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Vitamin C and Cortisol
Synergistic Infection and Toxin Defense

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OMNS (Dec. 11, 2021) Vitamin C and cortisol are the two most important and most powerful naturally-occurring anti-inflammatory agents. The mechanisms of their synergistic action indicate they are literally ***designed by nature*** to interact together to optimize the antioxidant impact needed to resolve the disease-causing oxidation that always results from toxins, infections, and stress. As inflammation in a tissue is the direct result of the oxidation, metabolism, and depletion of vitamin C levels in that tissue, it is of primary concern to normalize cellular vitamin C levels as promptly and completely as possible. Quite literally, when intracellular vitamin C levels are normalized in an inflamed tissue, the inflammation is completely resolved, and the cells are once again in a non-diseased, normal state.

A focal vitamin C depletion in a tissue is the primary identifying feature of focal inflammation. Greater degrees of focal inflammation only occur with greater degrees of focal oxidative stress. As can logically be expected, focal oxidative stress rises as focal vitamin C stores are consumed and are not restored. This lack of vitamin C at sites of inflammation explains nicely why the acute immune system response to a focally inflamed tissue is initially dominated by the appearance of monocytes (Tabas et al., 2017). The monocytes have an exceptionally high concentration of vitamin C in them, the highest of all the immune cells. Relative to the plasma concentration of vitamin C, these monocytes concentrate vitamin C in their cytoplasm 80-fold (8,000%) higher than the plasma. Other immune cells also have very high intracellular levels of vitamin C (Evans et al., 1982). It appears likely that the initial role of monocytes arriving at a site of inflammation is to effectively deliver antioxidant impact in the form of vitamin C, working to promptly alleviate whatever degree of oxidative stress is present.

In many hospitalized patients with significant infections, extremely low plasma levels of vitamin C are present. When the depressed levels of vitamin C are present throughout the body and not focal, the associated increased oxidative stress is generalized and typically reflected in elevated blood levels of C-reactive protein (CRP). CRP is a reliable index of systemically increased inflammation that is always present when vitamin C levels are significantly low (Carr et al., 2017). Circulating cortisol levels are also the lowest in the most severely ill patients.

As it turns out, cortisol significantly augments the uptake of vitamin C into cells (Fujita et al., 2001; Mikirova et al., 2019). More specifically, it appears to stimulate the production of the messenger RNA needed to increase the expression of the sodium-ascorbate co-transporters (SVCTs). This works to enhance cellular vitamin C uptake needed to maximize the protection of metabolically active cells against oxidative stress (Savini et al., 2008). ***This is very likely the primary function*** of cortisol in the body, as there is nothing more important for the resolution of tissue inflammation and the resulting tissue damage than normalizing elevated levels of intracellular oxidative stress as rapidly and completely as possible by normalizing intracellular levels of vitamin C. And when

intracellular levels of vitamin C are normal, cellular glutathione levels needed to protect the cell are also optimized.

General Disease Physiology

The physiology of all disease at the cellular and biomolecular level relates directly to the extent to which any of a variety of biomolecules are in the oxidized (electron-depleted) state. All pro-oxidants (toxins) ultimately inflict their damage by directly oxidizing biomolecules, or by indirectly resulting in the oxidation of those biomolecules (proteins, sugars, fats, enzymes, etc.). When biomolecules become oxidized (lose electrons) they no longer perform their normal chemical or metabolic functions. An oxidized enzyme, for example, can be completely inactive.

No toxin can cause any clinical toxicity unless biomolecules end up becoming oxidized. The unique array of biomolecules that become oxidized determines the nature of the clinical condition resulting from a given toxin exposure. There is no "disease" present in cells of the tissue involved in a given medical condition beyond the distribution of, identity of, and degree of oxidation in the biomolecules of an affected tissue. Rather than "causing" disease, the state of oxidation in an array of biomolecules **IS** the disease.

