DR. ARDIS'S
COVID 19/COVID 19 VACCINE DISEASE PROTECTION PROTOCOL

U.S. FOOD & DRUG ADMINISTRATION

CBER Plans for Monitoring COVID-19 Vaccine Safety and Effectiveness

Steve Anderson, PhD, MPP
Director, Office of Biostatistics & Epidemiology, CBER

VRBPAC Meeting
October 22, 2020

SLIDE 16 of FDA's CBER Report Dated 10/22/2020

FDA Safety Surveillance of COVID-19 Vaccines : DRAFT Working list of possible adverse event outcomes ***Subject to change***

- Guillain-Barré syndrome
- Acute disseminated encephalomyelitis
- Transverse myelitis
- Encephalitis/myelitis/encephalomyelitis/ meningoencephalitis/meningitis/ encepholapathy
- Convulsions/seizures
- Stroke
- Narcolepsy and cataplexy
- Anaphylaxis
- Acute myocardial infarction
- Myocarditis/pericarditis
- Autoimmune disease
- Deaths
- Pregnancy and birth outcomes
- Other acute demyelinating diseases
- Non-anaphylactic allergic reactions
- Thrombocytopenia
- Disseminated intravascular coagulation
- Venous thromboembolism
- Arthritis and arthralgia/joint pain
- Kawasaki disease
- Multisystem Inflammatory Syndrome in Children
- Vaccine enhanced disease

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The two slides above come from the FDA's internal document 2 months before COVID 19 Vaccines started being administered to the public. This report is 25 slides long, and Slide 16 of that presentation lists 22 bullet points of known serious and life threatening conditions that WILL BE CAUSED by the coming COVID 19 Vaccines.

Important to note, ALL Diseases are exacerbated or initiated completely by Oxidative Stress on cells in any tissues in the human body! The following references will help establish the protective mechanisms of 3 key nutrients PROVEN to protect ALL cells of the human body against Oxidative Stress caused by Viruses, Poisons, and trauma. These 3 Disease Preventing Nutrients if taken daily WILL GIVE YOU AND YOUR LOVED ONES the best chance of escaping the detrimental side effects of ALL viruses and Vaccine injury or Shedding!

https://cdn.intechopen.com/pdfs/35941/InTech-Oxidative_stress_cause_and_consequence_of_diseases.pdf

Oxidative Stress: Cause and Consequence of Diseases

Dmytro Gospodaryov and Volodymyr Lushchak
Precarpathian National University,
Ivano-Frankivsk,
Ukraine

1. Introduction

Oxidative stress, termed as an imbalance between production and elimination of reactive oxygen species (ROS) leading to plural oxidative modifications of basic and regulatory processes, can be caused in different ways. Increased steady-state ROS levels can be promoted by drug metabolism, overexpression of ROS-producing enzymes, or ionizing radiation, as well as due to deficiency of antioxidant enzymes. The plethora of ways

diabetes, can be exacerbated by oxidative modification of antioxidant enzymes. These assumptions demonstrate the potential of antioxidant therapy in particular cases.

At some pathological states, whatever the cause of the disease, oxidative stress is seen to be a powerful exacerbating factor. Type II diabetes, cardiovascular diseases and neurodegenerative diseases, associated with protein aggregation are among such pathologies. Indeed, enhanced level of glucose results in higher probability of protein glycation (Wautier & Schmidt, 2004).

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The FDA's Slide 16 on page 1 shows Acute Myocardial Infarction as a Side effect of Covid 19 Vaccines: Acute Myocardial Infarction is also called a Heart Attack… (they knew people would have heart attacks from the shots, 2 months before vaccines came out in Dec 2020) Risk factor for Heart Attack (acute myocardial infarction) is a Prolonged QT Wave on a EKG.

Spike Proteins found on COVID 19 Virus and the Spike Proteins the Vaccines will create in your body do cause Prolonged QT Wave, which is why people will experience death from the infection and also from the Vaccines. On Mayo Clinic's Website they explain the serious life threatening impacts of a prolonged (long) QT interval on an ECG reading…

mayoclinic.org/diseases-conditions/long-qt-syndrome/symptoms-causes/syc-20352518

Gmail YouTube Maps Dropbox - TruLab... FDA Dietary Suppleme...

