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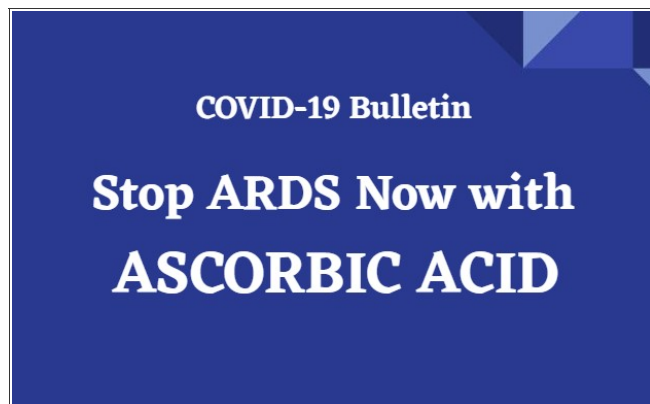
STOP ARDS NOW WITH ASCORBIC ACID

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ACE2 ACUTE RESPIRATORY DISTRESS SYNDROME ANTIOXIDANTS ARDS ASCORBIC ACID BHA CELL-FREE HEMOGLOBIN
 CORONAVIRUS COVID19 CYT561 EDEMA FLUIDS FLURIN H1F1A HYPOXIA IVC LUNGS NITRIC OXIDE
 OXIDANTS PANDEMIC PORPHYRINS RED BLOOD CELLS REDOX POTENTIAL S PROTEIN SODIUM ASCORBATE
 SODIUM BICARBONATE SVCT1 SVCT2 VIROPORIN VITAMIN C

17639 VISUALIZZAZIONI



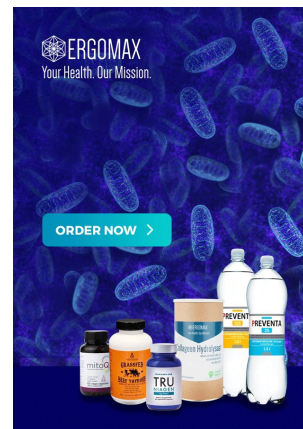
28 MAR Why Oral Ascorbic Acid Combined with IV C is the Ultimate Two-Pronged Attack That Will Turn the Tide on COVID-19 Pandemic – DorisLite Version



By Doris Loh



This article contains slides and notes from an exclusive webinar delivered to healthcare professionals on March 25, 2020. The central message in the presentation emphasized the oral form of ascorbic acid is a redox molecule that cannot be replaced by other antioxidants, perhaps even sodium ascorbate. The combination of high-dose oral ascorbic acid together with intravenous sodium ascorbate could



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significantly reduce the exponential increase of COVID-19 patients who require intensive care treatment after infection.

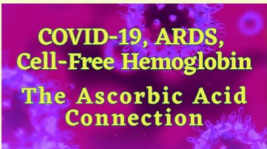
This article also includes a newly released study that identified the exact mechanisms used by SARS-CoV2 to destroy hemoglobin, resulting in cell-free heme that exacerbates ARDS.

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This Bulletin includes **NEWLY RELEASED** information on how COVID-19 attacks hemoglobin to cause ARDS

**COVID-19, ARDS & Cell-Free Hemoglobin
The Ascorbic Acid Connection**
By Doris Loh

<https://www.evolutamente.it/covid-19-ards-cell-free-hemoglobin-the-ascorbic-acid-connection/>



I want to use the time today to explain to all of you why the oral form of ascorbic acid must be used to reduce COVID-19 symptoms, especially the lethal ARDS, acute respiratory distress syndrome.

Most people think all the different forms of Vitamin C are the same. They are not. This difference can mean life or death in COVID-19 infected patients.

Acute Respiratory Distress Syndrome

- Increased ARDS in COVID-19 patients overwhelms healthcare systems
- 41.8% of COVID-19 patients develop ARDS¹
- Half of critical ARDS patients die
- SARS-CoV2 Spike Protein Binds to ACE2 Receptors²
 - ACE2 concentrated in AT2 (type II alveolar cells)
 - AT2 increase viral reproduction and transmission
- Furin Enzymes activate SARS-CoV2

1 Wu C, Chen X, Cai Y, et al. Risk Factors Associated With Acute Respiratory Distress Syndrome and Death in Patients With Coronavirus Disease 2019 Pneumonia in Wuhan, China. *JAMA Intern Med.* Published online March 13, 2020. doi:10.1001/jamainternmed.2020.0994
2 Yu Zhen, Zouan Zhao, Yujie Wang, Tingting Zhou, Yu Ma, Wei Zuo. *bioRxiv* 2020.01.26.919985; doi: https://doi.org/10.1101/2020.01.26.919985

Critically ill COVID-19 patients often develop acute respiratory distress syndrome (ARDS). When that happens, intubation and invasive mechanical ventilation is required to assist difficulty in breathing. Common ARDS symptoms include rapid accumulation of fluid in airspace, leading to edema and inflammation.

WHY DOES ARDS DEVELOP SO RAPIDLY?

SARS-CoV2 (virus) that causes COVID-19 (disease) binds to receptors for ACE2 (Angiotensin-converting enzyme 2). ACE2 receptor expression is concentrated in a small population of type II alveolar cells (AT2) in the lungs.

The main problem is, AT2 also expresses many other genes that increase viral reproduction and transmission. This means once SARS-CoV2 is activated in the lungs, the patient can deteriorate VERY QUICKLY, and if they do not have adequate ascorbic acid, they may progress to ARDS.

Why is Ascorbic Acid Important?