When antioxidants can donate electrons and restore a normal electron status back to previously oxidized biomolecules (reduction), the normal functions of these biomolecules are restored. This is the reason why sufficient antioxidant therapy, such as can be achieved by highly-dosed intravenous vitamin C, has proven to be so profoundly effective in blocking and even reversing the negative clinical impact of any toxin or poison. There exists no toxin against which vitamin C has been tested that has not been effectively neutralized (Levy, 2002).

Because of this, there is no better way to save a patient clinically poisoned by any agent than by immediately administering a sizeable intravenous infusion of sodium ascorbate. The addition of magnesium chloride to the infusion is also important to protect against sudden life-threatening arrhythmias that can occur before a sufficient number of the newly-oxidized biomolecules can be reduced and any remaining toxin is neutralized and excreted (Levy, 2019). The relationship between cortisol and vitamin C also mandates the addition of cortisol to such vitamin C infusions to optimize the rapidity and degree to which poisoned cells can normalize intracellular vitamin C. This directly and promptly reverses the abnormal increases of intracellular oxidation seen with any excessive toxin (poison or pro-oxidant) exposure.

As a practical point, then, the primary clinical point to take from the synergism of vitamin C and cortisol is the following:

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

Cortisol Physiology

Cortisol, referred to as hydrocortisone when given as a medication, is a hormone known as a glucocorticoid. This type of hormone is produced in the outer portion (cortex) of the adrenal glands that sit on top of the kidneys. In addition to having a pronounced anti-inflammatory effect, a glucocorticoid increases glucose levels in the blood through a process known as gluconeogenesis in the liver. This process utilizes amino acids and other non-carbohydrate molecules to produce more glucose. When cortisol or other corticosteroids are too highly-dosed and given for too long a period of time, a state of widespread protein breakdown (catabolism with muscle wasting) can result as the proteins are converted to glucose. Furthermore, this continued stimulation of glucose production in the liver can result in higher circulating glucose levels and sometimes even frank diabetes. These effects account for some of the most significant side effects of chronic and highly-dosed steroid (e.g., prednisone, dexamethasone) therapy. "Traditionally-dosed" long-term steroid therapy would never cause problems if much lower doses were employed (20 mg of hydrocortisone or less daily), especially when given in conjunction with multigram doses of vitamin C. The chronic ingestion of high doses of steroids without the

simultaneous intake (or internal production) of vitamin C is analogous to trying to shoot a high-powered gun without ammunition.

Of note, taking large enough amounts of vitamin C supplementation alone can eliminate the need for more cortisol to optimize intracellular uptake of the vitamin C. However, it is often not a practical option to administer the 50-, 75-, or 100-gram infusions needed to reach such optimal cellular vitamin C levels without the assistance of cortisol. Nevertheless, cortisol still greatly facilitates this process, and having enough cortisol in the bloodstream decreases the "wasting" of vitamin C by its elimination in the kidneys that would otherwise end up inside the cells.

> *Supplying the right amount of cortisol when it is chronically deficient and no longer being synthesized in normal amounts in the body is still absolutely essential to achieving optimal health, similar to the need for thyroid hormone administration when its levels are chronically low.*

Even though the body might have "normal" levels of cortisol upon blood testing at different times in the day, this does not rule out that under conditions of severe stress and new infection/toxin exposure the adrenals might no longer have the capacity to produce sufficient additional amounts of cortisol to deal with that stress. In fact, ***succumbing to an infection is a direct indication*** that more cortisol (and vitamin C) was needed by the body. It has been observed that a fatigued individual with known adrenal insufficiency can readily progress to an influenza-like state of malaise and generalized aching when the cortisol level is especially low. Patients with clear-cut influenza have markedly low cortisol levels, and the lowest cortisol levels occur in the sickest patients with the highest fevers and the lowest white counts. Any acute severe infection results in the same classical symptoms associated with just very low cortisol levels, as seen in patients with acute adrenal insufficiency (Jefferies, 2004).

