



**STATE OF
OHIO**
BOARD OF PHARMACY

COVID-19 Response Efforts - March 24, 2022

The State of Ohio Board of Pharmacy is committed to protecting the health and safety of Ohioans during the COVID-19 outbreak. The Board posted a document on its website that provides COVID-19 guidance and response efforts, including the issuance of waivers to assist licensees in addressing operational needs. **Waivers can be found starting on page 5 of this document:** www.pharmacy.ohio.gov/COVID.

For more information on the state's efforts to address coronavirus, visit www.coronavirus.ohio.gov or call 1-833-4-ASK-ODH.

COVID-19 Vaccine Booster Communications Toolkit

Ohio has reached a point in the pandemic where it is critically important for all eligible Ohioans in all age groups who haven't already gotten a COVID-19 vaccine booster shot to get one – especially Ohioans age 50+ who are at greatest risk of severe illness, hospitalization, and death.

Today, the Board posted a [communications toolkit for community partners](#) on its website that equips users in communicating the importance of getting a COVID-19 booster shot, answering technical questions, and providing locations where patients may receive a booster shot. The toolkit is designed for stakeholders to promote the importance of booster doses using their social media channels with sample text and graphics, or on their websites or in their publications.

Please note that if you choose to unsubscribe from receiving emails, this unsubscribes you from all emails that are sent by the State of Ohio Board of Pharmacy, including renewal reminders. If you believe you have unsubscribed in error, please email contact@pharmacy.ohio.gov for assistance. If you would like to change the email address, please log into your [eLicense Ohio](#) account to update the email address on file.

Public Health Communications Collaborative Resources for COVID-19

<https://publichealthcollaborative.org/>

Mayo Clinic COVID-19 Tool Kit

<https://www.mayoclinic.org/coronavirus-covid-19>

Boosting COVID-19 Vaccine Confidence: An Educational Toolkit for Providers

<https://primeinc.org/online/boosting-covid-19-vaccine-confidence-educational-toolkit-providers>

COVID-19 Incidence and Death Rates Among Unvaccinated and Fully Vaccinated Adults with and Without Booster Doses During Periods of Delta and Omicron Variant Emergence — 25 U.S. Jurisdictions, April 4–December 25, 2021

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On January 21, 2022, this report was posted as an MMWR Early Release on the MMWR website (<https://www.cdc.gov/mmwr>).

Previous reports of COVID-19 case, hospitalization, and death rates by vaccination status[†] indicate that vaccine protection against infection, as well as serious COVID-19 illness for some groups, declined with the emergence of the

B.1.617.2 (Delta) variant of SARS-CoV-2, the virus that causes COVID-19, and waning of vaccine-induced immunity (1–4). During August–November 2021, CDC recommended[§] additional primary COVID-19 vaccine doses among immunocompromised persons and booster doses among persons aged ≥18 years (5). The SARS-CoV-2 B.1.1.529 (Omicron) variant emerged in the United States during December 2021 (6) and by December 25 accounted for 72% of sequenced

lineages (7). To assess the impact of full vaccination with additional and booster doses (booster doses),[¶] case and death rates and incidence rate ratios (IRRs) were estimated among unvaccinated and fully vaccinated adults by receipt of booster doses during pre-Delta (April–May 2021), Delta emergence (June 2021), Delta predominance (July–November 2021), and Omicron emergence (December 2021) periods in the United States. During 2021, averaged weekly, age-standardized case IRRs among unvaccinated persons compared with fully vaccinated persons decreased from 13.9 pre-Delta to 8.7 as Delta emerged, and to 5.1 during the period of Delta predominance. During October–November, unvaccinated persons had 13.9 and 53.2 times the risks for infection and COVID-19–associated death, respectively, compared with fully vaccinated persons who received booster doses, and 4.0 and 12.7 times the risks

