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SUGGESTIONS FOR RESEARCH PROTOCOLS AND PRECAUTIONS IN THE ORAL ADMINISTRATION OF OXIDES OF CHLORINE

Introduction And Disclaimers

Since the publication of the experiences of Mr. Jim Humble, since the postings of numerous informative websites, since the postings of a plethora of blogs pertaining to the oral use of acidified sodium chlorite, and since the recent availability of solutions of sodium chlorite, numerous questions and concerns have arisen. The need for a biochemically and physiologically based commentary became apparent to this author. This is written to careful physicians for research purposes only. The writer offers this information for educational and safety minded purposes, exercising constitutionally protected freedom of speech and press. This is not to establish a doctor-patient relationship nor to provide medical advice. No promises nor guarantees nor labels of any kind are expressed or implied. The views, ideas, opinions, beliefs and suggestions expressed herein are subject to change without notice.

Oxidants, Reductants And Antioxidants

"Oxidants" are atoms or molecules which pull electrons off of or away from other atoms or molecules. Some atoms or molecules release electrons to oxidants. These are called "reductants". Strong oxidants in high enough doses are generally toxic to living cells, because they react with too many oxidant sensitive molecules all at once. At lower doses certain oxidants may cause stress or damage if certain oxidant sensitive molecules are eliminated more rapidly than they are replaced. This is an important mechanism in various diseases which involve chronic inflammation, radiation or chemical poisoning. In efforts to minimize damage from oxidative stress, the so called "antioxidants" have been researched and made available. Antioxidants react with oxidants preferentially and thereby protect more precious cellular components.

General Effects On RBCs

At low dose exposures (in other words received in amounts well below the toxic threshold) oxidants can stimulate certain beneficial physiologic effects. In live red blood cells exposed to oxidants some of their glutathione (GSH) is converted to glutathione disulfide (GSSG). This is usually harmless as living red blood cells can rapidly replace the lost hydrogen atoms and replenish the glutathione (GSH). The restoration process produces 2,3-diphosphoglyerate (2,3-DPG) as a by-product. Increased 2,3-DPG causes a beneficial side-effect. It attaches to hemoglobin, the main oxygen (O2) carrying protein in red blood cells, and causes this to hold oxygen more loosely. As a result, blood flowing through the peripheral tissues releases more oxygen (O2). This enables the affected tissues to generate more energy. This explains why many patients treated with low doses of medicinal oxidants subjectively experience a boost in pep and energy.

General Effects On WBCs

White blood cells respond differently to oxidants. At suitably low doses living white blood cells are induced to produce "cytokines". These are specialized protein messenger molecules, which diffuse throughout the body. When these cytokines contact other white blood cells of the immune system, such cells are stimulated to mount an enhanced attack against infection. This represents the usual or main benefit of oxidative medicine, to stimulate the immune system. If the oxidant dose is too high the effected white blood cells may be stunned because of oxidative stress and fail to produce cytokines. Therefore, protocols have been carefully developed to induce optimal immune stimulation without this contraproductive stunning effect.

General Effects On Pathogens

A third benefit of oxidative medicine is to use oxidants as disinfectants. These are traditionally applied externally. Examples are suitably calibrated solutions of iodine, hydrogen peroxide, sodium hypochlorite, ozone or chlorine dioxide. Many disease producing organisms are more sensitive to certain oxidants than are host cells. This is why at sites of infection activated white blood cells produce strong oxidants in vivo to directly kill pathogens. If a proposed medicinal oxidant can be safely tolerated internally, it becomes a candidate for internal use as an antimicrobial agent. This would mimic the natural immune function of using oxidants in vivo to destroy pathogens. However, this strategy will only succeed if the following conditions are met.

3 Conditions For Success
1. the pathogen must be sensitive to oxidation 2. a sufficiently high dose of oxidant must be delivered to the site of infection 3. the dose must be tolerable by the host

Oxides Of Chlorine As Orally Applicable Disinfectants

Sodium chlorite (NaClO2) and the more potent chlorine dioxide (ClO2) appear so far (anecdotally at least) to be good candidates for internal use. They both seem tolerable orally if appropriately dosed and suitably diluted in water prior to administration. Intermittent application in doses as high as 2 mg per kilogram per day have been safely administered. In many cases of malaria clinical success has been reported after as few as one or two treatments. Biochemical literature supports the view that Plasmodia are oxidant sensitive. Anecdotal reports of success in certain bacterial infections have also been noted. How this may work in the treatment of other infections remains to be carefully investigated and reported.