The potent anti-inflammatory effect of cortisol fits perfectly with its label as the anti-stress, "fight-or-flight" hormone. Physiologically, stress is effectively a surge of pro-oxidants (toxins) into the blood, whether from infection or another source. This results in a need for the body to immediately counteract or compensate with a surge of antioxidants. In a ***completely normal mammalian liver***, vitamin C is synthesized from glucose modified by a sequence of four enzymes. However, most humans are missing the fourth enzyme due to an epigenetic defect.

Part of the "fight-or-flight" reaction to stress in the body is also supported by the release of adrenaline (epinephrine) from the inner part (medulla) of the adrenal glands. Adrenaline works to mobilize glucose from its storage form (glycogen) in the liver and muscles, and it also stimulates gluconeogenesis to further increase glucose levels (Cryer, 1993). This would appear to be important in making sure that enough glucose is available to the fully-functioning liver to make whatever amount of vitamin C is needed to deal with a severe enough acute infection or toxin insult. Of note, vitamin C supplementation has been shown to decrease circulating cortisol and adrenaline levels in athletes following stressful exercise. This is consistent with the role played by these two substances to increase vitamin C levels following any form of stress. When enough vitamin C is already present, cortisol and adrenaline are no longer as necessary in supporting the response of the body to stress, and their levels are appropriately lower (Peters et al., 2001).

Nevertheless, the natural design of this anti-stress, antitoxin effect in the body is incredibly elegant when vitamin C synthesis can occur in a completely normal liver, as is the case with many mammals. It can be summarized as follows:

- The presence of pro-oxidant pathogens or other toxins ("stress") in the blood **results in**
- A compensatory increased liver production of vitamin C released directly into the blood to neutralize the toxin surge, along with an accompanying reflex release of cortisol and adrenaline from the adrenal glands, **which results in**
- An increased uptake of the newly-synthesized vitamin C into the toxin-exposed cells by the increased presence of the cortisol in the blood, **which is sustained by**
- A cortisol-induced increased glucose production (gluconeogenesis) in the liver and an adrenaline-induced release of glucose from its storage forms (glycogen) **which results in**
- An ongoing conversion of that increased glucose production into more vitamin C production with an ongoing release of cortisol to bring the vitamin C inside the toxin-challenged cells, **continuing until**
- The infection is resolved and/or the toxins are fully neutralized with electrons, metabolized, and excreted.

However, in the typical human who is missing the fourth enzyme in the liver needed to synthesize more vitamin C from glucose, the cortisol has only the pre-existing vitamin C circulating in the blood available for cellular uptake. At the same time, the cortisol-induced and adrenaline-induced production of more glucose will chronically contribute to its excess presence throughout the body since it cannot be used to fuel the production of more vitamin C in the liver. Of note, a recently-discovered olive-derived polyphenol appropriately-dosed appears to help overcome this epigenetic defect, or at least to boost systemic levels of vitamin C in the body [www.formula216.com]. Regular supplementation with this product appears to be very effective in optimizing vitamin C impact in the body.

When the acute oxidative stress is due to the onset of infection and not solely due to the presence of a new toxin in the blood, the cortisol also plays an important role in killing the pathogen. By facilitating vitamin C entry into the infected cell, cortisol serves to help upregulate the Fenton reaction. This reaction utilizes the electrons supplied by the cellular vitamin C to break down the cytoplasmic hydrogen peroxide into the highly lethal hydroxyl radical, which immediately oxidizes every biomolecule it encounters, ultimately resulting in pathogen death, programmed cell death (apoptosis), and/or frank cellular rupture (Levy, 2021).

Supporting Research

The nature of this important interplay between vitamin C and cortisol is further supported and clarified by a substantial amount of clinical, animal, and basic (*in vitro*) research data.