SARS-CoV-2 virus needs to be activated at the cleavage site in spike proteins. This cleavage site uses furin enzymes. This is the beginning of the story where SARS-CoV2 and ascorbic acid become enemies.

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COVID-19, ARDS & CYTOKINE STORMS – THE RECYCLING OF ASCORBIC ACID BY MACROPHAGES, NEUTROPHILS AND LYMPHOCYTES
5 APRILE 2020

IL COVID-19 E' UN VIRUS SELVAGGIO O CREATO IN LABORATORIO? PARTE 2
4 APRILE 2020

IL COVID-19 E' UN VIRUS SELVAGGIO O CREATO IN LABORATORIO? (PRIMA PARTE)
3 APRILE 2020

STOP ARDS NOW WITH ASCORBIC ACID
28 MARZO 2020

1 COVID-19, PNEUMONIA & INFLAMMASOMES – THE MELATONIN CONNECTION
14 MARZO 2020

2 LA FIBROMIALGIA E LA SINDROME DELLA STANCIEZZA CRONICA POTREBBERO ESSERE CAUSATE DAL GLUTINE...

STOP ARDS NOW

As of March 25, 2020

- 30,000 COVID-19 cases in NY State¹
- NY morgues expected to reach capacity in one week¹
- 40,000 will be in ICU in about 2 weeks¹
- NY hospitals are now using Vitamin C to treat COVID-19 patients²
- The form of Vitamin C used can make a huge difference in ARDS

¹ https://www.dailymail.co.uk/news/article-8151685/Make-shift-morgues-set-outside-NYC-hospital-amid-coronavirus.html?ito=social-tweet_rail_mails
² https://www.techtimes.com/articles/248118/20200324/overdose-of-vitamin-c-actually-effective-against-covid-19-doctors-think-so.htm?cid=swar_5m28J7Y9K629EY-c1_L05xa9B-542NTu05NcmMKRvU9NC4Fpg

Hospitals around the world, especially those in China, are using IV C for COVID-19 because it is extremely effective.

However, my recent research revealed that Vitamin C in the form of ascorbic acid may actually prevent COVID-19 patients from developing ARDS.

This is currently the NUMBER ONE priority for all healthcare systems around the world – how to reduce ARDS patient load in the ICU.

IV C is Extremely Effective for ARDS Patients

- IV C uses sodium ascorbate (SA)
- Liposomal Vit C is mostly in the form of sodium ascorbate
- IV C can shorten length of hospital stay and speed recovery

Intravenous delivery of Vitamin C must be buffered to match physiological pH. Most standard intravenous solutions use sodium ascorbate, which is ascorbic acid combined with sodium carbonate. The pH of pure ascorbic acid is too low for plasma delivery.

Many people cannot tolerate ascorbic acid and thus use liposomal forms. Most commercially available liposomal Vitamin C use the sodium ascorbate form of Vitamin C.

The question is, can sodium ascorbate prevent COVID-19 patients from developing ARDS?

Ascorbic Acid versus Sodium Ascorbate

- Ascorbic acid has a different molecular structure from Sodium ascorbate

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4 COVID-19, ARDS & CELL-FREE HEMOGLOBIN – THE ASCORBIC ACID CONNECTION

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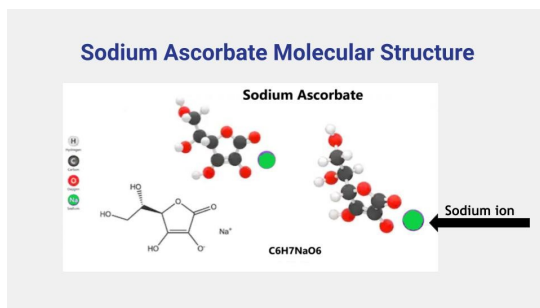
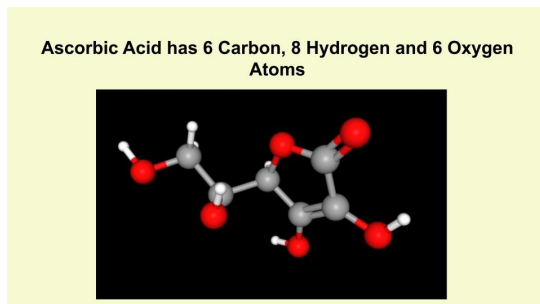
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When sodium bicarbonate is added to ascorbic acid, the parent molecule changes its molecular structure and becomes sodium ascorbate.

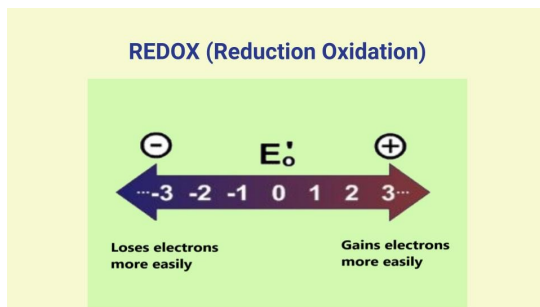


Sodium ascorbate has 6 carbon, 7 hydrogen, 6 oxygen, and one sodium atom that replaces the 8th hydrogen in its parent.

The addition of the sodium ion makes this molecule more reactive and less stable than its parent, ascorbic acid.

Thus, it is actually 'better' as an antioxidant, but this also makes it less suitable as a REDOX molecule.

Why?



REDOX reactions always involve a pair of molecules; one accepts an electron, the other donates an electron.

Energy of REDOX is measured in electron volts. One electron volt = 1000 mV.

The lower (more negative) the voltage, the easier it is for the molecule to give up or lose its electrons. The higher (more positive) the voltage, the easier it is for the molecule to grab or gain electrons.