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[†] A COVID-19 case in a fully vaccinated person occurred when SARS-CoV-2 RNA or antigen was detected in a respiratory specimen collected ≥14 days after completing the primary series of a COVID-19 vaccine with Food and Drug Administration (FDA) approval or emergency use authorization. The COVID-19 case definition, including criteria to distinguish a new case from an existing case, is per the July 2021 update to the national standardized surveillance case definition and national notification for 2019 novel coronavirus disease (COVID-19) (21-ID-01) (<https://ndc.services.cdc.gov/case-definitions/coronavirus-disease-2019-2021/>). Fully vaccinated persons were those with a completed primary series of 2 doses of the Pfizer-BioNTech or Moderna mRNA vaccine or a single dose of the Janssen vaccine (<https://www.cdc.gov/coronavirus/2019-ncov/vaccines/stay-up-to-date.html>). A COVID-19 case in an unvaccinated person occurred when the person did not receive any FDA-authorized COVID-19 vaccine doses before the specimen collection date. Cases were excluded in partially vaccinated persons who received at least one FDA-authorized or approved vaccine dose but did not complete a primary series

≥14 days before collection of a respiratory specimen with SARS-CoV-2 RNA or antigen detected. Ascertaining vaccination status for COVID-19 patients through active linkage of case surveillance and immunization information systems typically assumes that cases among persons who are unmatched to the registry are unvaccinated. This analysis represents the combined impact of the Pfizer-BioNTech, Moderna, and Janssen COVID-19 vaccines, which had different clinical efficacies against confirmed infection. Information on different FDA-authorized and approved COVID-19 vaccine products, including clinical efficacy, is available online. <https://www.cdc.gov/coronavirus/2019-ncov/vaccines/different-vaccines.html>

[§] On August 13, 2021, CDC recommended an additional Pfizer-BioNTech or Moderna primary series dose for persons moderately or severely immunocompromised (<https://www.cdc.gov/media/releases/2021/s0813-additional-mrna-mrna-dose.html>). On September 24, 2021, CDC recommended a Pfizer-BioNTech booster dose for certain Pfizer-BioNTech primary series recipients, including all adults aged ≥65 years and persons aged ≥18 years in certain populations and high risk occupational and institutional settings (<https://www.cdc.gov/media/releases/2021/p0924-booster-recommendations.html>). On October 21, 2021, CDC recommended a booster dose for adults aged ≥18 years who had received the Janssen vaccine and for Pfizer-BioNTech or Moderna primary series vaccine recipients, including all adults aged ≥65 years and persons aged ≥18 years in certain populations and high risk occupational and institutional settings (<https://www.cdc.gov/media/releases/2021/p1021-covid-booster.html>). On November 19, 2021, and November 29, 2021, CDC expanded recommendations for booster doses to include all adults aged ≥18 years (<https://www.cdc.gov/media/releases/2021/s1119-booster-shots.html>) (<https://www.cdc.gov/media/releases/2021/s1129-booster-recommendations.html>).

[¶] A COVID-19 case in a fully vaccinated person with a booster dose occurred when a person had SARS-CoV-2 RNA or antigen detected on a respiratory specimen collected ≥14 days after receipt of at least 1 additional or booster dose of any COVID-19 vaccine on or after August 13, 2021 (this definition does not distinguish between vaccine recipients who are immunocompromised and are receiving an additional dose versus those who are not immunocompromised and receiving a booster dose).



REVIEW

Vitamins, supplements and COVID-19: a review of currently available evidence

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Abstract

Background: In the midst of the COVID-19 pandemic, there has been an information overload of health data (both accurate and inaccurate) available to the public. With vitamins and supplements being readily accessible, many have turned to using them in an effort to combat the virus. The purpose of

this review was to analyse clinical trials regarding vitamins and supplements for the treatment of COVID-19 infections.

Methods: Articles were identified through a literature search utilizing online databases and bibliographic review.

Results: A total of seven articles were identified for review. All articles evaluated the use of vitamins and supplements for the treatment of COVID-19. Drug therapies included oral vitamin D, intravenous and oral vitamin C, oral vitamin D/magnesium/ vitamin B12, oral zinc, oral combination zinc/ascorbic acid, and intravenous alpha-lipoic acid. The end points of each study

varied, including the Sequential Organ Failure Assessment score, mortality, rate of intensive care unit (ICU) admissions, negativity of COVID-19 tests, oxygen requirements, and symptom burden.

Conclusion: Of the vitamins and supplements that were studied, vitamin D presented the most promising data demonstrating significant decreases in oxygen requirements, need for ICU treatment, SARS-CoV-2 RNA test positivity, and mortality. All of these benefits were exhibited in hospitalized patients. Other vitamins and supplements that were evaluated in studies did not demonstrate any statistically significant benefits. Common shortcomings of the articles included generally small sample sizes, varying sites of study (which could determine the virus variant), a lack of standard of care as background therapy, and utilization of doses that were higher than standard.

