

Dr. Ott's Oxygen (O2) Treatment and Therapy™

Scientific Data Sheet and Summary

By A. True Ott, PhD, ND

To fully understand the incredible function of the Oxygen Treatment and Therapy in the human body, the practitioner, health care professional, and/or inquisitive lay person needs to familiarize themselves with the functions of a cellular enzyme called Myeloperoxidase (aka MPO for short), as well as the functions of hypochlorous acid (HClO).

In the early 1920's, Dr. William F. Koch discovered that all disease states are merely different manifestations of OXYGEN DEFICIENCIES at the cellular level. He called these oxygen deficiencies the "Least Common Denominator" in maintaining good health, which is of course synonymous with PREVENTING disease states from forming in the first place. Therefore, proper oxygenation via all-natural mineral precursors should be the 1st and primary goal of cellular nutrition advocates as well.

As a gifted bio-chemist, Dr. Koch discovered that combining certain carbonyl-group mineral elements in vitro quickly produced a unique form of hypochlorous acid that he named "glyoxilide". I submit he named this catalyst glyoxilide because when it came in contact with the carbons in GLUCOSE (aka blood sugar) in vivo (in the body) – massive amounts of singlet, negatively charged oxygen molecules were produced. In turn, these oxygen molecules proved to be highly effective in eliminating anaerobic pathogens (harmful bacteria, viruses, fungi and parasites) from the body in a completely safe, non-obtrusive, and effective manner.

In 1925, Dr. Edward Carl Rosenow (who spent over 60 years conducting research at the Mayo Clinic) not only discovered that rheumatic fever was caused by a streptococcus germ – but that there are literally millions of tiny micro-organisms continually living and colonizing in the human body. Each different strain of "bug" causes different problems and health challenges. Some colonize in the hair and scalp resulting in dandruff. Others colonize in the mouth causing cavities and gum diseases.

Dr. Rosenow found that individuals with compromised immune systems were consistently low in blood oxygen. In turn, these microscopic critters would proliferate and be free to do such nasty things as: 1) consume the cartilage and sulfur of the joints causing painful rheumatoid arthritis 2) excrete waste material in the form of electron-deficient calcium that hardens bones and makes them brittle – and when lodged in the liver and kidneys would form stones 3) colonize in the lining of the heart arteries, leaving their excrement on the walls of the arteries in the form of plaque. 4) colonize in the Central Nervous System (CNS) of the spinal column and brain, making meals of the myelin sheath nerve coatings thus short-circuiting the central computer of the brain resulting in ALS, MS, ADHD, and Alzheimer's. 5) attack individual cells and enter the cellular membrane, eventually building cocoons around the DNA-damaged cell; cutting off the oxygen-carrying blood so that the cell can only live and function as part of cancerous tumor. Thus, it is safe to say that disruption of the ability of the cells to produce MPO, HClO, and "glyoxilide" results in low oxygen levels, which in turn eventually forms a DISEASE STATE.

Dr. Koch quickly learned that such a simple formula providing powerful, life-giving OXYGEN to the cell would not only prevent disease states from forming, but had the strong potential to REVERSE so-called “incurable” disease states as well. So, like any honest medical professional, Dr. Koch proceeded to test his research and theory – and the results were immediate and dramatic to say the least. Cancer tumors shrunk and disappeared, diabetes mellitus vanished, mental disorders reversed, and viral plagues were eliminated. (See www.williamfkoch.com)

Sadly, however, Dr. Koch also became painfully aware that reversing such chronic disease states in such a permanent manner meant drastically decreased PROFITS in the bank accounts of the burgeoning PHARMACEUTICAL HOUSES owned and operated primarily by John D. Rockefeller. It meant that in order for “Big Pharma” to flourish, Dr. Koch’s research and results must be hidden and discredited at all costs. This is exactly what happened – and this vitally important NUTRITIONAL PRODUCT was lost to the world for over 50 years. Untold millions have needlessly suffered and died horrible deaths in order to enrich a few evil men.

In simple, honest words, the O.T.T. (Oxygen Treatment and Therapy) produces the mineral catalyst that Dr. Koch named “glyoxilide”. When taken as instructed, the O.T.T. mineral catalyst is completely non-toxic and safe. When the catalyst is absorbed into the bloodstream and encounters carbon/glucose molecules, large amounts of OXYGEN is indeed created, and harmful NITROGEN is reduced. In turn, the free oxygen destroys anaerobic bacteria, viruses, yeast, and parasites the way NATURE intended – through the mechanism of OXIDATION in much the same way as OZONE IN WATER destroys the same harmful pathogens.

