The Only Cause of Disease

New Perspectives on Optimal Treatment

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Summary

- Redox (reduction/oxidation) biology and electron flow
- The regulatory role of vitamin C in support of health
- The relationship between cortisol, vitamin C, and the immune response
- The nature of the “fight or flight” response to new oxidative stress
- The relationship between insulin, vitamin C, and glucose

Reference Checking

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The Only Cause of Disease: A Unified Theory

Increased

Intracellular Biomolecule Oxidation (IBO)

Caused only by toxins, increased IBO exists when the production of free radicals (highly reactive pro-oxidants) exceeds the body's antioxidant capacity to neutralize (reduce) them, or to prevent their production in the first place by directing reducing the toxin

Elevated IBO **always** exists where there is a ***deficiency*** of antioxidants, an ***excess*** of free radicals (toxins), or ***both*** [16760481].

Redox Medicine Principles

Pro-oxidant (synonyms: toxin, oxidizing agent, free radical, reactive oxygen species, poison):

Takes, or causes to be taken, electrons away from reduced (chemically normal) biomolecules (**OXIDATION**)

Redox Medicine Principles

Antioxidant (vitamin C is the prototype)

Gives (or restores) electrons back to oxidized biomolecules (**REDUCTION**), returning them to their normal chemical state

Redox Medicine Principles

The antioxidant nature of vitamin C and the pro-oxidant nature of all toxins concisely explains why vitamin C, along with many other antioxidants, has been documented to be the optimal ***antitoxin*** against ***all*** toxins for which it has been tested, *in vitro* and *in vivo*, in plants, animals, and humans, and in clinical studies.

Properly administered vitamin C has never failed to neutralize an acute pro-oxidant/poison/toxin exposure or ingestion

Redox Medicine Principles

Even though there is a tremendous variety of molecular structure among all the known toxins, they ALL SHARE the property of stealing electrons from biomolecules and ***never returning them***, oxidizing those molecules and resulting in an overall state of increased oxidative stress, especially ***intracellularly***.

Redox Medicine Principles

If a molecule does not cause the loss of one or more electrons from another electron-filled and chemically stable biomolecule, it **CANNOT BE TOXIC**. Toxicity and any symptoms of toxicity cannot exist in the absence of excessive oxidation of biomolecules.

Redox Medicine Principles

All “disease,” then, **IS** the ***state of excess oxidation*** in biomolecules. Expressed differently, oxidative stress does not “cause” disease, but

IS THE DISEASE ITSELF.

No “additional disease” exists beyond this state of excess biomolecule oxidation.

Redox Medicine Principles

Biomolecules (nucleic acid, proteins, enzymes, sugars, fats, etc.) are chemically **inactive** or **significantly less active** when oxidized, and **optimally active** (normal) when reduced. This loss of biomolecule function, and ***nothing else***, results in the clinical expression of diseases and their symptoms.

Redox Medicine Principles

Therefore, the unique nature of any disease process depends ***solely*** on:

Type of biomolecule oxidized (fat, sugar, protein, enzyme, etc.)

Quantity of a type of biomolecule that is oxidized

Location of those oxidized biomolecules

Concentration of those oxidized biomolecules

Length of time in oxidized state (acute or chronic)

Redox Biology Principles

Contrary to what it commonly believed, the primary impact of an antioxidant such as vitamin C to general health and healing is ***not*** to supply NEW electrons into the body, but to optimize the **electron distribution and flow** of the **electrons already present** in the cells and tissues.

It is the diet that serves to sustain and/or increase the available electron “pool” in the body. Earthing and properly applied magnetic fields can also bring new electrons into the body.

Redox Biology Principles

In animal studies, vitamin C (ascorbic acid, AA) administered in its ***oxidized*** form (dehydroascorbic acid, DHAA) **minimized brain infarct size from induced ischemic stroke**. Furthermore, the DHAA promptly ended up INCREASING the quantities of reduced vitamin C metabolized in those brains [26968905, 16883179, 11573006, 19266157].

Redox Biology Principles

Confusion about DHAA (oxidized vitamin C) partially stems from the observation that elevated levels of DHAA are routinely seen in patients with significant infectious diseases, with elevated circulating toxin levels. However, this does not mean that the DHAA itself is toxic, but only that elevated levels of it reflect states of advanced oxidative stress and body-wide electron depletion or entrapment in the associated infection-generated toxic metabolites.