Oxidants have high reduction potentials, with large positive numbers

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Oxidants have high reduction potentials, with large positive numbers.

Antioxidants are the exact opposite. They have low reduction potentials with large negative numbers.

Ascorbic Acid is a REDOX Molecule

Reduction potentials:

- Ascorbic acid + 282 mV¹
- Glutathione - 240 mV at pH 7.0²

1. Akbari A., Jelodar G, Nazifi S, Sajedianfard J. An Overview of the Characteristics and Function of Vitamin C in Various Tissues: Relying on its Antioxidant Function, Zahedan J Res Med Sci. 2016; 18(11):4037. doi: 10.17795/zjms-4037
2. Jones DP. Redox potential of GSH/GSSG couple: assay and biological significance. Methods Enzymol. 2002;348:93-112. doi:10.1016/0076-6875(02)40039-2

When you compare ascorbic acid with a standard antioxidant like glutathione, you will see why ascorbic acid is really not a powerful antioxidant.

Ascorbic acid reduction potential is + 0.282 eV;
Glutathione is - 240 mV at pH 7.0

Between ascorbic acid and glutathione, there is a huge difference of 522 mV, or more than half an electron volt. Therefore, compared to glutathione, ascorbic acid is quite stable and unreactive.

Ascorbic Acid Regeneration

- NADH/ NADPH/ GSH are antioxidants
- NAD⁺/NADH ~ -320 mV¹
- NADP⁺/NADPH ~ -400 mV²

1. Xiao W, Wang RS, Handy DE, Loscalzo J. NAD(H) and NADP(H) Redox Couples and Cellular Energy Metabolism. Antioxid Redox Sign. 2016;28(3):251-272. doi:10.1089/ars.2017.7216
2. Hoedeker F.H, Carito C, Wenzerski F.J, Auwers J. The secret life of NAD⁺: an old metabolite controlling new metabolic signaling pathways. Endocr Rev. 2010;31(2):194-223. doi:10.1210/er.2009-0026

Ascorbic acid can be regenerated (reduced) by NADH, NADPH and Glutathione.

Due to the low reduction potentials of these molecules, NADH, NADPH and Glutathione are all powerful antioxidants.

NADPH has a lower reduction potential than glutathione; that is why NADPH is used to regenerate (reduce) glutathione.

ARDS in COVID-19

- ARDS is caused by cell-free hemoglobin (CFH)
- Iron in CFH is oxidized (Ferric)
- CFH causes permeability in membranes
- CFH decreases intracellular ascorbate
- Increasing intracellular ascorbate preserves barrier integrity

Kuck JL, Bastarache JA, Shaver CM, et al. Ascorbic acid attenuates endothelial permeability triggered by cell-free hemoglobin. Biochem Biophys Res Commun. 2018;495(1):433-437. doi:10.1016/j.bbrc.2017.11.058

Cell-free hemoglobin are unstable heme within hemoglobin with oxidized ferric iron centers. They bind to proteins and lipids, causing inflammatory free radical chain reactions such as lipid peroxidation.

Healthy endothelial cells maintain tight barriers that

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limits permeability.

CFH decreases intracellular ascorbate. CFH increases permeability inducing loss of barrier function that allows the passage of large macromolecules.

New study released on March 23, 2020, revealed the STARTLING MECHANISMS used by SARS-CoV2 to create Cell-Free Hemoglobin and Induce ARDS

COVID-19: Attacks the 1-Beta Chain of Hemoglobin and Captures the Porphyrin to Inhibit Human Heme Metabolism

Wenzhong Liu^{1,2*}, Hualan Li²

¹ School of Computer Science and Engineering, Sichuan University of Science & Engineering, Zigong, 643002, China;

² School of Life Science and Food Engineering, Yibin University, Yibin, 644000, China;

* Correspondence: liwz@suse.edu.cn;

COVID-19 Coronavirus is Dependent on Porphyrins



- COVID-19 Patients have low hemoglobin and neutrophils.
- Patient serum ferritin are significantly elevated. Ferritin binds free iron.
- COVID-19 **TARGETS hemoglobin and ATTACKS heme**.

SARS-CoV2 viroprotein Orf3a and other proteins Orf8, Orf10, Orf1ab attack hemoglobin. These proteins **DISSOCIATE iron from heme, oxidizing and releasing the iron**. After iron is removed, SARS-CoV2 proteins capture heme to form porphyrins without iron centers.

SARS-CoV2 viroprotein E protein binds to porphyrins. This allows COVID-19 to **DIRECTLY penetrate human cell membranes through porphyrin** in addition to ACE2 receptor binding by spike proteins.

- The authors concluded that coronavirus is an ancient life form that is dependent on porphyrins, or heme without iron.

Oral Ascorbic Acid for ARDS Prevention

- Ascorbic Acid turns oxidized ferric iron into ferrous iron
- Ascorbic acid is used exclusively by transmembrane redox enzymes for reduction of iron (ferric  ferrous)
- Red blood cells use ascorbic acid exclusively to manage iron redox
- Oxidized iron in heme cannot carry oxygen
- Hemoglobin with oxidized iron loses heme rapidly
- Cell-Free heme (CFH)  ARDS
- Ascorbic acid increases barrier integrity, prevents damages by CFH

Kuck et al., Badierische JA, Shaver CM, et al. Ascorbic acid attenuates endothelial permeability triggered by cell-free hemoglobin. *Biochem Biophys Res Commun.* 2018;495(1):433-437. doi:10.1016/j.bbrc.2017.11.058


Our bodies use electrons from ascorbic acid to turn the unstable oxidized iron back into the stable form. Ascorbic acid can add an electron back to the oxidized ferric iron, reducing it back into ferrous iron.