- In human lung microvascular endothelial cells, vitamin C and hydrocortisone work to synergistically and dramatically reverse lipopolysaccharide-induced (oxidative) barrier dysfunction (Barabutis et al., 2017).
- In a rat model of kidney reperfusion injury, vitamins C and E in combination with hydrocortisone appear to offer synergistic protection compared to that offered by the individual agents (Azari et al., 2015).
- Hydrocortisone function, or expression, depends on the redox status of its intracellular receptor. When a substantial percentage of the receptors are oxidized, the degree of hydrocortisone binding to its receptors is proportionately lessened, and hydrocortisone can no longer optimize vitamin C uptake into the cell (Okamoto et al., 1999).
- A vitamin C derivative restored electrons to oxidized hydrocortisone receptors, which allowed them to function (Okamoto et al., 1998). Such inactivated receptors are increased in number in the highly oxidized environment of infection. This means that sufficient vitamin C is needed to keep receptors activated and able to bind whatever hydrocortisone is present inside the cell, which then can further facilitate the uptake of more vitamin C. *A classical synergism: more cellular vitamin C leads to more cortisol receptor binding, and more cortisol receptor binding leads to more cellular vitamin C uptake.*
- In patients with asthma, vitamin C supplementation has been shown to permit a reduction in the corticosteroid dose required to maintain control of that condition, further supporting the similar physiological impacts of vitamin C and hydrocortisone (Fogarty et al., 2006).

The degree of infection (as with mild influenza versus advanced sepsis) largely determines whether the administration of hydrocortisone will be of significant additional benefit in the treatment protocol. Advanced sepsis is a condition in which systemic oxidative stress is about as maximal as it can be before proceeding to death. As such, a very substantial percentage of the intracellular hydrocortisone receptors are in a nonfunctional, oxidized state. Because of this, the body attempts to compensate by increasing the production of cortisol in the body, although this offers little to no benefit as long as the receptors remain oxidized and unable to bind any cortisol, and no vitamin C is being administered to activate the receptors.

In less advanced infections, and even in earliest stages of sepsis, the number of receptors is often increased and administering hydrocortisone can have clear-cut benefits, especially when vitamin C is given as well (Vardas et al., 2017). In fact, increased receptor function is essential to prevent a person or laboratory animal with early sepsis from proceeding to advanced sepsis and death. As the infection advances, receptor function is depressed due to the increased oxidation of a worsening infection, and the body then transitions to the increased production of cortisol in an attempt to compensate (Antonucci et al., 2014; Shibata et al., 2015). This almost never stops the clinical decline, and death ensues unless enough vitamin C is given to activate the oxidized receptors and significantly decrease overall oxidative stress by increasing intracellular levels of vitamin C. Similar findings have been seen in an animal model of sepsis (Bergquist et al., 2013).

The treatment of patients in septic shock with vitamin C, hydrocortisone, and thiamine was reported to be stunningly effective, with the mortality rate dropping from 40% to 9%, and with none of the deaths resulting directly from sepsis or septic complications (Marik et al., 2017). However, a similarly constructed study showed that basically the ***same result*** could be achieved with ***only the administration of vitamin C*** (Zabet et al., 2016). This fits with the observation that circulating endogenous levels of cortisol are already elevated in advanced sepsis, and it is vitamin C administration, not additional hydrocortisone, that is of most consequence at that point in the treatment of a septic patient. Of note, as primary or secondary therapy, vitamin C has attenuated sepsis-induced adult respiratory distress syndrome (Bharara et al., 2016), aspiration-induced adult respiratory distress syndrome (Kim et al., 2017), virus-induced adult respiratory distress syndrome (Fowler et al., 2017), and adult respiratory distress syndrome secondary to the complications of pustular psoriasis (Marik and Long, 2018).

The best therapy for any advanced sepsis patient would simply be very large doses of vitamin C intravenously, on the order of 25 grams every six hours (100 grams every 24 hours). Along with the pre-existing high circulating levels of cortisol, this would rapidly reduce elevated intracellular oxidative stress levels to normal or near-normal, and all but those sepsis patients who had already developed too much multi-organ damage would be readily saved.