intermittent application of 2 mg/kg/day

Many have justifiably criticized the use of elemental chlorine (Cl2) as a food or water additive because of its tendency to react with hydrocarbons (C-H) to produce organic chlorides (C-Cl), which are toxic byproducts. Similarly elemental chlorine (Cl2) reacts with various amines (C-NH2) to produce chloramines (C-NH-Cl), which are also toxic. Chorine dioxide and sodium chlorite on the other hand fail to produce any significant quantities of these toxic byproducts.

Optimal protocols for the oral administration of certain oxides of chlorine are discussed below. Comparative advantages and disadvantages of each protocol are discussed. It is the purpose of the author to minimize risk, while maximizing success, as these treatments are evaluated in the context of responsible and legal research. Special admonitions against acute overdosing and against long term overuse are included. Failure to heed appropriate warnings could cause unnecessary adverse reactions. This in turn could result in adversarial social or political or legal proceedings. This would wrongfully

condemn potentially beneficial therapies. Then many people who need these therapies will suffer unjustly as access is idealogically or forceably blocked. Special precautions must also be heeded which are necessary to insure that the therapy is effective. If such issues are not consistently respected, then rampant clinical failures could also bring the therapy into disrepute.

Sodium Chlorite Solutions

The preparation of chlorine dioxide (ClO2) for oral use is now described. This procedure is low in cost and easy to provide. Sodium chlorite (NaClO2) not to be confused with sodium chloride (NaCl) is available from most manufacturers as "technical grade". This is in essence actually 80% sodium chlorite and about 19% sodium chloride. The remaining 1% is sodium hydroxide (NaOH) and sodium chlorate (NaClO3). Chlorate (ClO3-) is left over from the manufacturing process and in this context can be considered a harmless excipient. A little hydroxide (OH-) is a necessary stabilizer to protect the chlorite (ClO2-) in water solution.

Much of the sodium chlorite solution available over the internet lately is being sold as the so-called "MMS". Mr. Jim Humble first recognized the effectiveness of oral sodium chlorite solution to treat malaria. He subsequently discovered enhanced effectiveness, if the sodium chlorite was acidified just prior to use. This process converts much of the chlorite into chlorine dioxide a more potent disinfectant. After careful experimentation with various concentrations, he came to prefer 28% technical grade sodium chlorite in water. The actual presence of sodium chlorite in such a preparation should therefore equal 80% times 28% or 22.4%. Therefore, every milliliter of this solution should provide 224 mg of actual sodium chlorite. He also preferred to dispense this solution using a dropper bottle. The particular droppers he favored dispensed 25 drops per ml. Therefore, using equipment modeled after his procedures delivers 224 mg / 25 = about 9 mg per drop. This would not present a problem if every dropper around the world were constructed exactly the same. "Drops" per se is a nonstandard means of communicating and metering dosages. The problem with "drops" is the high variability of drop sizes. Droppers are constructed which deliver drops as big as 15 per ml or as small as 30 per ml as any nurse or pharmacist could testify. Therefore to avoid misinterpretations and mistakes in dosing, all of the following protocol related information will be communicated in terms of internationally recognized units such as grams (g), milligrams (mg) and milliliters (ml) = cubic centimeters (CC). Those wishing to back-convert to "drops" must determine the exact drop size they are using. This is easy to do with a small graduated cylinder. Fill the dropper with fluid and then count the exact number of drops required to dispense exactly one cc of fluid into the graduated cylinder. Divide that into the known number of milligrams per cc of solution to calculate the actual milligrams per drop.

revised to 20 drops/ml = 11.2 mg/drop

The author favors the following procedures. It is relatively easy to weigh out 25 g of technical grade sodium chlorite and to dissolve this in 100 cc of water producing a 25% solution. Since technical grade is actually 80% sodium chlorite, the actual concentration of active ingredient is therefore 80% X 25% = 20%. This translates to a very convenient 200 mg per cc. Using a graduated pipette 1/2 cc dispenses exactly 100 mg. 0.2 cc dispenses 40 mg. 0.6 cc dispenses 120 mg. This technique is suitable for most adult dosages which range from 20 mg to 200 mg. If there is the need for smaller dosing such as in pediatrics, then 10% or 5% solutions of sodium chlorite can be prepared and appropriately labeled. 10% equals 100 mg per cc so 0.3 cc dispenses 30 mg. 5% equals 50 mg per cc so 0.1 cc dispenses 5 mg. See the table below to correlate sodium chlorite concentrations with actual dispensed quantities.

