Oregon State research shows hemp compounds prevent coronavirus from entering human cells

https://today.oregonstate.edu/news/oregon-state-research-shows-hemp-compounds-prevent-coronavirus-entering-human-cells

"Any part of the infection and replication cycle is a potential target for antiviral intervention, and the connection of the spike protein's receptor binding domain to the human cell surface receptor ACE2 is a critical step in that cycle," he said. "That means cell entry inhibitors, like the acids from hemp, could be used to prevent SARS-CoV-2 infection and also to shorten infections by preventing virus particles from infecting human cells. They bind to the spike proteins so those proteins can't bind to the ACE2 enzyme, which is abundant on the outer membrane of endothelial cells in the lungs and other organs."

Cannabidiol Inhibits SARS-CoV-2 Replication and Promotes the Host Innate Immune Response

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7987002/

ABSTRACT

The rapid spread of COVID-19 underscores the need for new treatments. Here we report that cannabidiol (CBD), a compound produced by the cannabis plant, inhibits SARS-CoV-2 infection. CBD and its metabolite, 7-OH-CBD, but not congeneric cannabinoids, potently block SARS-CoV-2 replication in lung epithelial cells. CBD acts after cellular infection, inhibiting viral gene expression and reversing many effects of SARS-CoV-2 on host gene transcription. CBD induces interferon expression and up-regulates its antiviral signaling pathway. A cohort of human patients previously taking CBD had significantly lower SARS-CoV-2 infection incidence of up to an order of magnitude relative to matched pairs or the general population. This study highlights CBD, and its active metabolite, 7-OH-CBD, as potential preventative agents and therapeutic treatments for SARS-CoV-2 at early stages of infection.

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Chloroquine is a potent inhibitor of SARS coronavirus infection and spread

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Ivermectin for Prevention and Treatment of COVID-19 Infection: A Systematic Review, Meta-analysis, and Trial Sequential Analysis to Inform Clinical Guidelines

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Review of the Emerging Evidence Demonstrating the Efficacy of Ivermectin in the Prophylaxis and Treatment of COVID-19

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Ivermectin for COVID-19

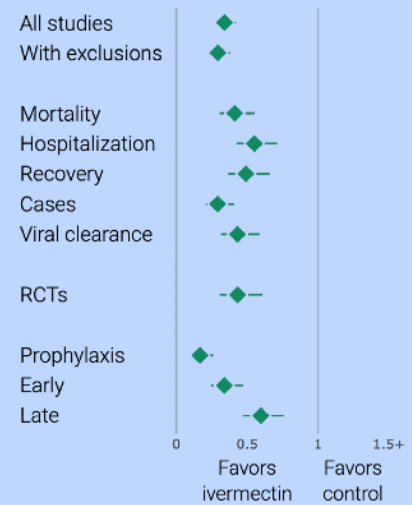
75 studies from 710 scientists
57,457 patients in 26 countries

Statistically significant improvement for **mortality**, **ventilation**, **ICU**, **hospitalization**, **recovery**, **cases**, and **viral clearance**.

83%, 66%, 40% improvement for prophylaxis, early, and late treatment CI [74-89%], [53-75%], [24-53%]

57% improvement in **32 RCTs** CI [39-69%]
59% lower **mortality** from **36** studies CI [44-69%]

COVID-19 IVERMECTIN STUDIES. JAN 14 2022. IVMMETA.COM



Azithromycin

Indications

Azithromycin is a broad-spectrum macrolide antimicrobial and is among the most prescribed antimicrobial drugs in the United States. It is a derivative of erythromycin with greatly enhanced activity against gram-negative bacteria (including Enterobacteriaceae) and provides coverage of many gram-positive organisms.[1][2]

- As an inhibitor of bacterial protein synthesis (rather than a peptidoglycan cell-wall inhibitor like beta-lactam agents), azithromycin is effective against many "atypical" bacteria such as chlamydiae (e.g., *Chlamydia trachomatis* and *Chlamydophila psittaci*), legionella (i.e., *Legionella pneumophila*), mycoplasma (e.g., *Mycoplasma pneumoniae*), and mycobacteria (e.g., *Mycobacterium avium*).[3]
- Together with its activity against *Streptococcus pneumoniae*, *Hemophilus influenzae*, and *Moraxella catarrhalis*, azithromycin is indicated—and FDA approved—for the treatment of community-acquired pneumonia (CAP).[4]
- Azithromycin also has approval for use in other upper respiratory infectious processes, including acute otitis media and acute exacerbation of chronic obstructive pulmonary disease (COPD).[5]
- Additionally, azithromycin has approval for the treatment of pharyngitis caused by *Streptococcus pyogenes*, as an alternative to a beta-lactam agent; skin or skin structure infection due to *S. pyogenes*, *Streptococcus agalactiae*, or *Staphylococcus aureus*; M. *avium* complex (MAC) infection treatment and prophylaxis for patients with advanced acquired immunodeficiency syndrome (AIDS); and sexually transmitted infections including chlamydia, gonococcal disease, chancroid (caused by *Hemophilus ducreyi*), and *Mycoplasma genitalium*.[6][7][8][9][10]
- Azithromycin also has efficacy against some protozoal organisms such as *Babesia sp.* (e.g., *B. microti*), *Plasmodium sp.* (i.e., malaria), and *Toxoplasma gondii* and is sometimes used off-label for the treatment of these parasitic diseases in combination with antiprotozoal drugs (e.g., atovaquone).[11][12][13]
- The role of azithromycin in the treatment of viral infections, including the respiratory syncytial virus and novel coronavirus SARS-CoV-2, is indeterminate.[14][15][16][17][18][17]
- Lastly, azithromycin is also used off-label as long-term prophylaxis for bronchiolitis obliterans (BO) in patients who have undergone lung transplantation.[19]