Erythrocytes have transmembrane enzymes that exclusively use ascorbic acid to add electrons back to oxidized iron, keeping them stable in the ferrous form, maintaining heme stability in hemoglobin.

Oxidized iron is unable to transport oxygen because it cannot bind oxygen. Cell-free heme with oxidized iron do not carry oxygen. They are the primary reason why ARDS patients cannot breathe and require intubation with invasive mechanical ventilation.

Ascorbic acid maintains heme stability, increasing oxygen-carrying ability of heme. Ascorbic acid prevents damage by CFH by restoring resistance to current flow, barrier integrity and reducing permeability.

How Cell-Free Hemoglobin Leads to ARDS

- CFH destroys nitric oxide¹
- Nitric Oxide suppresses SARS-CoV viral replication²
- Less nitric oxide  vasoconstriction, inflammatory cytokines, cellular damages³
- Ascorbic Acid increases nitric oxide

¹ Gaggler A, Patel RP. There is blood in the water: hemolysis, hemoglobin, and heme in acute lung injury. *Am J Physiol Lung Cell Mol Physiol.* 2016;311(4):L714-L718. doi:10.1152/ajplung.00312.2016

² Sara Alvestrom, Mehrez Mousavi-Jazi, Jonas Klingstrom, Mikael Leijon, Ake Lundkvist, Ali Mirzazini

Journal of Virology Jan 2005, 79 (3) 1966-1969, DOI: 10.1128/JVI.79.3.1966-1969.2005

³ Clara M. Shaver, Cameron P. Uebachs, David R. Jantz, Brandon S. Grove, Nathan D. Pritz, Nancy E. Wickersham, Sergey I. Dikalov, Lorraine B. Ware, Julie A. Badierische. *Am J Physiol Lung Cell Mol Physiol.* 2016 Mar 15; 310(6): L532-L541. Published online 2016 Jan 15. doi: 10.1152/ajplung.00155.2015

Free heme can scavenge nitric oxide (NO) up to 1,000 times faster than stable heme bound in red blood cells.


Loss of endogenous nitric oxide reduces the ability to suppress viral replication.

The rapid loss of endothelial nitric oxide bioavailability leads to hypertension, coagulation and development of systemic inflammation.

That is why COVID-19 patients with hypertension are at higher risks due to vasoconstriction created by nitric oxide depletion.

90% of inpatients with pneumonia have been found with increased coagulation activity, as a result of endothelial nitric oxide depletion.

Oral Ascorbic Acid Increases Nitric Oxide

- BH4 - Rate-limiting enzyme in Nitric Oxide production
- Ascorbic Acid regenerates BH4  MORE Nitric Oxide
- Exclusive Use of Ascorbic Acid in BH4 recycling¹
 - Thiols are INEFFECTIVE in regenerating BH4²

¹ Kuzkaya N, Weisemann N, Harrison DG, Dikalov S. Interactions of peroxynitrite, tetrahydrobiopterin, ascorbic acid, and thiols: implications for uncoupling endothelial nitric-oxide synthase. *J Biol Chem*. 2003;278(25):22546-22554. doi:10.1074/jbc.M30227200

² Mirzahasanini A, Nosrati B. Species-Specific Standard Redox Potential of Thiol-Disulfide Systems: A Key Parameter to Develop A Agents against Oxidative Stress. *Sci Rep* 6, 37596 (2016). <https://doi.org/10.1038/srep37596>

Endogenous nitric oxide can suppress viral replication. Nitric oxide production is dependent on the rate-limiting cofactor called tetrahydrobiopterin (BH4).




BH4 must be maintained in a reduced redox state during nitric oxide production. If BH4 is oxidized by free radicals and not subsequently regenerated, biosynthesis of nitric oxide cannot proceed.

BH4 regeneration is dependent upon ascorbic acid. Lack of ascorbic acid will lower endogenous nitric oxide production. Other antioxidants like thiols (glutathione) have been shown to be totally ineffective in the regeneration of BH4.

There is no substitution for ascorbic acid in nitric oxide production.

Ascorbic Acid is a Powerful Masterkey

A single ascorbic acid molecule opens many different biological doors

- Iron
 - CFH (Ferric Iron)  ARDS
 - Parkinson's¹ - Elevated Ferric Iron  Loss of Dopamine
- Mitochondria
 - SARS-CoV2 Viroporins - Membrane Permeabilization  Cell Death
- SARS-CoV2 Furin Cleavage Increased by Hypoxia
 - CFH Increases Hypoxia (lack of O₂)
 - Oxygen and Ascorbic Acid degrade HIF1a
 - Ascorbic Acid inhibits HIF1a stabilization, limiting Furin enzyme expression

¹ Pointing CP. Domain homologues of dopamine beta-hydroxylase and ferric reductase: roles for iron metabolism in neurodegenerative disorders? *Human Molecular Genetics*. 2001 Aug;10(17):1853-1858. DOI: 10.1093/hmg/10.17.1853.

The loss of dopamine in Parkinson's disease is the direct result of high oxidized ferric iron. Transmembrane Cytochrome b561 (Cytb561) uses ascorbic acid to manage iron homeostasis.

SARS-CoV2 coronavirus uses viroporins (E, Orf3a) to increase viral replication. Viroporins attack membranes causing depolarization. Mitochondria depolarization leads to cell death. Ascorbic acid is used by mitochondrial transmembrane enzymes VDAC1 to provide an alternate source of energy, preventing mitochondria from total collapse during depolarization events.