200 mg NaClO2/cc

dose 20 to 200 mg / day

Sodium Chlorite (NaClO2) Concentrations:
gross = percentage by weight of technical grade, prepared by weighing grams per 100cc water
net = actual percentage of active ingredient
mg/cc = milligrams per cubic centimeter using a graduated cylinder
mg/.1cc = milligrams per 1/10th cubic centimeter using a graduated pipette
mg/gtt = milligrams per drop using various droppers
if # = drops per cc for that particular dropper type

gross	net	mg/cc	mg/.1cc	mg/gtt if 30	mg/gtt if 25	mg/gtt if 20	mg/gtt if 15
28%	22.4%	224	22.4	7.5	9	11.2	14.9
25%	20.0%	200	20	6.7	8	10	13.3
12.5%	10.0%	100	10	3.3	4	5	6.7
6.25%	5.0%	50	5	1.7	2	2.5	3.3

Dosing Of Sodium Chlorite

Next is discussed the dosimetry of sodium chlorite (NaClO2). The weight of the patient should be determined in kilograms. If the weight is known in pounds, then that number must be divided by 2.2 to properly calculate the kilogram weight. For example, an adult weighing 187 lbs. also weighs 85 kg. A child weighing 8.8 lbs. also weighs 4 kg. The usual or appropriate dose so far seems to be about 1 mg per kg per day. Therefore the adult described above could take about 85 mg in one day. He/she might start at 20 mg for the first treatment. Later he/she could gradually work up at subsequent treatments to 170 mg maximum. The average 4kg child might take about 4 mg, starting at 1 mg or less, then working up to 8 mg maximum. Special care must be taken under all circumstances never to overdose using these oxidants. The lethal dose is estimated at about 100 mg per kg. Thus an adult could be killed taking 8 to 10 grams. This is about one hundred times (100x) the appropriate dose. An infant could be killed taking as little as 400 mg. One may consult the table below to avoid overdosing. Listed is the suggested dose per day in milligrams.

lethal dose 100 mg/kg

Probable Dosages For Various Body Weights					
Weight	Weight	Start	Average	Maximum	Lethal
9 lb	4 kg	1 mg	4 mg	8 mg	400 mg
13 lb	6 kg	1.5 mg	6 mg	12 mg	600 mg
22 lb	10 kg	2.5 mg	10 mg	20 mg	1 g
30 lb	14 kg	3.5 mg	14 mg	28 mg	1.4 g

weight		start	avg	max	lethal
44 lb	20 kg	5 mg	20 mg	40 mg	2 g
66 lb	30 kg	7.5 mg	30 mg	60 mg	3 g
100 lb	45 kg	11 mg	45 mg	90 mg	4.5 g
154 lb	70 kg	17.5 mg	70 mg	140 mg	7 g
220+ lb	100+ kg	25 mg	100 mg	200 mg	10 g

start at 25 mg/kg/ day, build to 200

Certain tests are reasonably expected to be useful to monitor toxicity. Elevated methemoglobin levels reflect overly oxidized blood. Elevated urea or creatinine levels reflect kidney damage. Whenever higher than usual doses are to be administered, special attention must be applied regarding kidney damage especially if the urine is acidic. Acid renders the oxides of chlorine more reactive. Alkalinity stabilizes oxides of chlorine. Urine pH should be measured if higher than average doses are going to be applied. If the urine is abnormally acidic (pH < 6), special measures should be taken to raise the urinary pH to protect the kidneys.

Acidifying Sodium Chlorite

Once the appropriate dose of sodium chlorite (NaClO2) is determined, this amount should be measured and dispensed into a test tube or cup. It may next be acidified by addition of an appropriately selected acid solution. The goal is to produce a pH of the reacting mix in the range of 2 to 3 pH units. This is optimal for the production of chlorine dioxide (ClO2) from chlorite (ClO2-). This can be accomplished using acetic acid, lactic acid, citric acid, tartaric acid or most other edible strong acids. Citric acid is preferred as it is available as a dry powder for easy packaging and delivery in dry plastic bags anywhere in the world. Citric acid is also relatively inexpensive and generally recognized as a safe food additive. Acetic acid and lactic acid are liquids which present special packaging and handling problems. Suitably diluted sulfuric acid, phosphoric acid or hydrochloric acid could be used but preparation from more concentrated source materials would be dangerous for those not experienced with chemical handling. Ascorbic acid must never be used as this is a reductant and would immediately destroy the oxidants in the preparation. Toxic acids must never be used.