Inhaled budesonide in the treatment of early COVID-19 (STOIC): a phase 2, open-label, randomised controlled trial

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Interpretation

Early administration of inhaled budesonide reduced the likelihood of needing urgent medical care and reduced time to recovery after early COVID-19.

We have shown that the inhaled glucocorticoid budesonide, given for a short duration, might be an effective treatment of early COVID-19 in adults. This effect, with a relative reduction of 91% of clinical deterioration is equivalent to the efficacy seen after the use of COVID-19 vaccines[14] and greater than that reported in any treatments used in hospitalised patients and patients with severe COVID-19.[15] Our study showed a 14% incidence of urgent health-care need and is consistent with other community-based studies.[16]

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Review Article

Melatonin interferes with COVID-19 at several distinct ROS-related steps

Olivia G. Camp [a, b, 1], David Bai [a, 1], Damla C. Gonullu [a], Neha Nayak [a], Husam M. Abu-Soud [a, b, c]

- SARS-CoV-2 infection generates overwhelming levels of neutrophil myeloperoxidase
- Hypochlorous acid and other reactive oxygen species destroy tetrapyrrole rings
- This causes nitric oxide, oxygen, and vitamin B12 deficiencies; markers of COVID-19
- Melatonin inhibits myeloperoxidase activity and scavenges reactive oxygen species
- Melatonin supplements can prevent pathophysiological consequences of COVID-19 disease

International Immunopharmacology
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Safety and efficacy of ozone therapy in mild to moderate COVID-19 patients: A phase 1/11 randomized control trial (SEOT study) ☆

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Highlights

- Ozone therapy as an adjuvant care can shorten hospitalization and need for intensive care.
- Ozone therapy enhances the quality of care in COVID-19 by exhibiting immune-modulatory effects.
- Ozone therapy as an adjuvant therapy is safe and effective in COVID-19 care .
- Ozone therapy relieves cardinal symptoms of SARS-CoV-2 like breathlessness, cough etc.

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PMCID: PMC7263077
PMID: 32463221

Endogenous Deficiency of Glutathione as the Most Likely Cause of Serious Manifestations and Death in COVID-19 Patients

Alexey Polonikov*

Endogenous glutathione deficiency appears to be a crucial factor enhancing SARS-CoV-2-induced oxidative damage of the lung and, as a result, leads to serious manifestations, such as acute respiratory distress syndrome, multiorgan failure, and death in COVID-19 patients. When the antiviral activity of GSH is taken into account, individuals with glutathione deficiency seem to have a higher susceptibility for uncontrolled replication of SARS-CoV-2 virus and thereby suffer from an increasing viral load. The severity of clinical manifestations in COVID-19 patients is apparently determined by the degree of impaired redox homeostasis attributable to the deficiency of reduced glutathione and increased ROS production. This assumption can be supported by our findings. In particular, COVID-19 patients with moderate and severe illness had lower levels of glutathione, higher ROS levels, and greater redox status (ROS/GSH ratio) than COVID-19 patients with a mild illness. Long-term and severe manifestations of COVID-19 infection in one of our patients with marked glutathione deficiency suggest that the degree of glutathione decrease correlates negatively with viral replication rate and that an increasing viral load exacerbates oxidative damage of the lung. This finding suggests that the virus cannot actively replicate at higher levels of cellular glutathione, and therefore, milder clinical symptoms are observed with lower viral loads.

Clinical Trial > J Infect Dis. 1981 Jan;143(1):101-5. doi: 10.1093/infdis/143.1.101.

Immunomodulating properties of dimethylglycine in humans

C D Graber, J M Goust, A D Glassman, R Kendall, C B Loadholt

PMID: 6163829 DOI: 10.1093/infdis/143.1.101