Complications

Proper medical treatment and lifestyle changes can help prevent complications related to long QT syndrome.

Complications of long QT syndrome include:

- **Torsades de pointes ('twisting of the points').** This is a life-threatening form of ventricular arrhythmia. Your heart's two lower chambers (ventricles) beat fast and chaotically, making the waves on an ECG monitor look twisted. The heart pumps out less blood. The lack of blood to the brain causes you to faint suddenly and, often, without warning.

  If the episode lasts for a long time, fainting can be followed by a full-body seizure. If the dangerous rhythm does not correct itself, then a life-threatening arrhythmia called ventricular fibrillation follows.
- **Ventricular fibrillation.** This condition causes the ventricles to beat so fast that your heart quivers and stops pumping blood. Unless your heart is shocked back into a normal rhythm by a defibrillator, ventricular fibrillation can lead to brain damage and sudden death.
- **Sudden death.** It's now known that long QT syndrome might explain some cases of sudden death in young people who otherwise appear healthy.
- **Unexplained fainting, drownings, seizures or other accident.** Long QT syndrome might be responsible for some otherwise unexplained deaths in children and young adults.

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There have already been **4,000 reported dead** so far from COVID 19 Injections alone, **20% are from Heart Attacks**. This Damage by the Vaccine and Virus to cause heart attacks is directly correlated to the spike proteins damage to heart muscle, mitochondria in heart muscle and nerves causing a prolonged QT Interval. Here is one referenced published article, and a screen shot, discussing Covid 19 Spike Causing Prolonged QT Interval.

http://www.sciepub.com/AJMCR/abstract/13067

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New Onset, Irreversible, Prolonged QT-Interval Requiring Permanent Biventricular Pacemaker in a COVID-19 Patient

Ahmad Jallad[1], Andrew V. Doodnauth[2], Justin Lee[1], Emmanuel Valery[2], Stephanie Myers[2], Dahlia Rizk[3] and Samy I. McFarlane[2,*]

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Pub. Date: February 03, 2021

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Abstract

Various electrocardiographic (EKG) manifestations have been reported in patients with coronavirus disease 2019 (COVID-19). There is growing evidence showing that new onset QT-prolongation is a common EKG finding in COVID-19 patients. In this report, we present a case of a 71-year-old man who was found to have a new onset, irreversible, prolonged QT-interval requiring permanent biventricular pacemaker despite testing negative twice for RT-PCR COVID-19 and correction of all known reversible causes. To date, there are a limited number of reports of irreversible QT-prolongation associated with COVID-19. This case report emphasizes the importance of a physician's clinical judgment in the setting of negative RT-PCR COVID-19 testing. A robust systemic inflammatory state seen in active COVID-19 infection is possibly the key mechanism precipitating the new EKG

Magnesium is proven in this study **to normalize the QT Interval** in these drug poisoned individuals… many drugs cause prolonged QT intervals that lead to further heart disease and risk of death. Of note, the COVID 19 Vaccine is a drug and as such presents many toxicities to the human body that will cause prolonged QT Intervals leading to arrhythmias and Heart Attacks. Here is one such study and a screen shot of highlighted benefits of magnesium to the heart and whole body.

https://journals.plos.org/plosone/article?id=10.1371/journal.pone.0098971

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Association between Low Serum Magnesium Level and Major Adverse Cardiac Events in Patients Treated with Drug-Eluting Stents for Acute Myocardial Infarction

Guipeng An, Zhongqi Du, Xiao Meng, Tao Guo, Rui Shang, Jifu Li, Fengshuang An, Wenjing Li, Cheng Zhang

Published: June 5, 2014 • https://doi.org/10.1371/journal.pone.0098971

Article | Authors | Metrics | Comments | Media Coverage

Abstract
Introduction
Subjects and Methods
Results
Discussion
Conclusions
Author Contributions
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Abstract

Objectives

We investigated the association of serum magnesium (Mg) levels and major adverse cardiac events (MACEs) after drug-eluting stent (DES) implantation.