SARS-CoV2 spike protein binds to ACE2 receptors. Spike protein must be activated by furin enzymes. Furin enzymes are double-edged swords as more than 100 proteins must be cleaved by furin enzymes before they become bioactive. Growth factors, cytokines, hormones, collagens, all kinds of receptors and membrane channels are cleaved by furin.

Maintaining furin enzyme homeostasis is critical during COVID-19 infection. Hypoxia disturbs furin balance by increasing expression of furin enzymes.

Lack of oxygen activates HIF1a in the hypoxia response pathway. Without oxygen and/or ascorbic acid, HIF1a will bind with HIF1b to form a stable complex. The formation of this complex will activate many genes, including the one that codes for furin enzymes.

More CFH means less oxygen. Less oxygen activates HIF1a. HIF1a will result in more furin enzymes. More furin enzymes will cleave more SARS-CoV2 virus for activation.

This is an EXTREMELY VICIOUS CYCLE that can be stopped quickly and effectively by supplying high levels of ascorbic acid as redox molecule.



Ascorbic Acid Intestinal Absorption Capacity

- High capacity SVCT1 can transport LARGE amounts of ascorbic acid quickly
- Higher pH of Sodium Ascorbate slows absorption

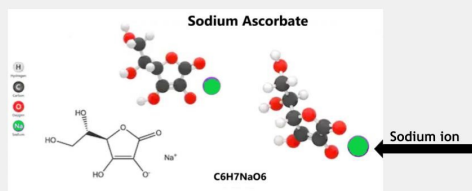
There is a preference in using IV C because most believe oral ascorbic acid cannot be absorbed in large quantities and efficiently. The thought is that absorption of ascorbic acid above 200 milligrams is greatly diminished. This is an incorrect interpretation of how ascorbic acid is utilized in our bodies.

There are two main Vitamin C transporters called sodium-dependent vitamin C transporters – SVCT1 and SVCT2.

In the stomach, where acidity is high and pH is low, ascorbic acid is rapidly absorbed by SVCT1, the LOW-AFFINITY, HIGH-CAPACITY transporter. SVCT1 will only transport in huge quantities when there is a high concentration of ascorbate.

If the pH is raised in the stomach, as in the case of sodium ascorbate, the transport becomes slower because the buffered stomach pH would inhibit and slow absorption.

Sodium Ascorbate Molecular Structure



It is entirely possible that sodium ascorbate is transported mainly by the high-affinity but LOW-CAPACITY SVCT2.

Do we have any evidence of this distinct difference between how ascorbic acid is absorbed in the intestines?

Owen Fonorow and Steven Hickey
March 13, 2020

TOWNSEND LETTER
The Journal of Alternative Medicine
Thursday, March 16, 2020

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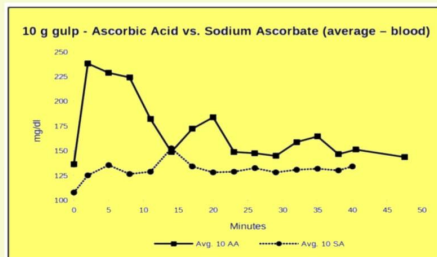
published March 11, 2020, prior to print publication.

© Owen Fonorow and Steve Hickey

The groundbreaking discovery by Owen Fonorow and Steven Hickey published on March 13, 2020, revealed without any

Without doubt, that ascorbic acid is a REDOX molecule that is constantly recycled, reused and regenerated by the human body.

Ascorbic Acid Achieves Higher Plasma Levels Than Sodium Ascorbate



This is a chart tracking minute-by-minute fluctuations of 10 grams of ascorbic acid and 11.3 grams of sodium ascorbate after single dose ingestions. 11.3 grams of sodium ascorbate was given to account for the additional weight of sodium in the compound.

Oral ascorbic acid levels in plasma almost doubled that of sodium ascorbate at the two-minute mark. The highest level reached by sodium ascorbate could not exceed the lowest point reached by ascorbic acid around the 14-minute mark.

Ascorbic Acid Intestinal Absorption Capacity

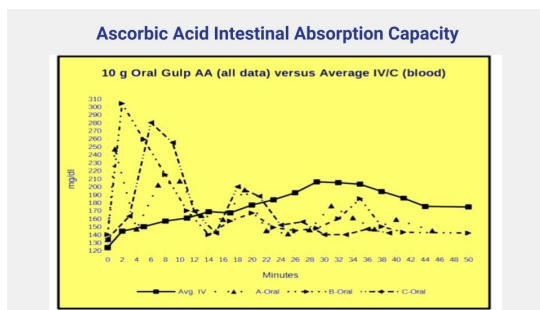
- High capacity SVCT1 can transport LARGE amounts of ascorbic acid quickly
- Higher pH of Sodium Ascorbate slows absorption

The only reason that could have accounted for the low level of plasma sodium ascorbate is the addition of the sodium ion, producing higher pH that causes this molecule to be transported by the high-affinity but **LOW-CAPACITY SVCT2**.

The following chart is the consummation of the recent work by Fonorow and Hickey. Minute-to-minute fluctuations in plasma levels of ascorbic acid and sodium ascorbate showed regeneration of ascorbic acid but **NOT sodium ascorbate**.

The three dotted graphs recorded three separate measurements of ascorbic acid levels in plasma of the same subject after 10 grams of ascorbic acid was taken in one single dose over a 50-minute period each time.

The solid curve measured plasma levels of the same amount of intravenous sodium ascorbate delivered over a 50-minute period.



Ascorbic Acid is a REDOX molecule that is regenerated, recycled and reused by the body

All three ascorbic acid graphs showed three successive sharp, distinct peaks and valleys with diminishing intensities.

