering is DB-RCT data published in a peer reviewed medical journal, and 2) all other data

points are useless and should be totally ignored. That may be a nice "low-risk" way to evaluate evidence but it is completely inappropriate during a pandemic.

As I noted in the evidence section above, if you only look at a subset of the evidence, the data is compelling. When you look at all the evidence (including understanding the mechanisms of action), most people would conclude we have evidence well beyond any reasonable doubt.

I've collected a lot of the evidence in the [Fluvoxamine public data repository](#) to make that easier.

How many of the people who say "we don't have enough data" have read the Gaultier paper? How many of them can describe the pathway by which Sigma-1 influences inflammation? How many of them have viewed Dr. Farid Jalali's videos on platelets and serotonin and can explain to you how that works? How many of them can point out an error in Dr. Jalali's videos? How many of them can explain how none of the 77 patients in the treatment group were able to have no symptoms after 2 weeks.

To the best of my knowledge, nobody has produced any evidence showing fluvoxamine doesn't work. Not even in even a single case. Or a plausible explanation that explains why there is really no effect here. Note they may do this in time, but the lack of counterevidence means we consider this drug very promising.

So by having a blind eye to the data on the table, it's really easy for people to say, "We need more data." It's a safe answer that tragically is costing the lives of hundreds of thousands of people.

People have advised me not to upset the FDA or NIH and just wait months until the trial finishes. I respectfully disagree--doing so would contribute to what is perhaps the largest needless loss of human life in our history. I do understand that few in the industry would want to speak out as this could jeopardize their NIH funding, etc.

Evidence that we have sufficient evidence to make an emergency call includes:

1. A key opinion leader (KOL) group from NIH, CDC, and leading academic institutions looked at **only** the two randomized trials and the mechanism of action and voted by more than a 2:1 margin for physicians to bring up fluvoxamine with patients in a "shared decision making" process.
2. Vikas Sukhatme, Dean of the Emory School of Medicine, [called for doctors to talk to their patients about fluvoxamine](#) and listed [fluvoxamine as the most promising repurposed drug to treat COVID outpatients](#).
3. An [op-ed published in a medical journal in Croatia](#) independently looked at the fluvoxamine evidence, did the research on the mechanism of action, and concluded:
 Fluvoxamine is **among the very few drugs** that have demonstrated therapeutic potential and safety profile in a double-blind, randomized clinical trial in humans. In addition, the **rationale** behind its immunomodulatory and protective effects in COVID-19 **is pathophysiologically sound**. When comparing the cost of fluvoxamine treatment

with the current treatment options, such as remdesivir, bamlanivimab, and/or casirivimab/imdevimab, a full 15-day treatment with fluvoxamine would respectively be 70, 37, or 45 times more affordable per patient. If used early in COVID-19 outpatients, **it could prevent many hospitalizations**, thus reducing patient mortality, improving allocation of healthcare resources, and creating significant savings in health care costs. Health professionals and decision makers should become aware of the therapeutic potential of fluvoxamine for COVID-19 patients.

It will take months for the Phase 3 trial to complete (or a DSMB review to happen). Should we wait? As of February 27, 2021 there were 300 people enrolled in the trial, but only 240 can be used and there were just 9 people who met the hospitalization criteria. So we'll be waiting many months to get higher numbers.

The January 22 KOL meeting suggests not. They only heard a subset of the evidence (just the 2 trials and the mechanisms of action) yet they overwhelmingly (by more than 2:1 margin) "shared decision making" for the drug based on the evidence now on the table.

We have sufficient evidence on the table today to make a determination because the benefits >> risk.

If our objective is to minimize the number of deaths (like our own), we should prescribe the drug(s) with the best evidence now.

If our objective is to minimize the chance that the NIH is wrong, we wait. This is of course the choice of the NIH and the medical community.

Which is more important to you personally? Saving your life? Or preserving their reputation?

I would argue waiting is not in the public interest. It will not save lives in the interim period until a phase 3 trial is complete.

Therefore, it would be reasonable for the NIH Guidelines committee to make a temporary emergency recommendation which is later re-evaluated when the Phase 3 trial reports out. That will maximize the number of lives saved in the interim.

Please see the full argument on Fluvoxamine - The backstory for more details.

NIH Director Francis Collins compared fluvoxamine with hydroxychloroquine on CNN. Is he wrong?

He's being extremely cautious, which may make sense from a policy perspective but not from a personal decision-making perspective.

In a CNN interview with Sanjay Gupta, Dr. Collins said:

> "Fluvoxamine looks promising right now, but it might be where hydroxychloroquine was a year ago and doesn't work. So I want not to sort of open the door for everybody to start using it. I want to say, 'Let's test that. Let's find out the answer.' "

To be clear, HCQ never had such good data as fluvoxamine. Fluvoxamine is not an HCQ, so this is an unfortunate comparison.