On the other hand, many critically ill patients who are not fighting sepsis will demonstrate significantly low cortisol levels (Marik et al., 2008), and they would benefit greatly from the administration of both vitamin C and hydrocortisone. Also, whenever there is a question of whether cortisol levels are already high in the body, the addition of further hydrocortisone does no harm and can readily be added to the protocol to "cover all bases."

> *Overall, the current scientific literature indicates that vitamin C and hydrocortisone individually promote increased antioxidant capacity. However, it is also clear that these two agents are very synergistic in promoting this effect, although properly-dosed vitamin C also appears to be very effective as a monotherapy in sepsis and septic shock.*

Vitamin C and Cortisol Safety

Before proceeding to the recommended applications of a combined therapeutic approach with vitamin C and hydrocortisone, the current state of propaganda directed at undermining and limiting the use of these agents should be addressed. Most physicians have been completely misled into believing that vitamin C is toxic to the kidneys and promotes the formation of kidney stones. ***Nothing could be further from the truth.*** As with all the other organs in the body, vitamin C, in multigram daily doses, only promotes good health throughout the body, including the kidneys. A Harvard study on 85,557 women with no history of kidney stones showed that a regular intake of vitamin C was not associated with any risk of developing kidney stones (Curhan et al., 1999). Another Harvard study actually found that individuals with the highest vitamin C intake had a ***lower*** risk of kidney stones than those individuals with the lowest vitamin C intake (Gerster, 1997). This was further corroborated in another study looking at blood levels of vitamin C in over 10,000 subjects. The subjects with the highest blood levels had the lowest incidence of kidney stones (Simon and Hudes, 1999).

Highly-dosed vitamin C given intravenously also causes no problems with kidney function and does not promote the formation of kidney stones. Such infusions achieve temporary blood levels much higher than with oral administration, yet still are completely nontoxic. A prospective study following 157 patients given such infusions showed no kidney problems developing over a period of 12 months. No stones were reported, even though 8% of the patients already had a history of kidney stones (Prier et al., 2018). Vitamin C, along with magnesium, vitamin D, and vitamin K2 all work to prevent stone formation ***as well as to dissolve and mobilize existing stones***. This is because stones are usually calcium oxalate, and the oxalate that can come from vitamin C metabolism will never produce a stone in the absence of an excessive calcium presence (Levy, 2013). In fact, even though it is chemically a weak organic acid, vitamin C (ascorbic acid) puts calcium carbonate into solution as readily as a concentrated inorganic acid, like hydrochloric acid (Ruskin, 1938).

Aside from the myth of vitamin C causing kidney stones, there are many physicians who just seem to think it must be toxic and would not even considering giving it intravenously. In fact, vitamin C might be the only substance for which a toxic level cannot be established. Continuous vitamin C infusions of 50 grams daily were given over an eight-week period in advanced cancer patients with no definable negative side effects (Casciari et al., 2001). A study surveying the administration of infusions routinely exceeding 25 grams in over 20,000 patients cared for by 172

complementary medicine practitioners revealed the infusion to be "remarkably safe" (Padayatty et al., 2010). At the Riordan Clinic in Wichita, KS over a 16-year period "...194,054 g, or 427 lbs of IV vitamin C" was administered to 275 patients with no significant side effects ever observed (Jackson et al., 2002). For even further perspective on this remarkable lack of toxicity by vitamin C, consider the fact that too much water ingested too rapidly can kill (Hayashi et al., 2005).

With regard to cortisol, all doctors and most of the public know that high doses of corticosteroids given for an extended period of time have severe and inevitable negative side effects. This has caused the more routine applications of much lower doses of cortisol to also be approached with unnecessary caution. In fact, very many people have abnormally low circulating levels of cortisol. And even more importantly, the degree of stress-induced cortisol release can be significantly decreased even when the non-stressed circulating levels are within the range considered to be normal. If most people were routinely tested for their circulating cortisol levels and the degree to which they are able to increase cortisol release in response to stress, the routine administration of cortisol in daily doses of 20 mg or less would be commonly employed in acute infections as well as in the long-term treatment of chronic medical conditions (Jefferies, 2004).