On the other hand, the IV sodium ascorbate curve is smooth, rising mildly at around the 30-minute mark, then descending slowly and gradually.

The initial surges of ascorbic acid during the first 8 minutes after ingestion is the result of rapid, complete absorption by the high capacity SVCT1 transporters. The initial surge easily doubled the amount of intravenous sodium ascorbate injected directly into plasma.

These unique characteristics of peaks and valleys in the ascorbic acid graphs mirrors how ascorbic acid is regenerated, recycled and reused by the body.

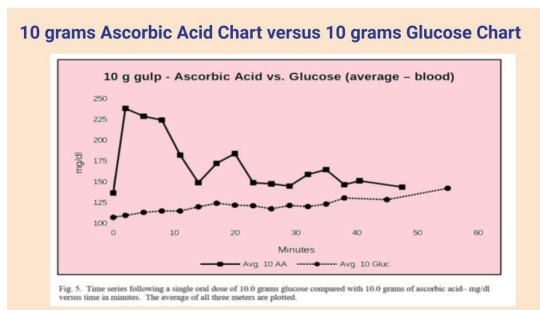
Did Fonorow and Hickey measure Ascorbic Acid or Glucose

The ascorbate molecule is very similar in structure to a glucose molecule, because its synthesis is based on glucose.

Fonorow and Hickey exploited this feature, using glucose meters to measure minute-by-minute results of the two different forms of vitamin C – ascorbic acid and sodium ascorbate.

Minute-by-minute tracking of plasma levels of vitamin C is completely novel. This has NEVER been done before because it is almost impossible to collect, store and measure so many samples effectively.

The following chart clearly shows that Fonorow and Hickey measured ascorbic acid and NOT glucose.

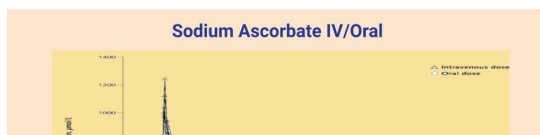


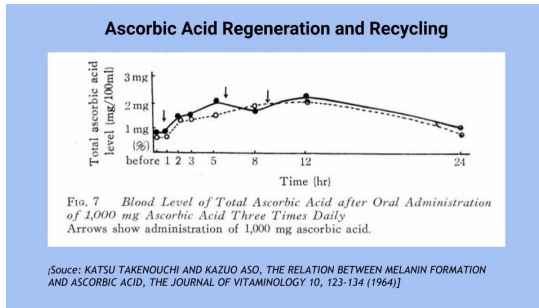
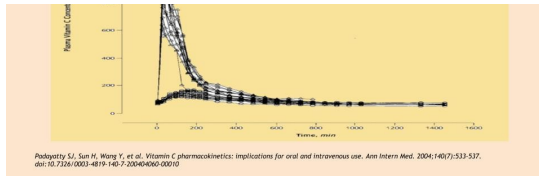
Evidence in literature that supports the findings of Fonorow and Hickey

Most studies that documented results of IV C using sodium ascorbate all showed similar initial surges followed by gradual flattening of the curves.

The chart below for IV sodium ascorbate recorded distinct surges within the first 60 minutes but no ensuing peaks and valleys similar to ascorbic acid.

The lower curve is obtained from oral vitamin C. However, the authors of this study failed to identify whether the source of their oral vitamin C was ascorbic acid or sodium ascorbate (or other forms), perhaps for obvious reasons.





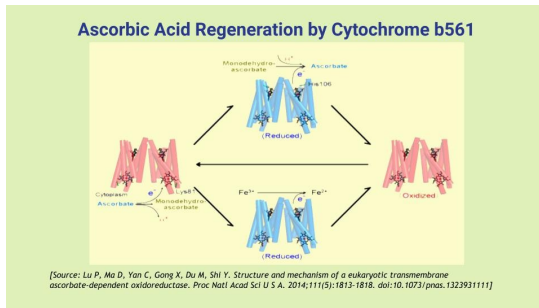
The chart above is taken from a long-forgotten but significant study by Katsu Takenouchi and Kazuo Aso published in 1964.

The chart showed the plasma levels of 1 gram of oral ascorbic acid administered three times during the daytime, measured over a period of 24 hours.

Arrows denote time of oral ingestions, and circles (open/closed) mark the times when measurements were taken from two subjects.

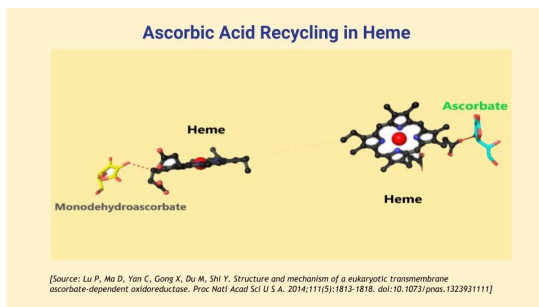
Their measurements also showed a distinct surge to the highest level at 3.5 hours after the final oral 1 gram dose.

What do these peaks and valleys in BOTH charts that tracked ascorbic acid via oral ingestion done almost 56 years apart ?



Red Blood Cells Use Cytochrome b561 to Regenerate and Recycle Oxidized Ascorbic Acid in Plasma

- Red blood cells use ascorbic acid exclusively to reduce oxidized ferric iron into ferrous iron to maintain heme in a stable form.
- They use transmembrane enzymes called Cytochrome b561 (Cytb561) to complete this task.
- Cytb561 captures ascorbic acid after it donates an electron, and uses antioxidants like NADH or even intracellular ascorbate to regenerate oxidized ascorbate.