Background

Mg depletion plays a key role in the pathphysiologic features of diabetes mellitus, hypertension, thrombosis, arrhythmias and coronary artery disease. Whether the depletion is related to the long-term prognosis of DES implantation is not known.

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Association between Low Serum Magnesium Level and Major Adverse Cardiac Events in Patients Treated with Dr...

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Abstract
Introduction
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We found serum Mg levels, independent of other risk factors, is inversely related to the incidence of MACEs in patients with DES implantation for acute myocardial infarction but not unstable angina. Compared with patients with the highest serum Mg level (>0.94 mmol/L), those with the lowest level (<0.86 mmol/L) showed an 8.11-fold higher risk for MACEs after DES implantation. This finding can allow us to better distinguish patients at increased risk and support further research into the effectiveness of Mg supplementation for this group of patients.

Mg deficiency enhances vascular endothelial injury, increases low-density lipoprotein concentration and oxidative modification, and thus promotes the development and progression of atherosclerosis [19], [20]. As well, it affects risk factors of myocardial infarction such as blood pressure, glucose metabolism, and lipid levels [21], [22]. In addition, Mg has antiarrhythmic effects, and chronic Mg deficiency may be proarrhythmic [23]. Several recent studies showed an association of low serum Mg level and increased risk of atrial fibrillation [24] and sudden cardiac death [25]. Therefore, low Mg serum level may be related to the prognosis of DES implantation. With a median follow-up of 24 months for ACS patients, our data confirmed this hypothesis, finding an inverse relationship between serum Mg level and MACEs in patients with DES implantation for acute myocardial infarction.

Mg is the physiological Ca antagonist, and its serum concentration is remarkably constant in healthy subjects. Low Mg level deteriorates endothelial function [26] and leads to the Ca overload that occurs after reperfusion [27]. Mg was found involved in platelet-dependent thrombosis and inversely related to platelet aggregation and adenosine triphosphate release [28], [29]. In clinical experiments, Mg supplementation could reduce acute platelet-dependent thrombosis [28]. In addition, Mg can halt smooth muscle cell proliferation and stimulate endothelial cell proliferation, which might translate into a beneficial effect in the setting of stent-associated vascular injury [30]. These may be potential mechanisms for the decreased MACEs seen in our patients with low Mg levels. However, the reason why the relationship was found only in patients with acute myocardial infarction but not unstable angina still needs to be investigated.

On slide 16 of the FDA's CBER report in October, multiple blood clotting disorders are included as risks of the COVID 19 Vaccines…particularly these 4.

Thrombocytopenia
Disseminated Intravascular coagulation
Venous Thromboembolism
Strokes

MAGNESIUM PROTECTS AGAINST BLOOD CLOT DISORDERS

If you have been vaccinated or are exposed or worried about shedding of the antibodies from those vaccinated. We know the spike proteins associated with Covid 19 and the Billions of Spike Proteins the Vaccines are making our bodies make, kill people and cause clotting problems, strokes and ALL listed diseases and outcomes on Slide 16. Great news however, regardless of what is causing damage to the blood vessels and damage to platelets in the blood from the spike proteins, Magnesium has been PROVEN to prevent and protect against blood clotting! Here is a few studies, and a screen shot of the conclusion.