Redox Medicine Principles

Methylene blue, another antioxidant with striking clinical parallels to vitamin C in its effects, rapidly resolves advanced infections, **including advanced septic shock**, when given in ***EITHER its oxidized or reduced forms*** [32435102, 21246318, 8977944, 34399199].

Of note as well is that methylene blue has resolved such cases of advanced septic shock as a **MONOTHERAPY** when all else has failed.

Redox Medicine Principles

In fact, most PubMed studies primarily describe the positive impact of **reduced** vitamin C and of **oxidized** methylene blue, clearly indicating that the **electron distribution capability of a given antioxidant** is its most important characteristic, not its ability to provide new electrons to the body, and not whether it is in an electron-filled or electron-depleted state when administered.

Redox Medicine Principles

To be clear, then, it is ***not necessary*** to administer various antioxidants in their reduced form to realize a positive clinical impact. MB is given in either its oxidized (most ***commonly***) or reduced forms. Same with the antioxidant **Coenzyme Q10** (ubiquinone or ubiquinol). Also with **vitamin K2** (menaquinone or menoquinol). However, "traditional" antioxidants are only marketed in their reduced forms, as the therapeutic value of oxidized antioxidants remains unappreciated, as well as the fact that they are not toxic.

Redox Medicine Principles

The physiological goal of any successful clinical protocol is to normalize as completely as possible the increased **intracellular biomolecule oxidation (IBO),** also known as increased **intracellular oxidative stress (IOS),** in all cells affected by the infection, disease, and/or toxin exposure. This is **always achieved** when intracellular levels of reduced vitamin C can be restored to normal.

Redox Medicine Principles

Vitamin C can only normalize in the cells when the elevated levels of intracellular **calcium** decrease and the lowered levels of intracellular **magnesium** increase. And when these three agents are restored to normal or near-normal levels, normal glutathione levels can then return as well, further supporting a healthy oxidative status in the cell.

Intracellular Biomolecule Oxidation (IBO) Modulators

The fundamental pathology in any disease at the cellular level is **increased intracellular oxidative stress (increased IOS)**. If IOS is not elevated, there is no disease or pathology. The primary ***determinants*** of this intracellular oxidative stress status are the intracellular levels of:

1. Calcium
2. Magnesium
3. Vitamin C
4. Glutathione
5. ATP

Intracellular Biomolecule Oxidation (IBO) Modulators

Calcium:

The intracellular calcium concentration is the primary determinant **and** marker of increased IBO. The higher the level, the greater the IBO. When the level is normal there is no longer excessive IBO and the cell physiology is in a normal state.

The manipulation of intracellular calcium levels appears to be the most straightforward way to positively impact IBO, and *thereby* positively impact ALL disease processes.

Intracellular Biomolecule Oxidation (IBO) Modulators

Calcium:

Increased calcium intake by both supplemental and dietary sources dramatically increases all-cause mortality (death from any disease) [23403980]. Calcium supplementation alone has also been shown to significantly increase the risk of heart attack [22626900, 20671013]

Intracellular Biomolecule Oxidation (IBO) Modulators

Magnesium:

When intracellular calcium levels are high, magnesium levels are low. As more magnesium goes into the cell, more calcium comes out. Magnesium is a **natural calcium channel blocker** and a general **calcium metabolism antagonist**. These calcium-impacting properties of magnesium readily account for its ***enormous*** positive health impact on all diseases or medical conditions.

Intracellular Biomolecule Oxidation (IBO) Modulators

Magnesium:

When intracellular magnesium levels are normal, there is no longer any increased calcium elevating the IBO, and the cell is physiologically normal. And when enough cells in a diseased tissue or organ can regain a normal level of IBO, **there is no longer any disease**.

Just as extra calcium **increases** all-cause mortality, more magnesium **decreases** all-cause mortality [18271493, 21703623]

Intracellular Biomolecule Oxidation (IBO) Modulators

Vitamin C:

When intracellular calcium levels are high and intracellular magnesium levels are low, **vitamin C levels are always low as well**. The delivery of more vitamin C inside cells also promotes increased magnesium levels and decreased calcium levels. There is always an **ongoing dynamic interplay** between calcium, magnesium, and vitamin C in the cytoplasm.

Whenever IBO is severely and chronically elevated, a state of ***intracellular scurvy*** is present.