For those wishing to have more “3rd party” validation of this science and my claims, I am including a paper authored by Dr. Maureen Petersen, MD, Cecilia Mikita, MD, MPH, and Javed Sheikh, MD on a condition called Myeloperoxidase (MPO) Deficiency, (but what should actually be called serum oxygen deficiency) and Wikipedia’s detailed report on hypochlorous acid. I have highlighted in yellow the more relevant parts.

A. True Ott, PhD, ND

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Myeloperoxidase Deficiency

Maureen M Petersen, MD, Fellow in Allergy and Immunology, Walter Reed Army Medical Center

Cecilia P Mikita, MD, MPH, Assistant Professor of Pediatrics and Medicine, Uniformed Services University of the Health Sciences; Associate Program Director of Allergy-Immunology Fellowship, Chief of Clinical Services, Staff Allergist/Immunologist, Walter Reed Army Medical Center; **Javed Sheikh, MD**, Assistant Professor of Medicine, Harvard Medical School; Clinical Director, Division of Allergy and Inflammation, Beth Israel Deaconess Medical Center; Clinical Director, Center for Eosinophilic Disorders, Beth Israel Deaconess Medical Center

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Introduction

Background

Myeloperoxidase (MPO) is a human enzyme in the azurophilic granules of neutrophils and in the lysosomes of monocytes. Its major role is to aid in microbial killing. Although MPO received little clinical attention until 1966, the enzyme was first isolated in 1941, and deficiency of MPO was first described in 1954. Some patients with MPO deficiency have impaired microbial killing, but most are asymptomatic.

The condition was initially believed to be very rare with only 15 cases were reported before the 1970s. However, modern laboratory techniques have allowed researchers to discover that MPO deficiency is actually more common than previously described but without clinical relevance.

Pathophysiology

Normal function of myeloperoxidase

MPO, a heme-containing protein, is found in the azurophilic granules of neutrophils and in the lysosomes of monocytes in humans; however, monocytes contain only about a third of the MPO present in neutrophils. When neutrophils become activated during phagocytosis, they undergo a process referred to as a respiratory burst. This respiratory burst causes production of superoxide, hydrogen peroxide, and other reactive oxygen derivatives, which are all toxic to microbes. During respiratory bursts, granule contents are released into the phagolysosomes and outside the cell, allowing released contents to come into contact with any microbes present. Experiments conducted in the 1960s showed that MPO catalyzes the conversion of hydrogen peroxide and chloride ions (Cl) into hypochlorous acid.¹ Hypochlorous acid is 50 times more potent in microbial killing than hydrogen peroxide.

MPO also directly chlorinates phagocytosed bacteria; thus, the MPO-hydrogen peroxide-Cl system seems to have an important role in microbial killing. Although the exact mechanism by which microbial killing occurs is controversial, researchers are fairly certain that MPO is important for the process to optimally occur.

In addition to killing bacteria, the products of the MPO-hydrogen peroxide-Cl system are believed to play a role in killing fungi, parasites, protozoa, viruses, tumor cells, natural killer (NK) cells, red cells, and platelets. The MPO-hydrogen peroxide-Cl system is also believed to be involved in terminating the respiratory burst, because individuals with MPO deficiency have prolonged respiratory bursts. It may play a role in downregulating the inflammatory response by regulating NK cells, decreasing peptide binding to chemotactic receptors, and auto-oxidizing and inactivating products of polymorphonuclear leukocytes (PMNs), such as α 1-proteinase inhibitor and chemotaxins.

Other functions of MPO include tyrosyl radical production and chlorination, generation of tyrosine peroxide, mediation of the adhesion of myeloid cells via β_2 -integrins, and oxidation of serum lipoproteins. MPO may have a role in atherosclerosis. Researchers have demonstrated that patients with stable coronary artery disease had an increased cardiovascular risk if plasma MPO levels were elevated.² A small study demonstrated that MPO deficiency may protect against cardiovascular disease.³ MPO may also have a role in carcinogenesis and degenerative neurological diseases. The understanding of MPO biology remains incomplete; much more remains to be discovered.