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https://pubmed.ncbi.nlm.nih.gov/12774935/

Antithrombotic effects of magnesium sulfate in in vivo experiments

Joen R Sheu [1], George Hsiao, Ming Y Shen, Yen M Lee, Mao H Yen

Affiliations + expand

PMID: 12774935 DOI: 10.1007/BF02982655

Abstract

In this study, magnesium sulfate was effective in reducing the mortality of adenosine diphosphate-induced acute pulmonary thromboembolism in mice, when it was administered intravenously at doses of 100 and 200 microg/g body weight. In addition, intravenous injections of magnesium sulfate (100 and 200 microg/g) significantly prolonged bleeding time in the severed mesenteric arteries of rats by approximately 1.7- and 1.9-fold, respectively, compared with normal saline. Continuous infusion of magnesium sulfate (20 microg/g per minute) for 10 minutes also significantly increased the bleeding time by approximately 1.7-fold, and the bleeding time returned to baseline within 60 minutes of cessation of magnesium sulfate infusion. On the other hand, platelet thrombi formation was induced by irradiating mesenteric venules with filtered light in mice pretreated with intravenous fluorescein sodium. When magnesium sulfate was administered at 300 microg/g during induction of platelet plug formation with 10 microg/kg fluorescein sodium, occlusion time was not significantly prolonged, but a dose of 600 microg/g did significantly prolong the occlusion time. Furthermore, aspirin (250 microg/g) also showed a similar activity in this experiment in prolonging the occlusion time. In conclusion, these results suggest that magnesium sulfate has an effective antithrombotic activity in vivo, and treatment with magnesium sulfate may lower the risk of thromboembolic-related disorders.

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and another study showing Magnesium's Protection again blood clotting, particularly pulmonary (lung) thromboembolism.

https://www.researchgate.net/publication/350399391_Increased_angiotensin-converting_enzyme_2_sRAGE_and_immune_activation_but_lowered_calcium_and_magnesium_in_COVID-19_association_with_chest_CT_abnormalities_and_lowered_peripheral_oxygen_saturation

KB potentially resulting in a positive feedback mechanism amplifying inflammation, oxidative stress and calcium dyshomeostasis (Berry et al., 2018, Dresselhaus and Meffert, 2019).

Lowered serum magnesium is associated with increased thrombosis risk, decreased fibrinolysis, endothelial dysfunction, mitochondrial dysfunctions, increased inflammatory and oxidative stress, and increased fatty acid production (Çiçek et al., 2016, Zheltova et al., 2016, Gromova et al., 2018, Nielsen, 2018). Furthermore, in vivo studies have demonstrated that

18

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magnesium has antithrombotic properties and decreases mortality in induced pulmonary thromboembolism (Sheu et al., 2003).

Quote above, "**Lowered serum magnesium** is associated with **increased thrombosis risk**, decreased fibrinolysis, endothelial dysfunction, **mitochondrial dysfunctions**, **increased inflammatory and oxidative stress.."**

Increased Inflammatory and Oxidative Stress is the Single cause of ALL diseases in the human body. Magnesium protects against ALL Inflammatory and Oxidative Stress our cells will encounter from Covid 19 virus, from the Vaccine and the poisons inside of it… and the trauma from its gene technology inside, (ie. mRNA in Moderna and Pfizer, and the Transgene Tech inside J & J and AstraZeneca Vaccines)

Inflammatory Oxidative Stress that is the underlying cause of ALL Disease, has several markers that can be measured in the blood, here is a list of some…COVID 19 and COVID 19 Vaccines are proven to cause each of these Inflammatory Oxidative Stressors in 28 different tissues in the

body, including brain, heart, kidney, liver, intestines and many others… these are the IOS indicated markers found in our blood, that lead to ALL diseases…

C-Reactive Protein (CRP)
Tumor Necrosis Factor (TNF-a)
Interleukin-6 (IL-6)
Reactive Oxidative Stress (ROS)
Superoxide Dismutase (SOD) activity

Vitamin C is proven to REDUCE ALL of these, this 2014 study shows VIT C ability to reduce ALL the markers of Inflammatory Oxidative Stress that comes from getting community acquired pneumonia… Can you recall what COVID 19 did to all seriously ill hospitalized patients, it caused secondary pneumonia! Vit C would have saved 100's of thousands of lives! Read the last sentence of the abstract, severe pneumonia was mitigated… death by Covid was complicated by contracted severe pneumonia. So many American Lives could have been saved!