Intracellular Biomolecule Oxidation (IBO) Modulators

Glutathione (GSH):

While technically the most concentrated and physiologically important of intracellular antioxidants, glutathione levels will **never be normal when calcium is up, magnesium is down, and vitamin C is down**. The maintenance of normal levels of GSH synthesis, along with normal GSH metabolism can only occur as increased cellular oxidative stress normalizes, and ***previously oxidized GSH-synthesizing intracellular enzymes are reduced and restored to optimal function***.

Intracellular Biomolecule Oxidation (IBO) Modulators

As the primary energy-delivering molecule in the body, fueling countless vital metabolic functions and chemical reactions, ATP (adenosine triphosphate) levels are always a direct reflection of the health and vitality of the cell.

Low ATP = low energy. A treatment protocol is not doing its job completely if ATP levels are low, and mitochondrial function (where ATP is formed and is most concentrated) is depressed or suboptimal.

Hormones and Oxidative Stress

Hormones generally work to:

- Positively modulate normal metabolism (optimize and accelerate certain pathways), and
- Minimize/normalize increased intracellular biomolecule oxidation (increased IBO).

Even though different hormones have widely differing biochemical impact, **they all share these two primary purposes**.

Hormones and Oxidative Stress

Insulin, cortisol (hydrocortisone), thyroid hormone, and sex hormone(s) are especially important hormones.

While technically not hormones, both vitamin C and magnesium are essential for the optimal functioning of **every** cell in the body as well.

Always think of combined applications of the above six agents in treating nearly all medical conditions, including infections.

The Final Goal

Remember that the GOAL of an optimally effective clinical protocol is to normalize the increased IBO that is present in ALL cells involved in any disease or medical condition. When this is accomplished, the cell is NO LONGER "diseased," since it is the presence and degree of oxidized biomolecules that determines whether disease exists, NOTHING MORE. When vitamin C (and ATP) levels are normal, the cell is once again normal.

The final goal, therefore, is to achieve and maintain normal intracellular levels of vitamin C.

The Final Goal

In other words, the physiological profile of a healthy cell is one that has a normal intracellular level of reduced vitamin (AA-ascorbic acid).

This also means that the basic disease process in any affected tissue or organ is one of **FOCAL SCURVY** of a variable degree. Focal infections and focal toxin accumulations always underlie any degree of focal scurvy, and they must be targeted in order to achieve an optimal clinical response in any treatment protocol.

Toxin Sources

The most common source of disease-causing toxins come from infections. In fact, the only way an infection makes someone ill or eventually kills them is the nature and degree (amount) to which that infection generates specific pathogen-related toxins or just the pro-oxidant byproducts of its growth and eventual death. Generally, such infections are not body-wide in nature (like the flu), but just focal in a limited area of tissue.

Common Toxin Sources

- Infections (especially **dental**); documented to strongly promote oxidative stress and lessen antioxidant capacity; focal infections anywhere else in the body as well
- **Chronic pathogen colonization** (especially aerodigestive tract [sinus, mouth, and pharynx], and upper respiratory tract)—the **PRIMARY** reason for leaky gut, abnormal microbiome, and MOST chronic digestive disorders.
- Known external toxin exposures [air, food, water] (heavy metals, pesticides, etc.)

Common Toxin Sources

- Toxic **iron** status (most people in "normal" range are toxic); also calcium and copper (The three "toxic nutrients"); pathogens release large amounts of free iron upon their death and breakdown.
- Dietary toxin exposures (food "enrichment", constipated gut, *Clostridium*); inadequate/poor nutrition and/or poor digestion; poor digestion is **worse** than poor nutrition in terms of overall negative impact on the antioxidant capacity of the body
- Critical hormone deficiencies (primarily sex, cortisol, thyroid)

Treatment Approaches (Toxin Management)

- **Prevent/minimize** new daily toxin exposure (environmental, dental, dietary, digestive)
- **Neutralize** toxins already present in the body
- **Excrete** toxin stores in a non-toxic, or minimally toxic, manner (detoxification causes some retoxification)
- **Resolve** infections and pathogen colonizations; OR take the measures needed to **KEEP FOCAL INFECTIONS FOCAL**
- **Supplement optimally** to maximize the antioxidant/nutrient status of the body as completely as possible
- **Address hormone imbalance**, typically deficiencies of testosterone, estrogen, thyroid hormone, and cortisol

What Has Vitamin C Already Been Proven to Do Against Pathogens?

Kill/inactivate all viruses ***in vitro*** against which it has been tested.