Citric acid solution is easy to prepare as a 10% solution, however, 5% to 20% could also be used. The important issue is the pH of the reacting mixture, which should be between 2 and 3 units. This can be checked using pH paper. It may seem that a large excess of citric acid solution would more assuredly accomplish the desired pH change. However, to get a decent yield of chlorine dioxide the volume of the reacting solution must be limited. Too large a reacting volume is actually contra- productive as this will diminish the rate of chlorine dioxide production. Upon mixing chlorite (ClO2-) with acid (H+) a small amount of chlorous acid (HClO2) is first produced. Chlorite (ClO2-) anions are stablized by their negative charge. They are repeled away from the very electrons they may want to oxidize. Chlorous acid (HClO2) however is neutral in charge and therefore more readily able to approach an electron and to abstract it. Therefore, if chlorous acid remains in close proximity to other chlorite anions it can successfully oxidize them. This explains exactly how chlorine dioxide (ClO2) is producible by acidifying a sodium chlorite solution. The following cascade of reactions occurs.

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ClO2-     +  H+      --->  HClO2
HClO2     +  ClO2-   --->  ClO2 + [HOClO-]
[HOClO-]  +  H+      --->  [HOClOH]  --->  ClO + H2O
ClO       +  ClO2-   --->  ClO2 + ClO-
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ClO-      +  H+       --->  HClO
HClO      +  ClO2-    --->  ClO2 + [HClO-]
[HClO-]   +  H+       --->  [HClOH]   --->  Cl   + H2O
Cl        +  ClO2-    --->  ClO2 + Cl-

Adding these equations together describes the overall reaction as:

5 ClO2-   +  4 H+    --->  4 ClO2  +  2 H2O  + Cl-
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Note that for these successively reduced species of chlorine compounds to be successful in oxidizing the chlorite anion, they must remain in close proximity. If these reactants are too widely dispersed by dilution the opportunities to react are severely limited. As a result production of chlorine dioxide slows markedly. Therefore one must add sufficient acid to start the reaction but not overly increase the reacting volume. It seems most practical to use 10% citric acid at a volume about equal to that of the sodium chlorite solution dispensed. Under most circumstances 3 minutes of reacting time is appropriate. Chlorine dioxide can be seen as a rapid color change to bright yellow. It smells exactly like elemental chlorine (Cl2). If for some reason a more rapid reacting time is desired, 20% citric acid can be used.

The freshly prepared solution is now properly designated as "acidified sodium chlorite". It contains unreacted sodium chlorite, freshly made chlorine dioxide, sodium chloride, citric acid and sodium citrate. This should be diluted about 100 fold before administrating to avoid burning the mouth and throat. One cup of water should be sufficient. The resultant drink is pale yellow and tastes like sour, salty, swimming pool water. This should be chased with more drinking water to minimize stomach irritation and nausea. This completes the treatment. To minimize stomach irritation the drink can be divided in half and administered in two separate sessions on the same day.

Side Effects

The usual direct side effects are a transient nausea, headache, sweating and drowsiness. The nausea is usually mild and lasts for under one hour. This usually readily remits upon drinking more water. It can be prevented or minimized by eating a starchy meal prior to treatment. Headache and drowsiness are highly individual in severity and duration.

nausea, up to 1 hr, can be decreased by eating a starchy meal prior to treatment. Headache and drowsiness vary. Die-off!

The most problematic side effects are not attributable to any direct irritating effect of the oxidants. Instead they are due to the rapid success of the oxidants in killing pathogens. As the disease causing organisms die off they disintegrate releasing antigens which in turn provoke an inflammatory response from the immune system. This is often observed in clinical practice using common antibiotics. The phenomenon and is conventionally designated the "Jarrisch-Herxheimer reaction" or "J-H reaction". This is a necessary physiologic attack and clean-up process. The severity of symptoms depends on the number of dying pathogens, the antigenicity of the debris, the sensitivity of the immune system and the site of the infection. Possible symptoms are noted in the table below. J-H reactions usually last a few hours or rarely as long as a few days. Once the J-H reaction is completed, the patient begins to experience remission. The good news is that usually the more severe the J-H reaction, the more extensive the die-off, the more complete the remission and the longer the disease free interval. In such cases the need for frequent retreatments is minimized.