Peaks and Valleys in Ascorbic Acid Plasma Charts Reflect the Consumption and Regeneration of Ascorbic Acid

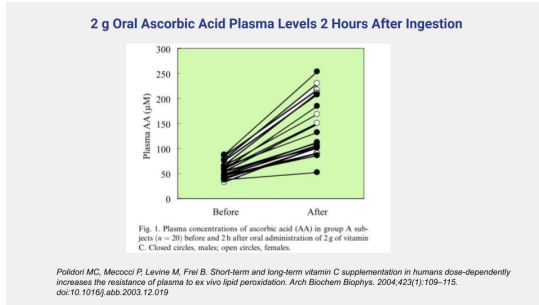
- Initial peak reflects complete absorption of oral ascorbic acid by high



- capacity **SVCT1** in intestines.
- Valleys form when oxidized ascorbate (monodehydroascorbate) is captured by transmembrane enzymes like Cytb561 for regeneration. These molecules are 'docked' inside the enzyme and cannot be detected by glucose meters.
- Subsequent peaks show replenishment of reduced ascorbate that gain an electron from transmembrane enzymes and released back into circulation for another round of REDOX activities.

This is how ascorbic acid is continuously consumed and recycled.

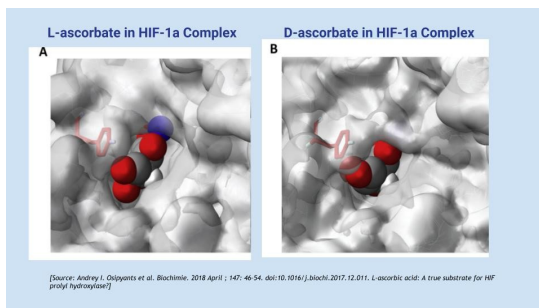
This is the reason why you see peaks and valleys in the ascorbic acid charts and not as much for sodium ascorbate.



10-fold Differences Observed in Plasma Ascorbic Acid Levels 2 Hours after Oral Ingestion

- The levels of ascorbic acid in plasmas of 20 individuals after ingesting 2 grams of ascorbic acid showed distinct differences of up to 10-fold.
- The amount of plasma ascorbic acid shown at 2 hours after ingestion can reflect regenerated ascorbic acid or slower/lower regeneration/recycling capacities of some individuals.

Ascorbic Acid Cannot be Replaced by Similar Molecules Including Sodium Ascorbate



HIF Complex Requires L-ascorbic Acid for Perfect Docking

The images above show the L-isomer of ascorbic acid, L-ascorbate, and its mirror image D-ascorbate nested in the hypoxia-inducible factor HIF1 enzyme complex.

The suppression of HIF1a stabilization is dependent on the availability of ascorbic acid in the natural anionic form of L-ascorbate. Ascorbic acid naturally deprotonates into L-ascorbate under physiological pH.

The HIF1 protein complex does not respond to other antioxidants such as NAC, N-acetyl cysteine.

Even though the mirror D-isomer of ascorbic acid can also reduce ferric iron back to ferrous iron, it is unable to suppress HIF-1a stabilization.

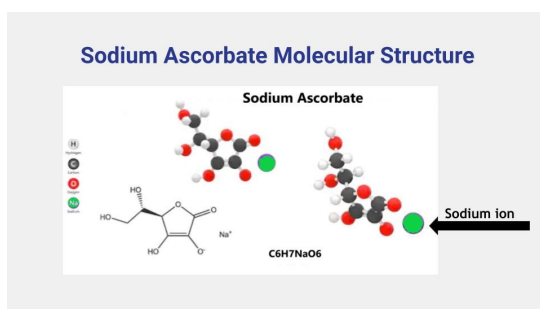
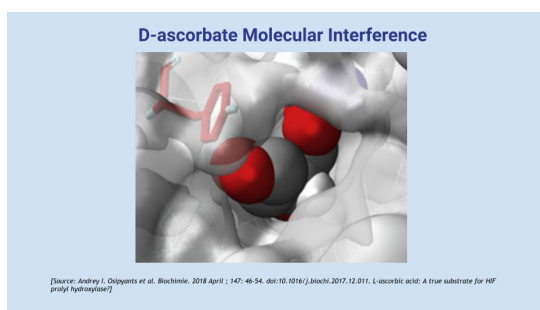
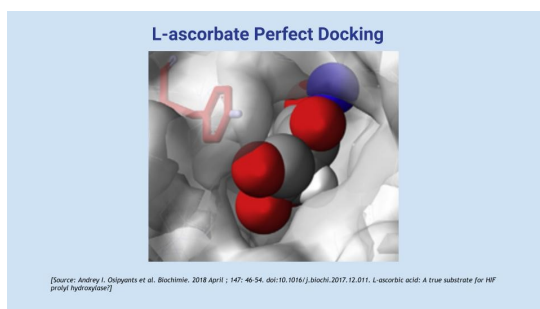




The binding sites in the HIF enzyme active centers allow for the natural docking of L-ascorbate molecules, but not its D-isomer.

It is imperative that everyone understands this significant difference in how a change in molecular structure of ascorbic acid can negatively impact important biological redox processes.

In COVID-19 patients, this difference can mean being almost asymptomatic after infection or rapid deterioration that will require hospitalization and intensive unit care.



Combination Oral Ascorbic Acid and IV C Sodium Ascorbate as Treatment for COVID-19

- Oral + IV C – a powerful two-pronged attack on COVID-19
- Sodium Ascorbate supports haptoglobin regeneration
- High dose Oral Ascorbic Acid prevents and slows ARDS in COVID-19

- IV C is an indispensable tool in the fight against ARDS in COVID-19.
- IV C provides a continuous stream of electrons from sodium ascorbate that can regenerate haptoglobin.
- Haptoglobin is used by the body to bind cell-free hemoglobin.
- Haptoglobin is dependent upon antioxidants to supply electrons to reduce ferric iron in order to maintain heme in a stable form.
- Haptoglobin is not specific to ascorbic acid.