Clinically resolve all ***acute*** viral syndromes (***in vivo***) for which it has been adequately dosed. Prominent examples:

Polio: Vitamin C cured acute polio (60 of 60 cases) [Klenner (July, 1949) The treatment of poliomyelitis and other virus diseases with vitamin C, *Southern Medicine & Surgery* pp. 209-214]

Hepatitis (acute):

Initial Rx was 500 to 700 mg of VC/kg body weight by vein, given every 8 to 12 hours. As well, a minimum of 10,000 mg VC orally every day. Routinely, resolution was seen in 2 to 4 days.

Klenner also resolved acute hepatitis with 5,000 mg of VC every four hours or so orally. Complete resolution was typically achieved in 4 days, utilizing a total of about 120,000 mg given.

What Has Vitamin C Already Been Proven to Do Against Pathogens?

Dramatic example:

Comatose New Zealand farmer with H1N1 “swine flu” directly prior to having life support discontinued (2010) [approx. 16 minute video]. See:

https://www.youtube.com/watch?v=GApXBaZuw14

What Has Vitamin C Already Been Proven to Do Against Pathogens?

Resolve all ***acute*** viral syndromes for which it has been adequately dosed. Prominent examples:

Measles (simple and complicated)

Mumps (simple and complicated) [18147027]

Chickenpox [14908970

What Has Vitamin C Already Been Proven to Do Against Pathogens?

Documented efficacy in non-viral infections.

Diphtheria, tetanus, staphylococcus, streptococcus, pseudomonas (all documented as ***curable*** with vitamin C therapy)

Malaria (very positive responses to very low doses)

Leprosy, typhoid fever, brucellosis, trichinosis

Dysentery (amebic and bacillary)

Trypanosomal infections (Chagas' disease)

What Has Vitamin C Already Been Proven to Do Against Pathogens?

Definite benefits and sometimes cure in the following:

Lyme, AIDS, ***chronic*** hepatitis

"Embedded pathogens;" vitamin C (or any other agent) cannot work optimally without physical access to the pathogen

Common cold; a very high requirement of vitamin C needed for the total quantity of virus usually present

Tuberculosis; slow-growing, slow-reacting; massive amount of literature documenting benefits of C for this

Pertussis; combination infection/toxin

What Has Vitamin C Already Been Proven to Do Against Toxins?

Documented as the **ultimate nonspecific antitoxin and poison antidote**, *in vitro* and *in vivo*:

- Toxic elements (mercury, lead, chromium, arsenic, cadmium, nickel, vanadium, aluminum, fluorine)
- Venoms (snake, spider)
- Alcohol [3304067]
- Barbiturates [5899011]
- Toxic mushrooms [6200941]
- Pesticides, six different types
- Strychnine, tetanus

What Has Vitamin C Already Been Proven to Do Against Toxins?

NO toxin has ever been reported in the literature that cannot be neutralized by vitamin C. This applies to all acute toxin exposures, as well as all acute infectious diseases, which are really just ***variations on a potent, acute toxin exposure***.

Remember that any infection UNTREATED is consuming vital vitamin C stores. Regardless of the rest of the protocol, vitamin C should be given to address this rapid depletion, which always worsen the prognosis.

What Has Vitamin C Already Been Proven to Do Against Toxins?

Of note, while effective against all acute infectious diseases and acute toxin exposures, vitamin C has repeatedly been shown to be ESPECIALLY and rapidly effective against viral infections, an especially important consideration in our current pandemic pathologies.

Properly Dosing Vitamin C

The experience at the Riordan Clinic in Wichita, Kansas clearly establishes the safety (and efficacy) of even the highest dosing regimens of vitamin C on a routine basis. Over the past **32** years, over **150,000** intravenous infusions of vitamin C have been administered at Riordan campuses. Doses have varied from ***7.5 to 250 grams daily***, with 50 grams being the most common dose administered. **NO** significant adverse side effects have occurred, and **NO** kidney stones have resulted. And most of these patients were dealing with significant illnesses, such as cancer.

There has never been established a level of vitamin C intake above which is clearly toxic.

Properly Dosing Vitamin C

There still remains a sizeable number of integrative medicine practitioners who appreciate the therapeutic value of multigram doses of vitamin C IV but continue to be needlessly wary of 50- to 100-gram infusions of vitamin C (or more). This unnecessary caution too often results in a total daily dose of vitamin C of 25 grams or **less** that can prove insufficient to save the patient with severe and widespread oxidative damage secondary to an advanced infection or other medical condition. Nevertheless, even such lower doses can oftentimes suffice, just not as reliably so. As the efficacy and safety of vitamin C has been increasingly witnessed during the COVID pandemic, the medical propaganda assaulting this agent has only increased, and the myth of "vitamin C toxicity" continues to be pushed.