Optimizing the Treatment of Spike Protein Persistence

While adding hydrocortisone to the administration of vitamin C can further improve an already excellent therapy, the use of this combined therapy appears to be an optimal way to approach syndromes that are characterized by persistence of the COVID-related spike protein in the body. Individuals experiencing problems following COVID vaccinations, as well as "long-haul" COVID, which is basically a low-grade and ongoing chronic COVID infection, should prove to be optimal candidates for treatment protocols that include combined hydrocortisone and vitamin C administration. As mentioned above, vitamin C alone given in sufficient doses can still effectively "saturate" the targeted cells, but the doses required simply make many physicians with only limited vitamin C experience too uncomfortable to give such doses, whereas the same result can be achieved with lesser doses of vitamin C combined with hydrocortisone.

The thorough and complete treatment of persistent spike protein is especially important for not only reducing long-term mortality but also for reducing a great deal of morbidity, or clinical illness, in the shorter term. While it now appears that an ongoing spike protein persistence can result in a very wide array of clinical syndromes, depending what organs or tissues most bind the spike protein in different individuals, many appear to maintain inflammation in the heart muscle. A substantial number of such patients appear to have a low-grade, smoldering myocarditis that will eventually evolve to cardiac "burnout" and a fatal congestive cardiomyopathy. For additional therapeutic guidance for these patients, see this article: [http://orthomolecular.org/resources/omns/v17n24.shtml]. This myocarditis (heart muscle inflammation) can manifest as fatigue, intermittent chest pain, shortness of breath, abnormal heart rhythms, and sometimes even the development of inflammation and blood clotting problems in the coronary arteries that can lead to heart attacks. It is vital that this inflammation gets vigorously treated and ***completely resolved***. As such, there should be a high index of suspicion of its presence in anyone with even minimal symptoms after having had a COVID infection, as well as in anyone who has had a COVID vaccination, which involves the direct administration of the spike protein. Simply assume the spike protein is present and ***replicating***, and proceed with an aggressive protocol to eliminate it completely.

Many viruses and pathogens, especially COVID, will typically persist in the body, especially in the upper and lower gastrointestinal tract. Because of this, anyone who feels completely recovered from COVID but who never received a definitive virus-killing treatment in the course of recovery (ivermectin, ozone, vitamin C, hydrogen peroxide nebulization, etc.) would be well-advised to follow the recommendations in the article noted above. Totally asymptomatic individuals who had blood microscopy examinations weeks after COVID vaccinations showed striking evidence of pathological red blood cell stickiness. This alone is clear justification for the application of vitamin C (with hydrocortisone if possible) along with any of a number of the other bio-oxidative therapies to resolve this stickiness as completely as possible. Of note, hydrogen peroxide nebulization is especially important in eliminating persistent pathogen presence anywhere in the gastrointestinal tract, which is the "pathogen reservoir" that most permits the persistence of COVID or any other pathogen following clinical resolution of the acute infection (Levy, 2021).

General Guide for the Administration of Hydrocortisone with Vitamin C

As is discussed at great length elsewhere, the importance of vitamin C in cellular physiology combined with the epigenetic defect in the liver preventing its synthesis in the body mandate that multi-gram daily doses of vitamin C should be part of the supplementation regimen of everyone

(Levy, 2002). Optimal health can never be achieved and maintained on the miniscule RDA of 75 to 90 mg of vitamin C per day for women and men. Optimal daily intake is much closer to amounts that are in excess of ***100-fold*** greater than these RDA recommendations. Furthermore, the amounts of vitamin C needed during periods of advanced oxidative stress can be in excess of ***1000-fold*** more than the RDA amounts. For an extensive guide to the multifaceted administration of vitamin C, see: Thomas-Levy-Guide-To-The-Optimal-Administration-of-Vitamin-C.pdf

While a clinical goal of normalizing health and returning abnormal laboratory tests to normal can often be achieved with many of the different approaches to vitamin C supplementation as outlined in the Guide above, there are also a number of clinical circumstances that do not readily normalize and would benefit greatly from the addition of hydrocortisone to optimize intracellular vitamin C levels. Furthermore, the appropriate addition of hydrocortisone to a vitamin C treatment protocol at the outset saves otherwise wasted steps in optimizing intracellular health as quickly as possible. **Any of the recommendations described below should be administered and followed with a qualified health care practitioner.** These recommendations are in addition to whatever else is being recommended in a treatment protocol, whether for an acute or a chronic condition.