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tps://pubmed.ncbi.nlm.nih.gov/25253919/

Vitamin C mitigates oxidative stress and tumor necrosis factor-alpha in severe community-acquired pneumonia and LPS-induced macrophages

Yuanyuan Chen [1], Guangyan Luo [2], Jiao Yuan [1], Yuanyuan Wang [1], Xiaoqiong Yang [1], Xiaoyun Wang [1], Guoping Li [3], Zhiguang Liu [4], Nanshan Zhong [5]

Affiliations + expand

PMID: 25253919 PMCID: PMC4165740 DOI: 10.1155/2014/426740

Free PMC article

Abstract

Oxidative stress is an important part of host innate immune response to foreign pathogens. However, the impact of vitamin C on oxidative stress and inflammation remains unclear in community-acquired pneumonia (CAP). We aimed to determine the effect of vitamin C on oxidative stress and inflammation. CAP patients were enrolled. Reactive oxygen species (ROS), DNA damage, superoxide dismutases (SOD) activity, tumor necrosis factor-alpha (TNF-α), and IL-6 were analyzed in CAP patients and LPS-stimulated macrophages cells. MH-S cells were transfected with RFP-LC3 plasmids. Autophagy was measured in LPS-stimulated macrophages cells. Severe CAP patients showed significantly increased ROS, DNA damage, TNF-α, and IL-6. SOD was significantly decreased in severe CAP. Vitamin C significantly decreased ROS, DNA damage, TNF-α, and IL-6. Vitamin C inhibited LPS-induced ROS, DNA damage, TNF-α, IL-6, and p38 in macrophages cells. Vitamin C inhibited autophagy in LPS-induced macrophages cells. These findings indicated that severe CAP exhibited significantly increased oxidative stress, DNA damage, and proinflammatory mediator. Vitamin C mitigated oxidative stress and proinflammatory mediator suggesting a possible mechanism for vitamin C in severe CAP.

The most common blood marker looked for to determine Heart attacks or strokes is CRP, C-Reactive Protein. CRP is not heart muscle or tissue specific, if can represent damage to any organs in the body. But check out this study… Vit C Proven to reduce damage to cells in the heart and rest of the body, thus lowering ALL totals of CRP circulating in the blood!

https://pubmed.ncbi.nlm.nih.gov/30332942/

A Meta-analysis of Randomized Control Trials: The Impact of Vitamin C Supplementation on Serum CRP and Serum hs-CRP Concentrations

Sadegh Jafarnejad [1], Virginia Boccardi [2], Banafshe Hosseini [3], Mohsen Taghizadeh [1], Zahra Hamedifard [1]

Affiliations + expand

PMID: 30332942 DOI: 10.2174/1381612824666181017101810

Abstract

Objective: The present meta-analysis was designed to assess the effects of vitamin C supplementation on serum C-reactive Protein (CRP) levels.

Methods: We conducted a comprehensive systematic search of the literature in Scopus, PubMed and Google Scholar until May 2018. The pooled Weighted Mean Difference (WMD) and its 95% Confidence Interval (CI) in baseline and at the end of the trial were calculated to assess the net change in serum CRP by using random-effects model. The heterogeneity was assessed by I2 test. Combined and stratified analyses were used in the metaanalysis.

Results: From 306 articles found and screened in our initial search, 12 studies were included with 446 participants in supplementation groups and 447 in control groups. The pooled effect size analysis showed a significant reducing effect of vitamin C supplementation on circulating CRP level (-0.23 mg/L, 95% CI, -0.44, -0.03, p=0.02), with a significant heterogeneity effect across the studies involved. Subgroup analyses showed that vitamin C supplementation significantly lowered CRP among trials. The most significant effect was found 1) on hs- CRP as the representative inflammatory marker (-0.43 mg/L, 95% CI -0.76, -0.1) 2) in subjects with a baseline CRP≥3 (-1.48 mg/L, 95% CI -2.84, -0.11) 3) in subjects under <60 years old of age (-0.23 mg/L, 95% CI -0.44,-0.01) 4) or using intravenous administration of vitamin C (-0.89 mg/L, 95% CI -1.49,-0.3).