Keywords: coronavirus, COVID-19, SARS-CoV-2, severe acute respiratory syndrome coronavirus, supplement, vitamin.

Citation

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Introduction

SARS-CoV-2, the virus causing COVID-19, was first reported to the WHO on 31 December 2019 and was declared a global pandemic on 11 March 2020.¹⁻³ To date, there have been more than 229 million reported cases and 4.7 million deaths

of COVID-19 infections, with the exception of vaccination. Throughout the course of this pandemic, many therapies have been proposed as having utility, with many, but not all, falling short of providing meaningful results in clinical trials. Some proposed therapies have never undergone clinical trials and medical claims are being made based on theoretical or anecdotal evidence.¹⁷ Since the public

Identification of *LZTFL1* as a candidate effector gene at a COVID-19 risk locus

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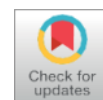
The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) disease (COVID-19) pandemic has caused millions of deaths worldwide. Genome-wide association studies identified the 3p21.31 region as conferring a twofold increased risk of respiratory failure. Here, using a combined multiomics and machine learning approach, we identify the gain-of-function risk A allele of an SNP, rs17713054G>A, as a probable causative variant. We show with chromosome conformation capture and gene-expression analysis that the rs17713054-affected enhancer upregulates the interacting gene, leucine zipper transcription factor like 1 (*LZTFL1*). Selective spatial transcriptomic analysis of lung biopsies from patients with COVID-19 shows the presence of signals associated with epithelial–mesenchymal transition (EMT), a viral response pathway that is regulated by *LZTFL1*. We conclude that pulmonary epithelial cells undergoing EMT, rather than immune cells, are likely responsible for the 3p21.31-associated risk. Since the 3p21.31 effect is conferred by a gain-of-function, *LZTFL1* may represent a therapeutic target.

The COVID-19 pandemic is estimated to have caused over 4.6 million deaths so far^{1,2}. The predominant cause of mortality is pneumonia and severe acute respiratory distress syndrome³. However, COVID-19 can cause multiple organ failure through cytokine release, microvascular and macrovascular thrombosis, endothelial damage, acute kidney injury and myocarditis^{4–6}. Genome-wide association studies (GWAS) are important for identifying candidate genes and pathways that predispose to complex diseases⁷; genetically validated drug targets are more likely to lead to approved drugs⁸. Two large GWAS were carried out to determine whether common variants drive susceptibility to severe COVID-19 (refs. ^{9,10}). Both studies identified a region of chromosome 3p21.31 as having the strongest association, while a third study also identified this locus as conferring susceptibility to infection¹¹. The 3p21.31 risk haplotype, which arises from Neanderthal DNA¹² and is currently unexplained with regards to the causal variant(s), causal gene(s) and specific role in COVID-19, confers a twofold increased risk of respiratory failure from COVID-19 (refs. ^{9,10}) and an overtwofold increased risk of mortality for individuals under 60 (ref. ¹³). Additionally, the risk variants at this locus are carried by >60% of individuals with South Asian ancestry (SAS), compared to 15% of European ancestry (EUR) groups, partially explaining the ongoing higher death rate in this population in the UK^{14,15}.

Identifying the causal gene(s) and mechanism(s) behind GWAS hits poses several challenges. First, a causative variant is usually in linkage disequilibrium (LD) with many other variants and these can take different forms (SNPs, insertions, deletions and structural polymorphisms). Second, the genetic signals are completely cell type-agnostic, which makes it challenging to identify appropriate experimental models for further investigation. Third, there are multiple mechanisms by which variants can have an effect. Alteration of the protein-coding sequence or RNA splicing, both of which are relatively straightforward to disentangle, account for fewer than 20% of associations in polygenic disease¹⁶. The remaining variants and their target gene(s) can be very difficult to decode. Many are thought to lie within *cis*-regulatory elements¹⁷, such as enhancers, which are short DNA sequences that often control tissue- and developmental stage-specific gene expression. Deciphering the variants that affect enhancers is challenging because many enhancers are only active in specific cell types or at specific times; enhancers are often distant in the linear DNA sequence (often 10⁴–10⁶ base pairs (bp)) from the genes they control and the effects of sequence changes are not straightforward to predict.

