Here is an overview of Ivermectin mechanisms of action. I state the mechanism and try to put it into somewhat lay terminology, and have attached references, as well. I hope this helps.

Overview of Ivermectin mechanisms (these mechanisms are viri-static, not viricidal, meaning the virus is still present but can't do what it wants to, but the virus isn't necessarily "killed" by ivermectin, but rather neutered. More importantly, Ivermectin is anti-inflammatory so it is effective preventatively, in early infection, in hospitalized mid infection and late, as well as in long haul symptoms in post Covid patients.

1. Inhibits binding at ACE2 an TMPRSS2 keeping the virus from entering our cells
2. Blocks alpha/beta importin (the virus cell taxi) keeping it from gettin to the nucleus
3. Blocks the viral replicase zipper (RdRp)
4. 3-Chimotrypsin protease inhibition (keeps the virus from assembling)
5. Ivermectin strengthens our natural antiviral cell activity by increasing our natural interferon production (this Counters SARSCOV2 activity which inhibits cellular interferon)
6. Decreases IL-6 and other inflammatory cytokines through NF Kappa Beta downregulation, taking the patient from a cytokine storm to a calmer inflammation profile.
7. Binds NSP14 necessary for viral replication and blocks it (equals less virus).
8. Most important mechanism is inhibiting binding to CD147 receptor on red cells, platelets, lung and blood cell lining. Ivermectin keeps the virus from binding here and decreases deadly clotting.

Detailed view

1. Ivermectin hinders binding of SARS-COV2 spike protein at the ACE2 receptor . Ivermectin binds not only to the virus spike, but also to the ACE2 receptor (yes, more strongly than remdesivir).This is the primary receptor on our cell surface where the virus binds and then gets gulped into the cell. If the virus can't bind, it can't get in. If it can't get in, it can't replicate. Both the key and the lock are altered and don't work together in the presence of ivermectin.

We have heard much about ACE2 but TMPRSS2 is a serine protease that is needed on the cell surface to prime the Spike protein. Ivermectin inhibits this.

https://pubmed.ncbi.nlm.nih.gov/32871846/

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7996102/

https://www.frontiersin.org/articles/10.3389/fmicb.2020.592908/full

https://www.cell.com/cell/pdf/S0092-8674(20)30229-4.pdf

2. It binds to the alpha/beta importing and saturates it. This is the "taxi/uber" the virus uses to ride into the cell to arrive at the area where it would replicate. So ivermectin essentials takes up that "taxi/uber" seats so the virus has trouble getting a ride in to where it needs to be to copy itself.

https://pubmed.ncbi.nlm.nih.gov/22417684/ Alpha/Beta importin ivermectin mechanism information we have known for almost a decade

3. Ivermectin binds and inhibits the viral RdRp (RNA dependent RNA polymerase). Basically this is an enzyme the virus needs to activate to replicate itself, essentially zippering back and forth. So ivermectin ends up being that annoying piece of fabric stuck in that zippering mechanism.

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7996102/

4. After the virus copies itself into its long form of all of its protein parts, enzymes clip it so those proteins can assemble into new virions. There are 11 sites on this long protein string that are clipped by the enzyme 3-Chimotrypsin protease. Ivermectin inhibits this protease by 85-100% forcing the virus replication to halt, because it cannot become its constituent building blocks.

https://www.nature.com/articles/s42003-020-01577-x

5. As important is the viral inhibitory mechanisms, are the immune modulation mechanisms. As mentioned in 2 above, the virus rides into the cell on alpha/beta, arrives in the nucleus and shuts down our interferon production. Interferon of many types are produced by our body. SARS-COV2 selectively shuts down this interferon pathway and allows itself to replicate and high jack the body's mechanisms more quickly. As in 2, ivermectin blocks the virus from getting to this point. It does this for countless other viruses as well.

https://pubmed.ncbi.nlm.nih.gov/27973612/

https://www.nature.com/articles/s41429-020-0336-z/tables/1

6. Oh yes the cytokine storm. Ivermectin inhibits many inflammatory cytokines including the prominent one IL-6. Also IL-1B, IL-10. Anti inflammatory effect by down regulating the nuclear transcription factor Kappa-B and mitogen activated protein kinase activation pathway. Basically this means it tunes down inflammatory cytokines such is IL-1beta and IL-10 as well as tumor necrosis factor alpha. In a nut shell it calms the immune system and decrease the cytokine storm in acute Covid patients but also shows effect in long haul patients suffering from a cytokine "trickle"

https://link.springer.com/article/10.1007/s00011-008-8007-8

7.Binds to NSP14 (non structural viral protein 14) ribonuclease which is necessary and critical for SARS1 and 2 and MERS to replicate. Ivermectin has a much stronger binding to this site than remdesivir and inhibits viral replication.

https://journals.asm.org/doi/full/10.1128/JVI.01246-20

https://www.frontiersin.org/articles/10.3389/fmicb.2020.592908/full

8. CD147 is a receptor found on our red blood cell, platelets and blood cell lining, as well as lung cell. SARS COV2 has a strong predilection for binding to this receptor. This causes clumping and clotting. COVID is a clotting disease!!!

https://www.nature.com/articles/s41392-020-00426-x

https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3636557

https://www.youtube.com/watch?v=BqSPJmh0bP0

https://www.youtube.com/watch?v=l3yw1ggunfo

https://www.biorxiv.org/content/10.1101/2020.12.21.423721v1.full.pdf

https://papers.ssrn.com/sol3/papers.cfm?abstract_id=3636557

9. Here is a comprehensive list of many additional mechanisms

https://pubmed.ncbi.nlm.nih.gov/34127807/

Additional links:

https://www.youtube.com/watch?v=JEO7Adv3tVI&t=134s

https://www.youtube.com/watch?v=GZoBAuR4ajs Great summary.

https://pubmed.ncbi.nlm.nih.gov/32251768/ Last year's study on in vitro effect of ivermectin killing SARSCOV2

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC6982461/ ivermectin also has potential against many cancers as well.

I could go down too many heady rabbit holes with very complex mechanisms, but here is the strongest overview. I hope this helps. Thanks as always,

All the best,

Ryan Cole, MD

Ivermectin is not the only early treatment medication that is very helpful in treatment of SARS COV2. Excellent studies also show benefit for early use of monoclonal antibodies, and the multiple medication use in the infected patient with any or many (not comprehensive nor confined to ) Hydroxychloroquine with zinc, Nebulized or inhaled Budesonide, Fenofibrate, Colchicine, Fluvoxamine, androgen blockers, Favipirivir, Famotidine, Ciproheptadine, Aspirin, Lovenox, N acetyl cysteine, nasal irrigation with dilutions of providing iodine, and oral irrigation with Chlorhexidine/Benzydamine containing mouthwashes, High dose vitamin D, vitamin C, melatonin, prednisone or solumedrol, at the appropriate disease course, when the inflammatory stage is present, etc. etc.

I personally have treated over 200 patients with ivermectin and other early treatment medicines and have had zero go to the ER or be hospitalized. Of about 80 I have prophylaxed with ivermectin, zero have acquired Covid. Countless of these treated patients were elderly, comorbid and high risk. My colleagues and I have collectively treated hundreds of thousands of patients with an 85% decrease of hospitalizations and death per the data presented by Peter McCullough MD.

Also see:

see the protocols at FLCCC.net, truthforhealth.org (see the McCullough protocols)

ivmmeta.com

c19early.com

c19study.com

Physicians that are online that will treat patients with multi drug therapies early outpatient therapies can be found at

myfreedoctor.com
speakwithaMD.com
frontlinesmds.com
pushhealth.com