What about Magnesium's impact on C-Reactive Protein, can it improve damage to our heart cells and other organs also, by demonstrating lowered CRP levels in the blood. Incredibly yes!

https://pubmed.ncbi.nlm.nih.gov/17479208/

Magnesium and C-reactive protein in heart failure: an anti-inflammatory effect of magnesium administration?

Dorit Almoznino-Sarafian [1], Sylvia Berman, Anat Mor, Miriam Shteinshnaider, Oleg Gorelik, Irma Tzur, Irena Alon, David Modai, Natan Cohen

Affiliations + expand

PMID: 17479208 DOI: 10.1007/s00394-007-0655-x

Abstract

Background: Little is known about the relationship between serum magnesium (Mg) and C-reactive protein (CRP) in heart failure (HF).

Aim of the study: To investigate the relationship, if any, between serum Mg and CRP in HF patients and, concomitantly, to test a hypothesis that Mg supplementation might affect serum CRP levels.

Conclusion was…

Results: Inverse correlation was found between serum Mg and log CRP (r = -0.28, P = 0.002). Compared to controls, patients with HF demonstrated higher baseline CRP levels, independent of coexisting conditions, and lower serum Mg values. Following Mg treatment, log CRP decreased from 1.4 +/- 0.4 to 0.8 +/- 0.3 in group A (P < 0.001). No significant changes in log CRP were demonstrable in group B. Serum Mg (mmol/l) rose significantly in group A (0.74 +/- 0.04-0.88 +/- 0.08, P < 0.001), and to a lesser extent in group B (0.82 +/- 0.08-0.88 +/- 0.08, P = 0.04). Intracellular Mg significantly increased only in Mg-treated group A (P = 0.01).

Conclusions: Oral Mg supplementation to HF patients significantly attenuates blood levels of CRP, a biomarker of inflammation. Targeting the inflammatory cascade by Mg administration might prove a useful tool for improving the prognosis in HF.

Here is another study showing that **Low Levels of Magnesium alone INCREASES CRP and Tumor Necrosis Factor (cancer causing enzyme) levels in the blood!**

https://pubmed.ncbi.nlm.nih.gov/21609903/

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Severe hypomagnesemia and low-grade inflammation in metabolic syndrome

Fernando Guerrero-Romero [1], Carmen Bermudez-Peña, Martha Rodríguez-Morán

Affiliations + expand

PMID: 21609903 DOI: 10.1684/mrh.2011.0281

Free article

Abstract

To evaluate the association between severe hypomagnesemia and the low-grade inflammatory response in subjects with metabolic syndrome (MetS), ninety-eight individuals with new diagnosis of MetS were enrolled in a cross-sectional study. Pregnancy, smoking, alcohol intake, renal damage, hepatic disorders, infectious or chronic inflammatory diseases, malignancy, use of diuretics, statins, calcium antagonist, antioxidants, vitamins, anti-inflammatory drugs, or previous oral magnesium supplementation were exclusion criteria. According serum magnesium levels, participants were assigned to the following groups: 1) severe hypomagnesemia (≤1.2 mg/dL); 2) hypomagnesemia (>1.2≤1.8 mg/dL); 3) Normal serum magnesium levels (>1.8 mg/dL). The low-grade inflammatory response was defined by elevation of serum levels of (hsCRP >1.0 ≤10.0 mg/L) or TNF-alpha (TNF-α ≥3.5 pg/mL). Severe hypomagnesemia, hypomagnesemia, and normomagnesemia were identified in 21 (21.4%), 38 (38.8%), and 39 (39.8%) individuals. The ORs, adjusted by WC, showed that severe hypomagnesemia (OR: 8.1; CI 95%: 3.6-19.4 and OR: 3.7; CI 95%: 1.1-12.1), but not hypomagnesemia (OR: 1.8; CI 95%: 0.9-15.5 and OR: 1.6; CI 95%: 0.7-3.6), was strongly associated with elevated hsCRP and TNF-α levels, and that normomagnesemia exhibited a protective role (OR: 0.32; CI 95%: 0.1-0.7 and OR: 0.28; CI 95%: 0.1-0.6) for elevation of CRP and TNF-α. Results of this study show that, in subjects with MetS, severe hypomagnesemia, but not hypomagnesemia, is associated with elevated concentrations of CRP and TNF-α.