For more information on the vitamin C-related research and results of the Riordan Clinic, see: https://riordanclinic.org/journal-articles/.

Optimizing Intracellular Vitamin C

Cortisol (hydrocortisone) promotes the cellular uptake of extracellular vitamin C after the cortisol binds to its intracellular glucocorticoid receptors, which results in an increased expression of vitamin C transporter proteins that traverse the cell membranes, enabling increased cytoplastic vitamin C levels [11502226, 17541511]

A study on 5 subjects given different forms of vitamin C with and without the administration of intravenous hydrocortisone showed a significantly increased uptake of vitamin C in monocytes in the presence of the hydrocortisone. (Mikirova et al. (2019) *Journal of Orthomolecular Medicine*, Vol 34, No 1.

Optimizing Intracellular Vitamin C

Most people with any chronic degenerative disease are **at least** minimally deficient in cortisol production from their adrenal glands. Furthermore, those who contract infections that do not readily resolve for extended periods of time are actually severely deficient in adrenal cortisol production, along with thelack of vitamin C needed to be ushered into the cells by the cortisol. ***In fact, normal cortisol production almost completely prevents the ability of any infection, especially viral, to take hold in the first place***.

Vitamin C and Inflammation

Inflammation in a tissue means that there is increased oxidative stress in the cells of that tissue. This ONLY occurs when a toxin, with or without an associated infection, oxidizes most to all of the **vitamin C** in that tissue.

Similarly, any tissue that is severely depleted of vitamin C is **inflamed**.

Vitamin C depleted tissue = Inflamed tissue

Inflamed tissue = Vitamin C depleted tissue

What is an Anti-Inflammatory Agent?

The relationship between inflammation and vitamin C deficiency/absence means one thing:

Vitamin C is the only true anti-inflammatory agent. Whatever promotes the presence of VC inside inflamed tissue has anti-inflammatory properties, but is NOT a direct anti-inflammatory agent itself.

Similarly, systemic inflammation indicates systemic (or body-wide), vitamin C deficiency.

What is an Anti-Inflammatory Agent?

Cortisol (or any of the synthetic corticosteroids) is NOT a direct anti-inflammatory agent. However, the clinical relief of inflammation as the corticosteroid agent increases vitamin C content inside the inflamed cell has resulted in it being CONSIDERED a powerful anti-inflammatory agent.

However, when there is little to no extracellular vitamin C waiting to be ushered into the cell by cortisol, there is no longer a significant anti-inflammatory impact on the inflamed tissue.

Vitamin C and the Acute Immune Response

- Acute inflammation triggers an acute immune response
- Acute inflammation is present ***because of*** acute antioxidant (vitamin C) depletion at the affected tissue site [“focal scurvy”]
- The **FIRST** immune cell to arrive at the area of inflammation (often infected) is the monocyte [29045897]
- The monocyte has ***at least*** an **80-fold (8000%)** greater concentration of vitamin C than in surrounding blood/plasma [7082619]

Vitamin C and the Acute Immune Response

The acute inflammation causes increased oxidative stress in the blood. This increased oxidative stress results reflexively in:

- Acute cortisol release from the adrenal glands
- This increased cortisol presence works to directly increase **immune cell** vitamin C uptake as well as in the **cells affected by the inflammation**.

Vitamin C and the Acute Immune Response

- In most animals new vitamin C is formed from glucose in the liver, and this entire immune response becomes much more powerful, as the oxidative stress from the acute infection/inflammation releases ***vitamin C into the blood at the same time as the cortisol release***.
- Looking at the pathology of all disease (increased oxidative stress), it appears that the primary role of the immune response is to ***restore depleted vitamin C levels*** in immune cells and in inflamed tissue cells.

Vitamin C and the Acute Stress Response ("Fight or Flight")

Nearly all humans have lost the ability to make new vitamin C from glucose in their livers as is seen in most of the wild animals (an epigenetic defect).

The oxidative stress from acute infection and toxin exposure results in the prompt release of cortisol into the blood. This SHOULD be matched by a large release of vitamin C from the liver while USING UP the increased glucose that is needed for its synthesis in the liver, as occurs in most wild animals.