Normal myeloperoxidase production

MPO is a dimeric molecule, consisting of a pair of heavy-chain and light-chain protomers and 2 iron atoms. MPO is encoded by a single gene located on band 17q22-23. The mature enzyme is synthesized from a single polypeptide product. Therefore, the expression of the gene and the synthesis of MPO primarily occurs during the promyelocytic stage of myeloid development, concurrent with development of the azurophilic granules. The *MPO* gene encodes for a primary translational product, which is glycosylated to yield an enzymatically inactive precursor, apopro-MPO.

Apopro-MPO reversibly binds to chaperone proteins, calreticulin and calnexin, during protein maturation. This results in the subsequent binding of heme.⁴ Heme insertion induces conformational changes in the protein yielding pro-MPO, an enzymatically active precursor.⁵ Pro-MPO undergoes several complex conversions and eventually becomes mature MPO in the azurophilic granules, but the exact mechanisms are still poorly understood.

MPO should be distinguished from eosinophilic peroxidase (EPO), a different enzyme produced by a different gene. Although patients with MPO deficiency have decreased MPO activity in the neutrophils and monocytes, these patients usually have normal levels of EPO in eosinophils.

Pathophysiology of hereditary myeloperoxidase deficiency

Hereditary MPO deficiency was initially thought to follow the classic autosomal recessive pattern. A number of genetic mutations resulting in MPO deficiency have been identified, and many others may still be undiscovered. Researchers now believe that most patients are compound heterozygotes, which means that they have a different mutation on each allele, one from each parent. As with several other genetic diseases, numerous allele combinations can lead to the phenotype of MPO deficiency, which partially explains the variability of clinical features. Some mutations result in posttranslational defects; others (which are not yet clearly defined) seem to cause pretranslational defects, possibly due to structural alterations in the regulatory parts of the *MPO* gene. See Causes for a discussion of individual mutations that have been identified and their effects.

Some authors have proposed a bigenic model involving the interaction of 2 genes, such as a production gene and a regulatory gene. Overall, the genetic basis of this condition is now thought to be quite heterogeneous and complex. Undoubtedly, much remains to be discovered.

Pathophysiology of acquired myeloperoxidase deficiency

MPO deficiency in acquired cases is usually transient and generally resolves once the inciting condition improves. In addition, acquired MPO deficiency is usually partial and involves only a fraction of the PMNs.⁶ **The following conditions can lead to acquired MPO deficiency:**

- Pregnancy
- Lead intoxication - Inhibits heme synthesis (a component of mature MPO)
- Iron deficiency
- Severe infection - Secondary to PMN activation and "consumption" of MPO
- Thrombotic diseases
- Renal transplantation
- Diabetes mellitus
- Neuronal lipofuscinosis
- Drugs - Cytotoxic agents and some anti-inflammatory agents such as dapsone, 5-aminosalicylic acid, and sulfapyridine
- Disseminated cancers - Probably related to administration of cytostatic agents
- Several hematologic disorders and neoplasms especially those involving the maturation of granulocytes:
 - Acute myeloid leukemia (AML)
 - Chronic myeloid leukemia (CML)
 - Polycythemia vera
 - Hodgkin disease
 - Refractory megaloblastic anemia
 - Aplastic anemia
 - Myelofibrosis with myeloid metaplasia
 - Myelodysplastic syndromes

Microbial killing in myeloperoxidase deficiency

MPO-deficient neutrophils are normally able to phagocytose most microbes. However, the ability of MPO-deficient neutrophils to kill bacteria seems impaired to varying degrees. For organisms such as *Staphylococcus aureus*, *Serratia* species, and *Escherichia coli*, killing is initially impaired but then reaches normal levels after a period of time. This suggests that an apparently slower, alternative mechanism of killing can take over in MPO-deficient neutrophils.

The capacity to kill certain fungi, however, seems completely absent in MPO-deficient neutrophils. In vitro studies have shown that *Candida albicans*, *Candida krusei*, *Candida stellatoidea*, and *Candida tropicalis* cannot be killed by MPO-deficient PMNs. In contrast, an MPO-independent mechanism can kill *Candida glabrata*, *Candida parapsilosis*, and *Candida pseudotropicalis*. Even more interesting is that the hyphal elements of *Aspergillus fumigatus* and *C albicans* cannot be killed, but the spores of *A fumigatus* and the yeast phase of *C albicans* can be killed by an independent mechanism. This leads to the conclusion that bacterial killing may not necessarily be a problem for patients with MPO deficiency, but the killing of certain fungi may be a problem, depending on the severity of the deficiency.