We developed a comprehensive platform for decoding the effects of sequence variation identified by GWAS¹⁶ (Extended Data Fig. 1a). This combines computational and wet lab approaches to delineate

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Possible Involvement of Adipose Tissue in Patients With Older Age, Obesity, and Diabetes With Coronavirus SARS-CoV-2 Infection (COVID-19) via GRP78 (BIP/HSPA5): Significance of Hyperinsulinemia Management in COVID-19

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Aging, obesity, and diabetes are major risk factors for the severe progression and outcome of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (coronavirus disease 2019 [COVID-19]), but the underlying mechanism is not yet fully understood. In this study, we found that the SARS-CoV-2 spike protein physically interacts with cell surface GRP78, which promotes the binding to and accumulation in ACE2-expressing cells. GRP78 was highly expressed in adipose tissue and increased in humans and mice that were older, obese, and had diabetes. The overexpression of GRP78 was attributed to hyperinsulinemia in adipocytes, which was in part mediated by the stress-responsive transcription factor XBP-1s. Management of hyperinsulinemia by pharmacological approaches, including metformin, sodium–glucose cotransporter 2 inhibitor, or β_3 -adrenergic receptor agonist, decreased GRP78 gene expression in adipose tissue. Environmental interventions, including exercise, calorie restriction, fasting, or cold exposure, reduced the gene expression of GRP78 in adipose tissue. This study provides scientific evidence for the role of GRP78 as a binding partner of the SARS-CoV-2 spike protein and ACE2, which might be related to the severe progression and outcome of COVID-19 in older and obese patients with diabetes. The management of hyperinsulinemia and the related GRP78 expression could be a therapeutic or preventative target.

The outbreak of the novel β -coronavirus severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, coronavirus disease 2019 (COVID-19), has rapidly spread worldwide and, to date, has resulted in over 169,000,000 human infections and more than 3,500,000 deaths. The development of SARS is the major factor for serious progression and mortality in COVID-19 patients (1). Emerging studies have shown that there is an increased risk of poor outcomes with increasing age, obesity, visceral adiposity, and diabetes (2–4), but the linked molecular mechanisms have not yet been explained. In this severe pandemic, further scientific information and therapeutic targets are required.

While adipose tissue plays an important role in the regulation of energy homeostasis, its abnormalities have harmful effects on systemic healthy states. The aging- or obesity-associated pathological expansion of adipose tissue, especially in the visceral region, contributes to the development of various metabolic diseases and their complications (5–8). Hyperinsulinemia, a chronic state of high insulin levels, is commonly found in older or obese patients (9,10) and causes detrimental cellular stress in adipose tissue, such as reactive oxygen species, endoplasmic reticulum (ER) stress, hypoxia, and inflammation (11–14). Recently, adipose tissue has been taken into

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Implications of suboptimal COVID-19 vaccination coverage in Florida and Texas

In July, 2021, another wave of COVID-19 began in the USA as the highly infectious delta (B.1.617.2) SARS-CoV-2 variant drove outbreaks predominantly affecting states with relatively low vaccination coverage. Some US states have shown the feasibility of rapidly achieving high vaccination coverage. Specifically, an average of 74.0% of adults had been fully vaccinated in Vermont, Connecticut, Massachusetts, Maine, and Rhode Island by July 31. By contrast, two states facing substantial delta-driven surges, Florida and Texas, had fully vaccinated only 59.5% and 55.8% of their adult residents, respectively.¹ Here, we estimate the deaths, hospital admissions, and infections that could have been averted if Florida and Texas had matched the average vaccination pace of the top-performing states and vaccinated 74.0% of their adult populations by the end of July.