When IV C is used to treat COVID-19 patients, it will free oral ascorbic acid to support critical cellular redox reactions. Ascorbic acid reduces ferric iron to inhibit the formation of cell free hemoglobin. Reduced CFH will lower



to inhibit the formation of cell-free hemoglobin. Reduced CFH will lower destruction of nitric oxide. Nitric oxide homeostasis supports immune functions.

Adequate oral ascorbic acid will protect mitochondria from cytotoxic effects of depolarization caused by viroporins. Ascorbic acid can also elevate lymphocytes, neutrophils and natural killer cells, helping to defeat COVID-19 infection.

All materials presented today are excerpts from the COVID-19 series by Doris Loh at Evolutamente.it

- 1 Mitochondria & The Coronavirus - The Vitamin C Connection (Part 3) - EvolutaMente.it <https://www.evolutamente.it/mitochondria-the-coronavirus-the-vitamin-c-connection-part-3/>
- 2 Furins & Hypoxia - The Vitamin C Connection - EvolutaMente.it <https://www.evolutamente.it/covid-19-furins-cancer-a-tale-of-vitamin-c-hif/>
- 3 COVID-19 Mutations, Vaccines & Nitric Oxide - The Vitamin C Connection - EvolutaMente.it <https://www.evolutamente.it/covid-19-mutations-vaccines-nitric-oxide-the-vitamin-c-connection/>
- 4 COVID-19, Pneumonia & Inflammasomes - The Melatonin Connection - EvolutaMente.it <https://www.evolutamente.it/covid-19-pneumonia-inflammasomes-the-melatonin-connection/>
- 5 COVID-19, ARDS & Cell-Free Hemoglobin - The Ascorbic Acid Connection <https://www.evolutamente.it/covid-19-ards-cell-free-hemoglobin-the-ascorbic-acid-connection/>

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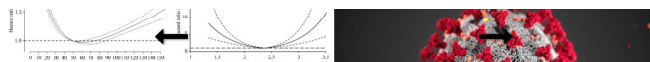
TAGS:

ACE2	acute respiratory distress syndrome	antioxidants			
ARDS	ascorbic acid	BH4	Cell-Free Hemoglobin		
Coronavirus	COVID19	Cytb561	edema	fluids	Furin
HIF1a	Hypoxia	IVC	lungs	Nitric Oxide	oxidants
Pandemic	porphyrins	red blood cells	redox potential		
S protein	Sodium ascorbate	sodium bicarbonate	SVCT1		
SVCT2	Viroporin	vitamin C			



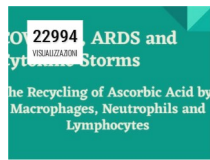
DORIS LOH

Doris Loh is an independent researcher and writer specializing in the investigation of familiar and innovative health topics using unique perspectives in traditional and quantum biology. Her training as a classical pianist allows her the freedom to explore concepts and theories with a curiosity that often results in distinctive conclusions. Recent works by Doris are focused on how health and disease are greatly affected by electromagnetic radiation that surround us everywhere we go. Her works on EMF offer insight and solutions to the challenges humans and other living organisms face during this era of change. Major works by Doris include an in-depth series on deuterium, as well as a startling series on the birefringent quantum properties of the major REDOX balancer, Vitamin C (ascorbic acid). The ongoing series on COVID-19 is recognized around the world for the in-depth coverage of the disease and discussion on holistic alternatives for prevention and treatment.





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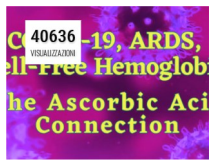
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COVID-19, ARDS @ CYTOKINE STORMS – THE RECYCLING OF ASCORBIC ACID BY MACROPHAGES, NEUTROPHILS AND LYMPHOCYTES



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COVID-19, ARDS @ CELL-FREE HEMOGLOBIN – THE ASCORBIC ACID CONNECTION

ABOUT ANGELO

Angelo 48 anni, ingegnere, papà di una bellissima figlia, ex ciclista e triatleta agonista con alcuni risultati apprezzabili. Dal 2004 ha sperimentato su se stesso la dieta paleo abbinata allo sport di endurance e seguito decine di altre esperienze analoghe su, forum tematici, di altri atleti ed appassionati come lui.

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14 MARZO 2020



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19 GIUGNO 2015



INTERVISTA AD ALESSIO FASANO, MASSIMO STUDIOSO MONDIALE DEGLI EFFETTI DEL GLUTINE SUL CORPO UMANO.

25 MAGGIO 2015



COVID-19, ARDS @ CELL-FREE HEMOGLOBIN – THE ASCORBIC ACID CONNECTION

24 MARZO 2020



ULTIMI POST



COVID-19, ARDS @ CYTOKINE STORMS – THE RECYCLING OF ASCORBIC ACID BY MACROPHAGES, NEUTROPHILS AND LYMPHOCYTES

5 APRILE 2020



IL COVID-19 E' UN VIRUS SEI VAGGIO O CREATO IN LABORATORIO? PARTE 2

4 APRILE 2020



IL COVID-19 E' UN VIRUS SEI VAGGIO O CREATO IN LABORATORIO? (PRIMA PARTE)

3 APRILE 2020



STOP ARDS NOW WITH ASCORBIC ACID

28 MARZO 2020



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