In a nutshell, the differences are in:

1. Quality of the data (e.g., DB-RCT published in JAMA, quasi-randomized study published in OFID, both studies "Editor's Choice" papers)
2. Consistency of the results (every study shows positive impact)
3. At least two very clear mechanism of action (S1R and serotonin uptake)
4. Effect size

With fluvoxamine, we have a very clear mechanism of action that was identified in 2019, it was confirmed in 5 independent retrospective studies, a double-blind placebo controlled study published in JAMA (where after 180,000 views, NOBODY has discovered any confounders that would negate the results) and confirmation in a real world evidence trial by a top researcher published as Editor's Choice in a top peer reviewed journal. So the data is high quality, it is all consistently positive, and it all makes sense. There are not even any anecdotes that it doesn't work.

All the evidence we know about says benefit >> risk.

And federal law requires the FDA to consider real word evidence in making decisions about new indications for existing drugs. But the FDA could argue that reliance on that data is unnecessary since a phase 3 trial will only take 4 more months to complete, so there is no need to consider RWE because there is an alternative (and the 200,000 people who would die in the meantime are not really a problem).

Wouldn't it be great if public health officials were to say, "We should prioritize saving lives now. This is the drug with the best evidence today, where the only rational explanation that fits all the data is that the drug works, and we should start using it now and if it turns out not to work, we should switch to the next most promising drug. But not making a decision on a drug which statistically is more than 99.99% certain to work based on the studies to date (as shown in the evidence section) would be putting preserving our reputation ahead of saving lives. Lives are more important."

I predict that the FDA will ignore everything except the double-blind RCT (as if it never happened), and then argue there isn't enough evidence to make a decision. I really hope I'm wrong.

What is the evidence for ivermectin?

It's extremely strong.

See the FLCCC website and COVID-19 studies of Ivermectin.

I'm not aware of any randomized controlled clinical trials with ivermectin where it didn't show some positive effect. This video done by WHO consultant Andrew Hill summarizes the evidence. Or watch this short video done by independent researcher Tess Lawrie.

The BIRD Recommendation on the Use of Ivermectin for Covid-19, where a large group of international experts looked at the evidence, the effectiveness of ivermectin in reducing hospitalization is around 85% or so, and on-par with fluvoxamine which I think will be comparable or better (based on the evidence we have today). The conclusion did not call for further study. Instead, "*the group issued sweeping recommendations for the **immediate global use of ivermectin**.*" See the full BIRD report here.

You can pick a healthy cohort and have a very long follow up period and show ivermectin has less of a benefit in terms of time to resolution, as was done in this ivermectin DB-RCT study just published on March 4, 2021 in JAMA. But that doesn't negate the other studies it simply adds to the body of evidence showing it makes less of a difference in healthier patients (and you'd need more patients to show a statistically significant difference. Dr. Angela Reiersen wrote: "I think it is unlikely for anyone to find a significant difference between treatment groups in an RCT with time to symptom resolution as an outcome measure. Symptoms are too heterogeneous, can fluctuate, and can tend to linger quite a while even after a person is mostly recovered. There was a hint of a difference between groups on some other outcomes that were more closely related to clinical deterioration, but for those outcomes they did not have enough statistical power to show whether there is a true difference."

Here's an EU-based recent double-blind placebo-controlled study showing positive impact for ivermectin. They made it available over the counter in Bulgaria and there are long lines for the drug.

Here's a summary of the double-blind trial in Israel: Randomized Double-Blinded Clinical Trial at Sheba Medical Center: Ivermectin Materially Reduces COVID-19 Viral Shedding

This website summarizes the evidence for ivermectin so far.

Ivermectin has been hampered because each study uses a different dosing regimen and different endpoints; some used inadequate dosing to show a significant benefit, so it's important to look at studies with adequate dosing and timing in order to gauge the effectiveness.

The ivermectin data has been greeted with skepticism by the CETF SAB, internal review at one med school I'm aware of, the NIH COVID Guidelines Committee, the IDSA, and by the drug manufacturer (Merck). While I strongly disagree with those assessments, it is what it is today. **But this is a perfect example of how the institutions we are supposed to trust may not be giving us the best information.**

It's difficult to explain the evidence in plain sight if ivermectin didn't work. Peru is a perfect example. Every time ivermectin is introduced to a region, hospitalization rates go down dramatically. Every time the drug is revoked in a region, hospitalization rates go up dramatically. No one has been able to explain this (other than as a result of the drug).

Are there any drugs that are as good as or better than fluvoxamine to treat COVID early?

Yes, ivermectin might be as good as or even better than fluvoxamine.