We adapted our agent-based model of SARS-CoV-2 transmission^{2,3} to the demography, contact patterns, and age-stratified vaccination trajectories of Florida and Texas. We further accounted for the emergence and spread of the alpha (B.1.1.7), gamma (P.1), iota (B.1.526), and delta variants, in addition to the original strain.^{2,3} Vaccine efficacies against infection and symptomatic and severe disease for different vaccine types, each variant, and by vaccine dosage were parameterised from clinical studies (appendix

pp 4–5). The model was calibrated to the reported incidence in each state between Oct 1, 2020, and Aug 31, 2021 (appendix p 6). Using the calibrated model, we

daily vaccine doses distributed to achieve 74.0% coverage of fully vaccinated adults by July 31, 2021, and continued with the associated daily rates of vaccine rollout. We then simulated the epidemiological trajectories of outbreaks in Florida and Texas and compared them with the observed cases, hospital admissions, and deaths in these two states from Dec 12, 2020, to Aug 31, 2021.

We found that enhanced vaccination would have markedly blunted the increase in cases, hospital admissions, and deaths in Florida and Texas (figure; appendix p 6). From the start of vaccination on Dec 12, 2020, until Aug 31, 2021, Florida had reported 2 221 520 COVID-19 cases and Texas had reported 2 142 833. Achieving 74.0% vaccination coverage by July 31 and continuing with the associated daily rate would have averted 664 007 additional cases (95% credible interval [CrI] 419 219–848 020) in Florida and 647 906 additional cases (507 298–789 885) in Texas (appendix p 7). By Aug 31, the enhanced vaccination in Florida would have reduced hospital admissions by 61 327 (95% CrI 49 723–73 501) and deaths by 16 235 (13 243–19 473). The reduction in hospital admissions in Texas during the same period would have been 37 587 (95% CrI 31 575–44 659) and the reduction in deaths would have been 6353 (5227–7501). Collectively, these two states could have averted more than 95 000 hospital admissions and 22 000 deaths had they reached the vaccination coverage achieved by the top five states and continued at the same pace until Aug 31, 2021.

We further projected the epidemiological impact of a 50% increase in the daily vaccination rate in Florida and Texas compared with the status quo from Sept 1, 2021, (figure

Oct 31, 2021, such acceleration of vaccination would prevent more than 26 000 cases and 1200 deaths in the two states.

Hospitals and intensive care units in several US states are currently overwhelmed by a surge in symptomatic COVID-19 illness almost entirely among unvaccinated individuals. The combination of relatively lower vaccination rates in southern and central US states, especially among younger people, is even more concerning as schools return to in-person classes and non-pharmacological measures such as mask wearing and physical distancing are relaxed. As the pandemic continues, efforts to increase vaccination will be crucial



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See Online for appendix

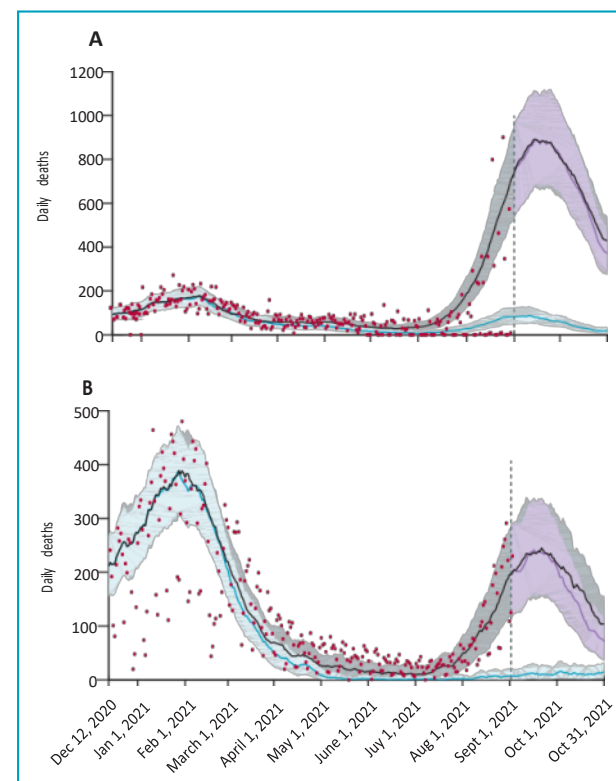


Figure: Model projections of daily deaths in (A) Florida and (B) Texas

Black lines show mean estimates, with uncertainty bounds of simulations shown in grey shaded areas. Red dots are reported data. Blue lines and shaded areas show the model projections for mean estimates and uncertainty bounds under the counterfactual scenario of enhanced vaccination with 74.0% coverage of adults by July 31, 2021. The purple lines and shaded areas show the model projections for mean