Frequency

United States

Incidence rates from screening studies range from 1 case per 1400-2000 population.

International

One series found the prevalence of total or subtotal MPO deficiency to be 1 case per 2727 population.⁷ Prevalence rates in Japan have been reported to be much lower, with one study finding the prevalence

of total and partial deficiency to be 1 case per 57,135 population and 1 case per 17,501 population, respectively.⁸

Until the 1970s, only 15 cases of MPO deficiency had been reported worldwide. Because most cases are asymptomatic, very few people were evaluated for the deficiency. However, modern laboratory techniques, particularly the wider application of automated flow cytometry for determining WBC differentials, have allowed the screening of large study populations to determine the true prevalence of MPO deficiency.⁷

Mortality/Morbidity

European researchers evaluated patients with complete MPO deficiency and found that about half of the patients had infectious complications; the other half were asymptomatic. Approximately 10% of the cases involved life-threatening infectious complications. Other studies have reported that severe infections occur in fewer than 5% of patients with MPO deficiency; however, this frequency may be based on the inclusion of both complete and partial deficiencies. Generally, infections only occur in patients who have concomitant diabetes mellitus.

Clinical

History

- Recurrent infections
 - Most individuals with partial or total myeloperoxidase (MPO) deficiency have no increased frequency of infections, probably because MPO-independent mechanisms in the polymorphonuclear leukocytes (PMNs) can take over. In general, it is considered a relatively benign immunodeficiency and was removed from the Classification of Primary Immunodeficiency Disease by the Primary Immunodeficiency Disease Classification Committee of the International Union of the Immunologic Societies in 2005.
 - Severe infections are uncommon, occurring in fewer than 5% of patients with MPO deficiency. If infectious disease occurs, it is usually a fungal infection (particularly candidal, such as *C albicans* or *C tropicalis*) that occurs in a patient who also has diabetes mellitus. Patients without diabetes mellitus rarely have problems, although the reason for this is unknown. Possibly, MPO deficiency becomes clinically significant only in the presence of an additional defect in the host defense, or perhaps the MPO-independent system is defective in some patients with diabetes mellitus.
 - Physicians should entertain the diagnosis of MPO deficiency in cases of invasive fungal infection in a patient with no known predisposing immune defects (eg, chemotherapy, corticosteroid treatment) or in a patient with concomitant diabetes mellitus. Some consider peroxidase staining of the peripheral blood smear to be part of the complete evaluation of a patient with a suspected immunodeficiency.
- Increased incidence of malignancy
 - A strong association between total MPO deficiency and malignancies has been reported by several independent investigators. In vitro, MPO-deficient neutrophils have decreased destruction of malignant cells demonstrating that the MPO system plays a central role in tumor surveillance.⁶
 - MPO is released from neutrophils in lung tissue in response to pulmonary insult including damage secondary to tobacco smoke exposure. MPO has been shown to

convert the metabolites of benzo[a]pyrene from tobacco smoke into a highly reactive carcinogen. Researchers have demonstrated that decreased MPO can decrease lung cancer risk.⁹

Causes

- Hereditary cases can be caused by a number of mutations, including R569W, Y173C, M251T, G501S, and R499C.
 - R569W: This is the most common defect identified to date. Tryptophan is substituted for arginine at codon 569. Tryptophan cannot form electrostatic bonds. Most patients described have been compound heterozygotes, but one has been homozygous for this mutation. The mutation results in a maturational arrest at the stage of apopro-MPO that is unprocessed, enzymatically inactive, and undelivered to the azurophilic granules.¹⁰
 - Y173C: Cysteine is substituted for tyrosine at codon 173. This leads to an additional site for intramolecular disulfide bonds, which presumably leads to abnormal folding of the protein. Apopro-MPO is converted into pro-MPO, which is malformed. This malformed pro-MPO seems to be sequestered by calnexin (a molecular chaperone) and retained in the endoplasmic reticulum. The trapped precursor then undergoes degradation in the endoplasmic reticulum. Pro-MPO is prevented from entering the secretory pathway and cannot proceed to become mature MPO in the azurophilic granules. Therefore, MPO deficiency resulting from this mutation occurs because of an abnormality of protein folding. Interestingly, abnormalities in protein folding have also been described in cystic fibrosis and protein C deficiency.
 - M251T: In this defect, mature subunits are formed, but their enzymatic activity is markedly low.
 - G501S: This mutation is a missense mutation within part of the heme-binding pocket. It has been identified in a Japanese patient with complete MPO deficiency.¹¹
 - R499C: This mutation is a nonsynonymous mutation that results in an arginine to cysteine substitution in the exon 9 coding region. The mutation was identified in a Japanese patient with complete MPO deficiency. Further genetic analysis revealed mRNA was transcribed into an appropriate peptide sequence, but no MPO protein was evident on Western blot findings.¹²
- As time goes on and more cases are analyzed, more mutations are being identified. Some pretranslational defects have been described that could be caused by mutations in the regulatory portion of the *MPO* gene or by the presence of mutations in other genes involved in the regulation of the *MPO* gene.
- Acquired MPO deficiency is less common than the hereditary form. This condition can be transient. The enzyme defect is corrected when the underlying condition has resolved. In most cases of acquired deficiency, the deficiency is partial and affects only a proportion of neutrophils (see Pathophysiology).