Common Symptoms of J-H Reactions:		
fever	chills	malaise

sweating	nausea	pain
tenderness	headache	body aches
fatigue	diarrhea	

With this in mind most patients prefer to accept the transient discomforts of the J-H reaction. However, such a level of courage and resolve may not always be necessary. If according to the clinical judgement of the treating physician or the tolerance of the individual patient an abortion of the J-H reaction is desired, four options are available.

Options For Treating J-H Reactions

1. Firstly, any residual unreacted chlorine dioxide in the body can be rapidly quenched by taking a large dose of ascorbic acid (aka vitamin C). One to ten grams should suffice.
2. Since J-H reactions are primarily just manifestations of common inflammatory processes, any systemically active antiinflammatory drug can be taken. Examples would be: aspirin, acetaminophen, ibuprofen, naproxen, etodolac, celecoxib, et cetera.
3. Omega-3 oil supplements might also be found helpful.
4. Corticosteroids could also be used, if a superpotent antiinflammatory effect is deemed appropriate.

Once the J-H reaction is aborted a suitable rest period may be determined. Afterwards retreatment at a lower dose may be applied.

Mechanisms Behind Treatment Success Or Failure

If the pathogen is especially sensitive to oxidation, retreatment should not be necessary. However, if one or more of the following variables contravenes, relative treatment failure may occur, and the treatment may need to be repeated.

Confounding Factors

1. too little oxidant was administered;
2. the pathogen is fairly resistant to oxidants;
3. the pathogen converts to alternate forms such as spores, cysts or eggs;
4. the pathogen is sequestered in sites remote from oxidant penetration;
5. too many antioxidants or drugs were present at the time of treatment, which quenched the medicinal oxidants.

Incompatibilities

There are important substance-oxidant incompatibilities which must now be addressed. Various classes of substances must not be present in the stomach at the time of the acidified sodium chlorite treatment, if any beneficial results are to be expected. Of paramount importance is the avoidance of antioxidants together with the treatment. Antioxidants are usually thiol compounds or phenolic compounds, which can specifically eliminate chlorine dioxide. Chlorine dioxide is used in industry to specifically target and to destroy thiols and phenols, because they readily react together and destroy each other. Examples of chlorine dioxide quenching compounds are: N-acetyl-L-cysteine, glutathione, alpha-lipoic acid, ascorbic acid, polyphenols, tocopherols, bioflavonoids, anthocyanidins, benzaldehyde, cinnamaldehyde, juice concentrates and many herbal remedies. Most fruits especially grapes and berries are rich sources of polyphenolic antioxidants. Examples of herbs rich in antioxidant polyphenols are: chocolate, tea, coffee, turmeric, silymarin, licorice, ginkgo, olive. Sulfur

rich foods also eliminate chlorine dioxide if present in the stomach at the time of treatment. Examples include: garlic, onion, leek, asparagus, beans, peas, egg, milk and even white potatoe (due to alpha-lipoic acid). Protein must also not be present in the stomach at the time of treatment. Proteins are made of amino acids which present an abundance of phenols, organic sulfides, thiols and secondary amines, which react with and eliminate chlorine dioxide on contact. L-tyrosine has a phenol group. L-methionine is a sulfide. L-cysteine is a thiol. L-tryptophan, L-proline and L-histidine have secondary amino groups. Certain B-complex vitamins are similarly reactive such as: thiamine, riboflavin, folate, pantothenate. Finally many drugs contain secondary amines, tertiary amines, thiols, sulfides or phenols. Under physician direction these may also need to be identified and withheld on the day of treatment or at least not taken at the time of treatment. While antioxidants and vitamin supplements are generally speaking healthy for preventive and longevity purposes, and while these are beneficial in the treatment of many chronic diseases, these are incompatible at the moment of the acidified sodium chlorite treatment. Therefore, fruit, fruit juices, fruit concentrates, wines, green drinks, herbs, protein, most vitamins and most drugs should not be taken at the time of treatment and certainly not mixed with the acidified sodium chlorite solution. If these principles are not respected, little if any oxidants will survive to kill pathogens and no benefit should be expected.

wait 4 hours after a protein or fruit meal. After eating, wait 3 hours.