Vitamin C and the Acute Stress Response ("Fight or Flight")

Instead, the anti-inflammatory impact of the cortisol response is ***only as effective*** as the amount of vitamin C being ingested by diet or supplementation, along with how much vitamin C is already present in the extracellular spaces awaiting cellular uptake assistance from the cortisol. Even in humans, an initial very positive anti-inflammatory impact is seen in the short-term because the adrenal glands release their VC stores (where VC concentration is high) in response to the oxidative stress. This adrenal release of VC actually occurs slightly BEFORE the adrenal cortisol release [17616774]

What does the epigenetic defect cause?

Without more vitamin C, however, the stress response quickly burns out. Treatments with potent pharmaceutical steroid agents such as prednisone or dexamethasone quickly reach this burn-out point, and the side effects of very high NON-PHYSIOLOGICAL corticosteroid dosing then emerge, especially as it results in more protein getting broken down to make even more glucose (gluconeogenesis).

“Fight-or-Flight” Response Results from VC/Cortisol Synergism

In a typical animal liver, vitamin C is ***continuously produced*** from the liver's glucose reserves. When there is a significant presence of pro-oxidants, whether due to infection or toxicity in the bloodstream, an ***increased*** amount of vitamin C is synthesized, sometimes **massively**, as in the goat and some other animals. This process is accompanied by a corresponding release of cortisol from the adrenal glands.

“Fight-or-Flight” Response Results from VC/Cortisol Synergism

This physiological response to oxidative stress is designed to enhance the delivery of vitamin C to the immune cells and for the repair of the inflamed cells and to ***additionally provide fresh circulating vitamin C to counteract newly introduced toxins before further oxidative damage occurs***. (It is important to remember that vitamin C has been clinically established as the **ultimate antitoxin**.)

“Fight-or-Flight” Response Results from VC/Cortisol Synergism

In most human livers, VC cannot be synthesized because of an ACQUIRED epigenetic defect that appears to block the needed ribosome-mediated translation of the mRNA coding for GULO enzyme that completes the conversion of glucose to vitamin C.

Of note, infants commonly make their own vitamin C and then gradually lose the ability [4830116, 6496385, 13315928]. This rules out a fixed genetic defect in the sequencing of the coding DNA.

“Fight-or-Flight” Response Results from VC/Cortisol Synergism

At least one agent (hydroxytyrosol) has been found to double vitamin C levels in human volunteers [28063380], and it appears that this nutrient polyphenol allows a “readthrough” phenomenon that lets the ribosome skip over or past an abnormally placed stop codon in the translation process, permitting the formation of at least a partially functional form of the GULO enzyme. Hydroxytyrosol is especially concentrated in olives and olive-derived products (olive leaf extract).

"Fight-or-Flight" Response Results from VC/Cortisol Synergism

In a study involving two volunteers who consumed hydroxytyrosol, the intake of substantial amounts of alcohol (a toxin) led to significant and immediate ***increases*** in vitamin C levels, which remained elevated for 24 hours until the alcohol was fully metabolized. Without a reflexive synthesis of VC in the liver (along with cortisol from the adrenal glands), a high dose of toxin would cause marked ***decreases*** in VC blood levels as it is consumed in neutralizing the toxin presence

See also: http://orthomolecular.org/resources/omns/v18n14.shtml

“Fight-or-Flight”
What SHOULD Happen?

Large oxidative stress in the blood, causing an

- Adrenal release of cortisol and a liver release of newly-synthesized vitamin C,
- Resulting in increased intracellular vitamin C in the immune cells and throughout the body, but especially in infected cells already targeted by an immune cell response to inflammation
- Along with increased cortisol-induced glucose synthesis, supplying more substrate to make new vitamin C,
- Continuing until the oxidative insult is resolved

"Fight-or-Flight" What DOES Happen?

In nearly all humans:

- Oxidative stress leads to the release of cortisol, but there is **NO** reflexive liver synthesis of new VC, and only the very limited reserves of VC in the adrenal gland are released into the blood.
- The ongoing production of glucose induced by cortisol (gluconeogenesis) is not utilized in the creation of new VC, and glucose excess syndromes can emerge
- The new oxidative stress can only be resolved quickly when it is limited and when the necessary amount of VC available for cellular uptake is already present in the extracellular spaces in the body.

“Fight-or-Flight” What DOES Happen?

The continued stimulation of glucose production by the corticosteroid along with the inability of the human liver to use up that glucose to make vitamin C results in elevated blood glucose levels, diabetes, and elevated markers of long-term damage from the presence of excess glucose (A1C levels).