For all the science minds wanting to learn more about just how the spike proteins of the COVID 19 Virus and The Spike Proteins elicited in your body from the COVID 19 and how they cause oxidative stress… check out and read this published article. The exploration is the Spike proteins ability to damage cell receptors called ACE2 inhibitors, that then set the stage to infect a human cell. This article explains exactly what the spike protein dose to cause OXIDATIVE STRESS to disease the cell and organs in the human body.

https://pubs.acs.org/doi/full/10.1021/acsomega.0c02125

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Impact of Thiol–Disulfide Balance on the Binding of Covid-19 Spike Protein with Angiotensin-Converting Enzyme 2 Receptor

Sanchita Hati and Sudeep Bhattacharyya*

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https://doi.org/10.1021/acsomega.0c02125
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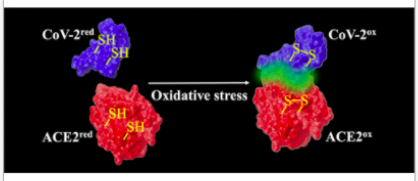
SUBJECTS: Disulfides, Redox reactions, Free energy, Peptides and proteins, Receptors

Abstract

The novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), has led to an ongoing pandemic of coronavirus disease (COVID-19), which started in 2019. This is a member of Coronaviridae family in the genus *Betacoronavirus*, which also includes SARS-CoV and Middle East respiratory syndrome coronavirus (MERS-CoV). The angiotensin-converting enzyme 2 (ACE2) is the functional receptor for SARS-CoV and SARS-CoV-2 to enter the host cells. In particular, the interaction of viral spike proteins with ACE2 is a critical step in the viral replication cycle. The receptor-binding domain of the viral spike proteins and ACE2 have several cysteine residues. In this study, the role of thiol–disulfide balance on the interactions between SARS-CoV/CoV-2 spike proteins and ACE2 was investigated using molecular dynamics simulations. The study revealed that the binding affinity was significantly impaired when all of the disulfide bonds of both ACE2 and SARS-CoV/CoV-2 spike proteins were reduced to thiol groups. The impact on the binding affinity was less severe when the disulfide bridges of only one of the binding partners were reduced to thiols. This computational finding possibly provides a molecular basis for the differential COVID-19 cellular recognition due to the oxidative stress.



Magnesium studies confirm that Magnesium promotes CHONDROGENISIS which are cells that repair and grow new cartilage in the body after damage from disease or surgery or injury! For those that don't know, cartilage is one of the supposed most difficult tissue to regrow or repair.

Trust the healing power of **Magnesium and Vitamin C and Selenium as these 3 nutrients provide protection against ALL Intracellular Oxidative Stress**! **It is my clinical and professional opinion that at this moment in time there is nothing better you can do than to take these 3 nutrients required by the body to protect it from ALL disease pathways.**

Selenium is essential at promoting Glutathione production in the liver and Glutathione is protective against ALL Oxidative Stress created by all infection or poisons also!

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Let's explore benefits of Selenium:
http://www.immunehealthscience.com/benefits-of-selenium.html

Benefits of selenium and its role as a glutathione cofactor

Health benefits of selenium boil down to its crucial antioxidant role as part of the enzyme glutathione peroxidase (GPx).

Glutathione peroxidases, also known as selenoproteins, are a family of antioxidant enzymes that speed up the reaction between glutathione and free radicals, particularly toxic hydrogen peroxide, which selenium-containing glutathione peroxidases help transform to harmless water. These enzymes act both inside and outside the cells maximizing antioxidant protection.