If a person already ate some incompatible food such as protein or fruit prior to a scheduled treatment, then they must wait at least four hours for these items to pass through the stomach before taking the treatment. The next day after treatment the above described incompatible substances can be resumed. Protein could probably be eaten as soon as 3 hours after treatment.

Anyone who claims success taking fruit juices with acidified sodium chlorite has succeeded in spite of this quenching problem. Higher and higher doses of oxidants would have to be administered to get past the antioxidants. If someone is already apparently tolerating especially high doses of oxides of chlorine, because these oxidants are being taken with antioxidants, then such a person is at risk of oxidant overdose if the concomittent antioxidants are suddenly stopped. The most appropriate action would be to hold the antioxidants and to back down to a much lower dose of the oxidants.

Nutrient poor white starches on the other hand may be present in the stomach at the time of treatment. These may even be taken with or mixed with the diluted solution. These do not react readily with chlorine dioxide. Examples of allowable junky starchy foods are: white bread, casava, grits, white wheat pasta, white rice, saltines. Note that white potatoes are not included in this list because they are rich in alpha-lipoic acid a sulfur based antioxidant. Even though most sulfur compounds react with chlorine dioxide, oxidized sulfur compounds such as DMSO, MSM, taurine or sulfate are probably not reactive. Pending further knowledge it seems likely that carotenoids and polyunsaturated fatty acids do not quench chlorine dioxide.

starches:
white rice
white wheat
casava
grits
saltines
NOT potatoes

Incompatible Substances
Classified According To Reactive Groups:

- aldehydes
- enediols
- phenols & polyphenols
- anilines
- secondary or tertiary amines
- thiols, sulfides, disulfides
- transition metals in lower oxidation states

allowed sulfurs:
DMSO
MSM
taurine
__ sulfate
carotenoids
pufas

Note: Most drugs contain one or more of the above reactive groups. A drug reference showing the structural formula must be consulted. When in doubt do not take most drugs with these oxidants.

Incompatible Foods:

all ascorbates
all proteins, for example:
wheat germ, nuts, peas, beans
fish, poultry, meat, milk, eggs
most antioxidants, for example:
N-acetyl-L-cysteine, alpha-lipoic acid, SAME,
glutathione, quercetin, BHT, BHA, tocopherol
most B-complex vitamins, for example:
thiamine, riboflavin, niacin, pantothenic acid, folic acid,
para-aminobenzoic acid, cyanocobalamin, biotin, carnosine
most fruit especially berries, apples, oranges, grapes,
cherries, figs
most herbs, for example:
chocolate, green tea, coffee, turmeric,
silymarin, licorice, ginkgo, olive, cinnamon
Allium species:
onion, leek, shallot, garlic, chive
Brassica species:
cabbage, kale, broccoli, cauliflower,
turnip, mustard, wasabi
Asparagus species
Solanum tuberosum = white potato

Choices Among Protocols

Four oral protocols will now be described and the relative advantages and disadvantages discussed.

4 Oral Protocols

1) regular daily dosing of sodium chlorite
2) occassional dosing of sodium chlorite
3) regular daily dosing of acidified sodium chlorite
4) occassional dosing of acidified sodium chlorite

Occassional can mean as often as every other day, once per week, or as rarely as once per month. In all protocols the starting dosage of sodium chlorite should be about 0.25 mg per kg per day or less and gradually increased to a maximum of 2 mg per kg per day as tolerated. Starting especially low is important in severely ill or debilitated patients who may have difficulty tolerating the oxidant primarily or who may experience an intolerable J-H reaction. In most cases of chronic infection protocols 2) and 4) are preferrable to 1) and 3) because the treatment free interval allows time to complete J-H reactions and to determine if there is a sufficient remission or a subsequent need for retreatment. If all signs and symptoms completely remit, then there is no good purpose to repeat the treatment. Unused precursor solutions may be saved in a cool dark place for future needs. In certain cases of chronic fatigue syndrome in which a chronic infectious illness is suspected but not identified, weekly dosing using protocol 2) or 4) may be appropriate initially. Treatment free intervals may be extended if warranted by long remissions. This minimizes any possible adverse effects of repeated oxidant exposure and only applies the oxidants when needed. In choosing between protocols 2) and 4), the advantage of 4) is the much greater potency of chlorine dioxide in killing pathogens as compared to sodium chlorite. The clinical success rates should be much higher, less frequent

