

Horowitz: The indefensible approval of Pfizer and Merck drugs compared to the snubbing of ivermectin

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Later this week, the FDA [plans to approve](#), as the first outpatient COVID drugs, therapeutics that are extremely dangerous and unproven, even as the agency goes to war against cheap, safe, and proven drugs with a track record of no serious adverse events. The approval is as shocking as it is revealing and should serve as a warning to those who don't believe the FDA would approve vaccines that aren't safe and effective.

We already know that every drug the FDA has approved so far for inpatient treatment has an FDA "black box warning" for serious adverse events. At present, the only approved drugs in-patient are remdesivir, baricitinib, and tofacitinib. None of them have demonstrated any efficacy over a year of their use, and remdesivir [is known to cause liver toxicity](#) and renal failure. Baricitinib (brand name Olumiant) has an [FDA black box warning for blood clots](#), of all things! Tofacitinib (brand name Xeljanz) [has a black box warning](#) for "serious infections and malignancy." Now, let me introduce you to the first candidates for outpatient treatment: Merck's molnupiravir (brand name Lagevrio) and Pfizer's Paxlovid.

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I've already written extensively on molnupiravir. Even the FDA advisory committee admitted that the drug poses a risk of birth defects, is mutagenic, has a dangerous mechanism of action, and was never studied for carcinogenicity, and its second-phase trial showed greater "efficacy" in the placebo group than the trial group. Even the mainstream media has warned that the drug really is not up to snuff, yet shockingly, the FDA is set to give it approval, as if basic safety and efficacy facts no longer matter. This move in itself, in conjunction with what we know about the approved inpatient drugs, should tell you everything you need to know about the juxtaposition of the vaccine approval to the war on ivermectin and hydroxychloroquine and the refusal to approve or encourage the use of numerous other safe and effective drugs.

But what about Pfizer's Paxlovid? Doesn't that have a safer mechanism of action, similar to that of ivermectin? And wasn't it proven 89% effective in reducing mortality and hospitalization?

Efficacy of Pfizer's Paxlovid

Unlike Merck's drug, which has a known dangerous mechanism of action as a nucleotide analogue, Paxlovid is more of a defensive drug as a 3CL protease inhibitor. Dr. Ryan Cole, a clinical and anatomic pathologist who has studied the replication process of SARS-CoV-2 and its treatments in more depth than almost anyone on the planet, explains the mechanism as follows:

When COVID replicates inside our cells, part of the process is formation of a long string of amino acids within our cell's ribosome (hijacked by the virus to use as a protein manufacturing site), forming a chain of proteins called a polyprotein. In order for the proteins to form the parts of the virus,

this chain must be clipped and broken down into the viral protein parts. An enzyme called a protease does this cutting and clipping. Paxlovid is a protease inhibitor, meaning it binds to this enzyme “scissors” and keeps the cutting from happening, so the virus cannot reassemble.

Sounds terrific, right?

Here’s the problem. Do you know what else is also the most effective protease inhibitor on the market? [Ivermectin](#). And it also has at least 19 other mechanisms of action, which include anti-coagulant (inhibits CD147 receptor binding) and anti-inflammatory (decreases IL-6 and other inflammatory cytokines) modes of action. Paxlovid has none of these mechanisms. So why would we rely on an expensive drug with one of ivermectin’s 20 mechanisms of action – [yes, 20](#) – that does not have an established safety profile when we can use an off-patent drug with the safest profile imaginable and mechanisms that work even in advanced stages? Also, Cole explains that because Paxlovid only has one mechanism of action, “viruses can eventually mutate around this mechanism.” Dr. John Campbell [offers a superb presentation](#) on the similarities and differences, showing why ivermectin is superior to Paxlovid.

Consider that earlier this year, a study in [Nature](#) of dozens of potential protease inhibitors against SARS-CoV-2 found ivermectin to be the only one to fully bind the 3LC enzyme. Out of 13 off-target drugs tested, “only ivermectin completely blocked (>80%) the 3CLpro activity at 50 μ M concentration.”

So now that we are championing this mode of action, why wouldn’t we exalt the cheaper, more established medicine that is also an anti-coagulant and anti-inflammatory and that has shown the ability to [turn around even some patients on ventilators](#)? At best, Paxlovid would likely

only work during the first three days of onset of symptoms, which is how the trial was conducted.

Moreover, as anyone who treats this virus will tell you, Delta has been a game changer. This virus is so aggressive and novel in the way it enters the cells and replicates, there is no drug alone without adjuncts that will achieve 89% success against mortality. It's complete bull. Ivermectin likely achieved that level of success in the original strain, but with Delta, even the most ardent supporters will tell you it needs several adjuncts to keep more people out of the hospital. There is no way Paxlovid with one mechanism of action can achieve 89% success when the king of 3CL protease inhibition can't do that with several other modes of action.

Now, we all hope that Omicron will completely vanquish Delta and thus will be easier to treat. But why not go with a drug (and more importantly, combo of several drugs) that is safer and has more mechanisms of action? As Cole warns, "A protease inhibitor is only useful when used early when the virus is replicating. We know the Delta and Omicron variants replicate very quickly, so protease inhibitors are only potentially helpful in the first few days of infection."

Paxlovid contains a dangerous AIDS drug

We haven't even discussed the safety problems. While the new drug itself in Paxlovid, although unproven, is probably not as dangerous as Merck's drug, the media has failed to inform the public that it's combined with AIDS drug, ritonavir.

Why is it combined with the AIDS drug? According to Cole, "In order to work, the protease inhibitor has to last long enough in the body. Another protease inhibitor, ritonavir, usually part of an HIV regimen, is used to prevent the rapid breakdown of Paxlovid."

Incidentally, in the [Nature study](#) of various antiviral drug efficacy against 3CL protease binding, ritonavir had less than 20% success, while ivermectin achieved 85%.

Typically, whenever a pharmaceutical company produces a combo drug, it must conduct separate clinical trials on each component and demonstrate to the FDA why the combination is necessary. But in the pandemic, all rules have been thrown out the window when it comes to Pfizer. No such trial was conducted. Why is this a problem?

According to Cole, “Ritonavir is not without its side effects, which can include life-threatening liver inflammation, pancreatitis, and heart arrhythmias. It may also cause nausea, diarrhea, dizziness, confusion, high cholesterol, high blood sugar, stomach or intestinal bleeding, numb hands and feet, a skin rash, as well as countless other side effects.” The FDA has issued [a black box warning](#) for many serious contraindications with ritonavir.

Can you come up with a non-sinister explanation as to how our government will not only approve, but purchase this untested and dangerous product while declaring war on its broader, safer, cheaper, and more established counterpart? If you do, I have some remdesivir to sell you at \$3,000 a pop, but unfortunately it won't cover your kidney transplant.