There are 4 reasons we could argue that fluvoxamine is at the top of the list of promising drugs:

1. To our knowledge, no one has reported anything else with 100% effect sizes for preventing hospitalization from COVID respiratory symptoms.
2. The long-haul COVID symptom data documented in the Seftel paper is extraordinary (0% vs. 60%).
3. Vikas Sukhatme, an expert in repurposed drugs, believes that fluvoxamine is at the top of the list along with ivermectin. See the table in his op-ed [A Call To Action: Immediate Deployment Of Select Repurposed Drugs For COVID-19 Outpatient Treatment](#)
4. The closest competitor to fluvoxamine would be ivermectin, and the ivermectin effect may actually be stronger than fluvoxamine. We don't know because they haven't yet been tested side by side.

Fluvoxamine has some antiviral efficacy because it is a FIASMA, but primarily functions as an anti-inflammatory when used with COVID. This is significant because the main damage in COVID is done by the inflammatory response rather than the virus itself. Our immune system (most of the time) eventually defeats the virus just like we recover from influenza and common colds.

It is certain that TMPRSS2 inhibitors (camostate, [bromhexine](#), and [proxalutamide](#)) will emerge as totally complementary treatments with fluvoxamine. The published data on these drugs is spectacular.

Similarly interferon lambda could emerge as a very potent treatment as well since it can stop the virus early too. This would be a complementary treatment as well. There were divergent studies, but it could be due to patient selection bias since for healthy patients it didn't make a difference.

Finally, ivermectin, when properly dosed, may turn out to be as good as or even better than fluvoxamine.

Time will tell. It's nice to have more than one option.

Does fluvoxamine help with COVID symptoms or just hospitalization risk?

The Seftel study showed it dramatically reduced COVID symptoms.

There are 15 long-haul symptoms that were used in the Seftel study.

In the treatment cohort, no patient had any of the 15 symptoms after 2 weeks, whereas 60% of the patients in the no treatment group suffered from one or more symptoms after 2 weeks and 29% had 4 or more symptoms!

The 15 symptoms used in the Seftel study are:

1. persistent body aches, muscle or joint pain
2. brain fog, difficulty concentrating, or memory challenges
3. persistent, intermittent non-productive cough
4. fatigue
5. Headache
6. intermittent heart palpitations/tachycardia
7. insomnia
8. persistent anxiety
9. dizziness
10. diarrhea
11. elevated temperature
12. episodic chest tightness, pressure, or pain
13. inability to exercise
14. chills or sweats
15. shortness of breath or difficulty breathing

How does fluvoxamine work? What are the 8 mechanisms of action?

The virus is like a fire, fluvoxamine is like water. The sooner you pour the water on the fire, the faster you recover and the less damage is done.

So no matter what your symptoms, no matter how old you are, the sooner you put the water on the fire, the better the outcome.

More technically, the virus attacks the platelets which release serotonin which causes havoc. SSRIs like fluvoxamine vacuum up the excess serotonin.

The second mechanism is Sigma1 receptor activation. Fluvoxamine is a strong Sigma1 agonist. The endoplasmic reticulum is somewhat like a factory for building proteins into their final form. In general, chaperone proteins can assist in proper folding and maturation of other proteins that are being produced in the endoplasmic reticulum. One S1R function is to act as a chaperone to facilitate the maturation of certain proteins. But it also has other functions, such as regulating the actions of other ER proteins (including a protein called IREI that is involved with the ER stress response and inflammatory responses). IRE1 is a protein that can activate another protein called XBP1 which can then promote the production of cytokines. In the presence of a S1R agonist, S1R can essentially turn off IRE1, so IRE1 will not activate XBP1, so that the cytokine production will decrease.

So any SSRI gets you a 50% reduction. The Sigma1 activation gives you another 50%, so that's why fluvoxamine gives you the strongest protection vs. other SSRI… it's the Sigma1 advantage.

Bottom line: Fluvoxamine is like "pouring water" on the "fire."

See Fluvoxamine public data repository for more information (see the "mechanisms of action" directory and the serotonin directory) on all 8 mechanisms of action.

What do infectious disease experts think?

That fluvoxamine is promising.

A key opinion leader (KOL) panel, people from NIH, CDC, and top academic institutions, met on January 22 to review the fluvoxamine evidence. **They voted afterward to support "shared decision making" TODAY** (between doctor and patient) **on fluvoxamine by more than a 2:1 margin** (note only 20 of the 30 cast votes and the vote was 11 in favor to 5 against; 4 had no opinion).

They were voting in their PERSONAL capacity, and NOT in their OFFICIAL capacity. So even though the NIH Guidelines panel will wait for a phase 3 trial because of the "public trust" issue in their recommendations, the doctors personally overwhelmingly agreed that the weight of the evidence merited a discussion today between doctors and patients (and NOT waiting for more data).

What this means is if you are interested in maximizing your life expectancy, you must look outside the CDC guidelines.

Have you applied for an EUA?