treatments should be required and longer remissions should be expected. On the other hand using the less potent sodium chlorite without acid activation should result in less nausea and less severity of J-H reactions. Another advantage of the intermittent protocols 2) and 4) is that beneficial nutrients, antioxidants and drugs may be continued between treatment days. Protocols 1) and 3) present the disadvantage of a constant conflict between the oxidants and beneficial nutrients, antioxidants and necessary drugs. Uninterrupted strong oxidant exposure over the long haul as in protocols 1) or 3) could dangerously deplete oxidant sensitive molecules of the host. This could contribute to chronic degenerative disease from oxidative stress. Continuous dosing of strong oxidants of any type would never allow effected cells to heal themselves through the normal physiologic process of antioxidant adaptation. If regular daily dosing is kept low enough to not defeat antioxidant adaptation and cellular restoration, then that dose would probably be too weak to kill any pathogens. Furthermore, chronic or repeated exposure of fetuses, infants or young children above EPA allowed limits of 0.8 mg/liter in public drinking water is thought by many to risk nervous system damage. The thyroid hormones T3 and T4 are phenols and therefore subject to destruction by chlorine dioxide. Therefore, it is preferable to avoid regular daily use of any oxides of chlorine except in special circumstances. For example, in severe life threatening acute infections such as pneumonias, bacteremias, cavitary abscesses or meningitides the risk of permitting the infection to progress may be far greater than any risks of oxidative stress or risks of thyroid hormone destruction.

Possibilities With Cancer

While it is too early to conject with any certainty, it is theoretically possible that protocol 1) may be the best option if oxides of chlorine are to be tested in the treatment of cancer. Repeated dosing using sodium chlorite should be better tolerated than acidified sodium chlorite, because alkaline or neutral chlorite is less reactive than chlorine dioxide towards most oxidant sensitive molecules of host cells. This should make plain sodium chlorite safer to continue repeating long term. In most cancer cases a selective advantage theoretically exists. Most tumors produce relatively high levels of carbonic acid and lactic acid. These acids in the tumor should activate the chlorite producing more potent oxidants as described by the chemical equations noted above. If these highly reactive oxidants are only produced in the tumors and nowhere else in the host, then highly acidic tumors could safely be destroyed.

Unfortunately, tumors are not the only tissues known to produce or to concentrate acids. Isometric contraction of muscles is famous for rapidly producing large quantities of carbonic and lactic acid. Ischemic tissues anywhere in the body resulting from arterial obtruction or from localized swelling similarly suffer from acid build-up. Kidneys must at times concentrate acid for excretion. The parietal cells of the stomach actively produce hydrochloric acid for digestion. Therefore, if unusually high doses of sodium chlorite are needed, special attention and care must be applied to avoid damage to the muscles, ischemic tissues, kidneys and stomach. Risk of harm to the kidneys might be minimizable, if alkalinizing supplements are provided so that an acidic urine will not be produced. Incidently, most cancer patients, most allergic patients, and many chronically ill patients present initially with acidic urine and acidic saliva. A reversible inhibitor of hydrochloric acid production in the parietal cells should suffice to protect the stomach from side-effects including nausea. However, such inhibitor would need to be nonreactive with the oxides of chlorine. Unfortunately, every histamine-2 blocker, every azole-type proton pump inhibitor, and every anticholinergic in common use, that the author has checked structurally, contains functional groups probably reactive with chlorine dioxide.

Conclusions

Sodium chlorite proper or acidified sodium chlorite due to their unique chemical properties may sooner or later be proven to be powerful infection fighting remedies. Furthermore, these may find application in diseases for which no remedy currently exists and in diseases that have acquired resistance to current remedies. Research to support or refute these hypotheses is urgently needed. The author hopes this complilation of procedural instructions and admonitions is understandable and helpful. Physicians interested to legally investigate the oxides of chlorine in the treatment of difficult infections and possibly cancer are to be supported and commended.

Please direct inquiries, reports and suggestions to Dr. Hesselink,
bioredox1/at/gmail.com (/at/ is written here in place of the @ sign to thwart automated junk email programs.)

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