Article Continues:

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The benefits of selenium are the result of the activity of glutathione in our bodies. Research done with selenium proves this connection – low selenium levels are noted in the health conditions that show low glutathione levels. The progression of all these conditions and success with therapies depend on glutathione levels and proper functioning of glutathione peroxidases:

- heart diseases
- atherosclerosis
- liver diseases
- kidney diseases
- pancreatitis
- HIV/AIDS
- Crohn's disease
- immune system disorders
- cataracts
- impaired detoxification of heavy metals (arsenic, cadmium, mercury and lead)
- asthma
- cystic fibrosis
- cancers
- Parkinson's disease
- Alzheimer's disease
- and more

One recent randomized clinical trial investigated the effects and benefits of selenium supplementation in forty-five patients with chronic kidney disease. The participants supplemented daily with 200 mcg of selenium for three months. Plasma selenium concentrations and red blood cell activity of glutathione peroxidase increased significantly in all patients. (*Effect of selenium supplementation on glutathione peroxidase enzyme activity in patients with chronic kidney disease: a randomized clinical trial.* Sedighi O, Zargari M et al. Nephrourol Mon. 2014 May 4;6(3):e17945. doi: 10.5812/ numonthly. 17945.)

Supplementing 200mcg of Selenium Daily will promote the liver to increase levels of disease protecting Glutathione to be produced. Here is one study demonstrating the Glutathione ability to protect against Oxidative stress and thus protect against ALL diseases.

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Protective Effect of Glutathione against Oxidative Stress-induced Cytotoxicity in RAW 264.7 Macrophages through Activating the Nuclear Factor Erythroid 2-Related Factor-2/Heme Oxygenase-1 Pathway

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Abstract

Reactive oxygen species (ROS), products of oxidative stress, contribute to the initiation and progression of the pathogenesis of various diseases. Glutathione is a major antioxidant that can help prevent the process through the removal of ROS. The aim of this study was to evaluate the

Disease Protection Protocol is on the Next Page

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COVID 19 and COVID 19 VACCINE
SHEDDING, DISEASE & DEATH PROTECTION PROTOCOL

Vitamin C (ascorbic acid)

10,000mg: divided into two or 3 doses/day
Children 6- 12 (5,000mg/day)
Children 2-4 (2,000mg/day)

Note: Vitamin C best practice for appropriate dosing (for ALL AGES) is to start with 1 or 2k mg a day and add 1000mg every day until you get diarrhea. Whatever amount you took the day before loose stools occurred is the best dose for your body daily to take. Every person is different, some people have bowel tolerance of 2k mg per day, and some people daily take 20k, 30k, even 50k mg without loose stools or bowel tolerance ever occurs. I need to be clear, there is NO TOXIC dose of VIT C that has ever been determined, EVER. The best practice is to take as much daily that does not cause loose stools and stay on that indefinitely. I order my VIT C in bulk from bulksupplements.com. During periods of viral infections, I suggest during days of symptoms to be sure to get 3k mg daily from the form of VIT C called Liposomal Vit C, LivOnLabs has my favorite form, and it can be found online. The remaining daily dose during sickness can come from typical forms of VIT C (ascorbic acid).

Magnesium Chloride

13 years old and up: Start with 500mg
Children 6- 12 (300mg/day or just below bowel tolerance
Children 2-4 (200mg/day)

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According to Cardiologist Thomas Levy MD, research also supports there is NO TOXIC amount of Magnesium for the human body. Levy explains that Magnesium daily dosing to prevent ALL disease processes is determined the same way I described how to determine highest amount of daily VIT C. Start with 500mg of Magnesium Chloride and daily increase by 100mg until loose bowels occur then back down when that occurs, to the amount you took the day before, and stay on this just below loose bowel dose every day. I also recommend as he does to take that much magnesium everyday of your life. To learn more about the incredible healing power of magnesium and disease prevention of Magnesium, check out his book published in 2019, Magnesium Reversing Disease. @ medfoxpub.com

Selenium
200mcg daily

Note: Every day forever! My favorite forms are chelated forms of Selenium, check the Supplement Facts Panel for form.