There is also muscle wasting as the stimulation of new glucose production by the corticosteroid consumes muscle protein in the process.

Proper use of cortisol (not any other steroid) has NO side effects and only enhances good health. A hormone deficiency is being properly treated.

Cortisol Safety

Remember that restoring deficient natural hormone levels is not "prescriptive medicine," but **CLASSIC ORTHOMOLECULAR MEDICINE (Restoring a deficient biomolecule or natural agent**). Furthermore, you never get hormone excess side effects when you are working to restore a hormone deficiency. Instead, you just get benefits. But you will sustain/worsen illness with an uncorrected hormone deficiency. Hormone deficiencies must always be properly addressed. Great harm comes from either hormone **deficiency or excess**.

Optimizing Intracellular Vitamin C

The clinical response of combined vitamin C-cortisol applications (IV great, oral still excellent) is generally ***stunning*** relative to other treatment modalities. The FIRST LINE treatment of any new infection should be large doses of VC matched with sizeable doses of cortisol (for example, 5 grams of VC with 20+ mg hydrocortisone several times throughout the day). But ***cortisol only*** and not other synthetic steroids.

Optimizing Intracellular Vitamin C

It is important to realize that this dramatic "fight or flight" stress response of VC and cortisol to a new infection/toxin in the body is optimized at the ***early*** stage. In advanced sepsis, there is already a **compensatory high level of circulating cortisol** "trying" to unsuccessfully bind the **oxidized** intracellular glucocorticoid receptors resulting from the advanced oxidative stress present in this state [23933016]. But until the oxidative stress can be sufficiently relieved, there won't be enough normal, reduced receptors for the cortisol to bind.

Optimizing Intracellular Vitamin C

At this point, the patient cannot benefit from further cortisol support. Only massive doses of vitamin C, on the order of 100 to 150 grams intravenously daily in divided doses, will overcome the excess oxidation and likelihood of imminent death. A continual infusion providing this same amount of vitamin C over each 24-hour period would prove excellent as well. The reported success seen with VC-cortisol-thiamine in advanced sepsis patients was also seen with a comparable dosing regimen of ***VC alone***, further supporting this premise [27940189, 27162802]

VC/Cortisol Synergism
Clinical Takeaways

Whenever cortisol is clinically indicated, its impact will be greatly enhanced by the simultaneous administration of vitamin C.

Whenever vitamin C is clinically indicated, its impact will be greatly enhanced by the simultaneous administration of cortisol.

VC/Cortisol Synergism
Clinical Takeaways

Very large doses of vitamin C alone can eliminate the need for simultaneous cortisol to optimize intracellular uptake in the short-term.

Cortisol intake alone only has a powerful impact when the provoking oxidative stress is self-limited, and enough extracellular vitamin C is already available to be drawn into the affected cells.

Nevertheless, adrenal fatigue must always be addressed with cortisol replacement, even in the absence of VC administration.

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

Insulin facilitates the intracellular uptake of glucose and vitamin C in a **competitive** manner [1988772, 9550452] Both glucose and oxidized VC enter the cells via the GLUT transporter membrane proteins. VC can also utilize other transporter proteins as well (SVCTs) [33352824, 20494648] (which are induced by cortisol)

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

Insulin has also been shown to stimulate VC recycling along with promoting its intracellular accumulation [9421397]

Large amounts of either glucose OR VC will trigger comparable amounts of insulin release from the pancreas

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

In the human liver, glucose that does not get converted into VC ends up in the bloodstream instead of being turned into VC, partly because cortisol enhances glucose production in preparation for a conversion to VC that **never occurs**. It is important to note that prolonged use of synthetic steroids can lead to glucose intolerance and frequently results in overt diabetes, as the glucose produced does not convert into VC.

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

IF, however, the human liver was continuously producing VC from glucose around the clock, the blood concentrations of VC would be ***significantly higher***, while glucose levels would be ***considerably lower***.

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

A VC-synthesizing liver means that **much less glucose** would be entering the cells and driving ATP production via the Krebs cycle.

Furthermore, all cellular health is driven by electron availability and electron flow, ***which is precisely what vitamin C does in the intracellular space when its cytoplasmic levels are optimized***. ATP serves the same functions, although the metabolic pathways required to donate needed electrons are not identical. And in the relative absence of glucose, vitamin C will be even more effectively ushered into the cells with the help of insulin (and cortisol).