Yes, we filed our [initial EUA paperwork with the FDA](#) on January 29, 2021.

We hired [Proxima CRO](#) to do the filing. The entire team has been really awesome work with.

Our Pre-IND number is 154615.

The FDA told us it will take at least 5 weeks to consider the data we submitted before we will hear anything at all back from them.

It is notable that it took an key opinion leader panel **only 1 hour** to make a determination based on the evidence, and it took an FDA advisory panel only a few hours to unanimously approve the J&J vaccine, yet it takes the FDA more than 5 weeks to answer questions as to whether the evidence we have today is sufficient to support an EUA.

Even though every piece of evidence we know about shows the benefits>>risks, we fully expect the FDA to say in early March that they will not make a decision until there is data available from a large US-based DB-RCT. This is because they don't want to make a mistake. The loss of lives in the meantime is not factored into their decision. There are no consequences if they are wrong; they can argue that they are just being careful.

Ivermectin is another example. The FDA essentially told the manufacturer of ivermectin that they wouldn't approve ivermectin until there is a large US-based Phase 3 trial of Ivermectin. They FDA is being cautious with their reputation here, but the reality is that they are degrading their credibility. There was a very large independent international panel that reviewed the ivermectin data according to medical standards (the [BIRD report)](#).

This is a broken system, in my opinion. If you are drowning and someone throws you a life preserver that was only tested 10 times (and worked every time), they would never say, "well I don't think this life preserver was tested enough times and I don't want to throw you something that might not work" You would simply throw them the life preserver with the best evidence on the table at the time and if that didn't work, they'd throw you the life preserver with the next best evidence.

Why is this happening? A prominent doctor wrote me:

> I think the committees in regulatory agencies everywhere (not only at the FDA) are full of not practicing health professionals (scientists, academics, pharmacists, economists, ...), who have never looked death in the eyes, never saw a patient struggling to breathe and have never been faced with a desperate urgency to somehow treat their patients and try to save their life. Such committees are also susceptible to political pressure as can be seen from the CQ/HCQ example, so it makes sense (to them), in a way, that they should now tread very carefully with regards to the fluvoxamine EUA.

Is there a Phase 3 clinical trial for fluvoxamine?

Yes. You can enroll from the comfort of your home and it's free.

You must be at least 30 years old and enroll within 7 days of your first COVID symptoms.

www.stopcovidtrial.com

Have other SSRIs been tested?

Yes. See Association between antidepressant use and reduced risk of intubation or death in hospitalized patients with COVID-19: results from an observational study.

Observational retrospective data from TriNetX and UCSF/Stanford study showed fluoxetine (Prozac) appears to perform even better than fluvoxamine, but this has not yet been tested in randomized trials.

Sertraline is the least effective SSRI because it is a Sigma1 antagonist, so it blocks the receptor from being activated.

The order of SSRIs in terms of Sigma1 activation (i.e. agonist) is described in this paper: fluvoxamine > fluoxetine > escitalopram > citalopram >> paroxetine.

Sertraline is the least effective SSRI against COVID because it is a Sigma1 antagonist, so it is extremely effective in blocking the receptor from being activated which means if you are on this drug and you get COVID, you would have the greatest incentive to switch to fluvoxamine.

In the Hoertel paper, the effect size of the drugs lined up exactly based on Sigma1 activation. That's remarkable because that only has a 1 chance in 120 to happen by luck and when they wrote the paper, they had no clue whatsoever about the Sigma1 effect, so they couldn't fudge the numbers.

The Hoertel paper didn't look at fluvoxamine because they had only 1 patient on it who was hospitalized, so not enough data.

This is a problem in general with fluvoxamine: there are too few records of hospitalized patients to analyze for retrospective studies.

You might assume that is because it works so well, but it is market share related. Eli Lilly is such a marketing powerhouse that their drug advertising for fluoxetine (Prozac) has basically taken market share from fluvoxamine, despite the fact that many people believe that fluvoxamine is the SSRI with the lowest side effect profile. Lilly doesn't advertise Prozac anymore because Prozac has been off patent since 2002.

Who else is writing about fluvoxamine?

See News articles and op-eds about fluvoxamine.

What's the probability we save more lives if we start prescribing fluvoxamine?

100%. Fluvoxamine doesn't kill people, the virus does. Fluvoxamine won't add to the death toll; it can only make it smaller.

Where can I find out more information?

1. Steve Kirsch Home page (evidence summaries, press articles, presentations, op-eds, ….)
2. Fluvoxamine public data repository (hours of source information on fluvoxamine)
3. Steve Kirsch's answer to What is the current treatment for Covid-19? (other COVID treatments backed by scientific evidence)
4. Fluvoxamine - The backstory (which talks about deficiencies in the current system and how we could improve it)

See a question not answered here? Email me at stk@treatearly.org