For acute infections

When intravenous vitamin C is an option:
For 25 to 50 grams vitamin C infusions, 50 mg of hydrocortisone can be added to each infusion (or given as an IV push after infusion is started); lesser amounts of vitamin C (7.5 to 25 grams as an infusion or even as an IV push) can still be given with a total of 25 to 50 mg of hydrocortisone in the syringes as well [Riordan-Clinic-IVC-Push-Protocol]. If only oral hydrocortisone is available, 20 mg should be given orally approximately one hour before the vitamin C infusion or IV push is administered. This timing synchronizes the peak blood levels of the hydrocortisone and vitamin C. This approach can be continued until the acute infection (usually one to two weeks or less) is resolved.

When intravenous vitamin C is not an option:
5 grams of oral liposome-encapsulated vitamin C [www.livonlabs.com], along with 4 to 6 grams of sodium ascorbate powder (heaping teaspoon) in water or juice. This can be repeated several times daily based on clinical response. Other forms of oral vitamin C can be similarly-dosed. One vitamin C administration can be accompanied with 5 to 15 mg of hydrocortisone orally. It is best not to exceed a cumulative daily dose of 15 mg of hydrocortisone if this oral option is intended to be continued indefinitely.

For chronic infections and chronic diseases

It is optimal for patients in this group to receive testing to determine baseline and stress-related blood levels of cortisol. This establishes clearly the underlying adequacy of the adrenal glands for producing cortisol under both baseline and circumstances of acute oxidative stress. While everybody can benefit from the vitamin C-hydrocortisone combinations being presented, this testing can better identify those patients who most need this kind of antioxidant support indefinitely. Optimizing intracellular vitamin C must be a lifelong therapeutic goal.

As caring for patients with chronic infections and chronic diseases is highly individualized, there can be no fixed recommendations. Availability, convenience, and expense are important dictating factors in how often someone can receive vitamin C infusions. When this is an early part of a long-term treatment protocol, the recommendations noted for acute infections can be employed, and after a couple weeks, the oral vitamin C/hydrocortisone approach can be adopted. When vitamin C infusions are given intermittently but indefinitely, as one or more times monthly for a cancer patient, the hydrocortisone can always be added.

Many patients can benefit from simply taking 5 mg of hydrocortisone orally every time they take their oral form of vitamin C, up to three times daily (15 mg of hydrocortisone total per day). However, all of these possibilities can only be realized with the guidance of a physician or other health care professional who is closely following the clinical response and serial blood testing of a given patient, and who is able to prescribe the oral hydrocortisone tablets. The potential variations in the application of vitamin C with hydrocortisone are numerous.

Summary

Hydrocortisone plays an active role in facilitating the uptake of vitamin C into the cells of the body. Since the ultimate health of any cell is directly reflected in the vitamin C status in the cytoplasm, attention should always be paid to taking whatever measures are available for optimizing the concentrations of vitamin C in all the cells of the body. Furthermore, both vitamin C and hydrocortisone have been established to be the most potent and naturally available anti-inflammatory agents in existence. It would appear that the ability of hydrocortisone to augment cellular vitamin C uptake is likely the primary reason that it has its potent anti-inflammatory properties.

While very highly-dosed vitamin C does not require "assistance" to optimize its intracellular levels, relatively few physicians are comfortable applying such dosages. Because of this, combining hydrocortisone along with lower-dosed vitamin C can greatly increase the number of patients who can still optimize their health with vitamin C therapy.

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