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

This begs the question:

How important would the Krebs cycle be in generating needed cellular energy if very little glucose was available and vitamin C was abundant?

One possibility:

The Krebs cycle has *evolved* as a significant source of cellular energy (electrons) to partially (or drastically?)compensate for the loss of vitamin C production by the liver.

Glucose, Vitamin C, Insulin, and the Krebs Cycle: A Hypothesis

This hypothesis means that diabetes and elevated blood glucose levels would be as rare in humans as in wild animals (where it is virtually nonexistent).

This hypothesis also means that the **most important** role of insulin in the body is to facilitate **transport of vitamin C** into the cells of the body **rather than glucose**, and that the powerful healing properties of properly applied insulin is primarily due to restoring normal levels of intracellular vitamin C.

General Suggestions for VC/Cortisol Administration

Cortisol administration can be given one to four times daily; individual clinical response will determine how much adrenal support is needed over time, dictating an optimal dosing regimen. But even after a satisfactory clinical response is seen for an extended period of time, adjustments up or down can still be made in the dose size and frequency of administration of the cortisol if a change in clinical status warrants. Periodic routine laboratory examinations will directly indicate as well whether the metabolism is stable, deteriorating, or improving. Salivary cortisol levels are best for monitoring this.

General Suggestions for VC/Cortisol Administration

A minimum of one gram of vitamin C should be taken with each dose of hydrocortisone (a higher dose of vitamin C is perfectly fine and even better if it does not cause loose bowels). Sodium ascorbate, ascorbic acid, and liposome-encapsulated vitamin C can be given as well.

General Suggestions for VC/Cortisol Administration

Cortisol can generally be given up to 20 mg daily total in patients with clear clinical evidence of adrenal fatigue on a **chronic** basis. Slightly more can be given with careful physician monitoring. However, 35 to 40 mg is a common full replacement dose given to individuals who no longer synthesize cortisol at all (Addison's disease), and it would be too high a daily dose for patients still making some cortisol, although long-term steroid side effects ***would still not be anticipated***.

General Suggestions for VC/Cortisol Administration

The lower the daily cortisol dose that can be given with what is considered an optimal clinical response by the physician is the goal.

Doses can range from 2.5 mg one to four times daily up to 5 mg one to four times daily. Depending on clinical response, the dose size can vary at different times of the day, as well as the dosing frequency. Some patients will do well on a single daily (usually morning) dose of 10 to 20 mg cortisol. Evening dosing of cortisol frequently results in poor sleep.

General Suggestions for VC/Cortisol Administration

Short-term cortisol dosing when dealing with an acute infection or toxin exposure can be ***substantially higher***. Up to 100 mg of cortisol daily in a single dose or in divided doses **WITH** a significant vitamin C dose can be given very safely under such circumstances, but not continued at that level beyond a few days. When started early in the course of a new respiratory virus (including COVID), the infection can typically be quickly resolved. However, with a clearly positive clinical response, the regimen can be extended to a week or so.

Short-term cortisol/VC administration can be expected to help anyone with an acute onset infection or medical condition, as more vitamin C is needed inside the cells regardless of what is consuming it.

The Super 8 Supplements

The four supplements that reduce intracellular calcium levels and **individually reduce all-cause mortality**:

- Vitamin C (multigram doses)
- Magnesium (multigram doses) [vigorous supplementation **essential** if baseline blood level is low, and/or seizure disorders or cardiac arrhythmias are present]
- Vitamin D3 (to reach a level > 50 ng/mL)
- Vitamin K2 (500 micrograms to several mg)

Except for vitamin D3 all of these can be increased as desired for better clinical impact, as toxicity is almost nonexistent.

The Super 8 Supplements

The four ATP-generating supplements that power the electron transport chain:

- Niacinamide (500 mg, multigram with brain and nervous system problems)
- Riboflavin, 400 mg daily
- Coenzyme Q10 (600 to 1,200 mg)
- Methylene blue 20 to 30 mg daily

Super 8 Supplements

Optimizing calcium status inside all the cells (vitamin C, magnesium, vitamin D3, vitamin K2)

Optimizing ATP production in all the cells (niacinamide, riboflavin, coenzyme Q10, and methylene blue)

These 8 supplements ultimately work to optimize vitamin C and ATP levels and eliminate excess oxidative stress inside the cells. **They will augment ANY clinical regimen**, and they will serve many individuals well as stand-alone supplementation. **They will also promote heavy metal mobilization and elimination**.

For Contact and Further Information

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