Differential Diagnoses

Chronic Granulomatous Disease

Glycogen-Storage Disease Type I

Hyperimmunoglobulinemia E (Job) Syndrome

Kostmann Disease
Leukocyte Adhesion Deficiency
Shwachman-Diamond Syndrome

Other Problems to Be Considered

Neutropenia (of any cause)
Chediak-Higashi syndrome
Neutrophil actin dysfunction
Myelokathexis
Specific granule deficiency
Lazy leukocyte syndrome
Any of the conditions that can cause acquired (secondary) myeloperoxidase (MPO) deficiency

Workup

Laboratory Studies

- The presence of myeloperoxidase (MPO) can be determined using numerous techniques, including histochemical staining, immunocytochemistry, and flow cytometry. Depending on the assay used, one must ensure that eosinophilic peroxidase (EPO) from eosinophils does not cause false-positive results.
- The easiest technique is to perform direct visualization of neutrophils on a peripheral blood smear that has been stained for peroxidase. The clinician can ask the pathologist to examine the neutrophils for peroxidase when a peripheral smear is requested.¹³
- Dihydrorhodamine 123 (DHR) assay, a flow cytometric assay, is often used to measure the presence of reactive oxygen intermediates in the work-up of a patient with suspected immunodeficiency. This assay is easier, more reliable, and more sensitive than nitroblue tetrazolium dye reduction assay in the diagnosis of chronic granulomatous disease (CGD). At this time, a DHR assay should not be used as a screen for MPO deficiency because of variable results and poor sensitivity in detecting partial MPO deficiency. If a DHR assay is consistent with a diagnosis of CGD but the clinical history is more consistent with MPO deficiency, further laboratory testing should be performed (eg, genetic sequencing or intracellular staining with anti-MPO antibody).¹⁴

Treatment

Medical Care

In general, routine treatment with prophylactic antibiotics is not recommended because most patients with myeloperoxidase (MPO) deficiency have no increased incidence of infections.

- Exercise caution in patients with concomitant diabetes mellitus. If infection does occur, initiate prompt and aggressive treatment with antimicrobials. Every effort should be made to identify causative agents and administer specific antimicrobial therapy.
- If possible, avoid any treatments that might increase the likelihood of developing fungal infection (eg, use of broad-spectrum antibiotics, prolonged courses of antibiotics).

Medication

See Medical Care.

Follow-up

Inpatient & Outpatient Medications

- See Medical Care.

Prognosis

- A group from Europe who studied patients with complete myeloperoxidase (MPO) deficiency found that about half had infectious complications, while the other half were asymptomatic. Infectious complications were life threatening in about 10% of cases.
- Others have reported severe infections occurring in fewer than 5% of patients with MPO deficiency (this frequency may be based on the inclusion of both complete and partial deficiencies). Infections generally occur only in patients who have concomitant diabetes mellitus.

Miscellaneous

Medicolegal Pitfalls

- Because most patients with myeloperoxidase (MPO) deficiency do not have serious or life-threatening infections, failure to diagnose MPO deficiency may have no adverse consequences. Indeed, because at least half of patients with MPO deficiency are asymptomatic, most cases are undiagnosed. However, failure to make the diagnosis in a patient with MPO deficiency with recurrent serious infections could lead to medicolegal consequences.
- If an infectious disease occurs in a patient with MPO deficiency who also has diabetes mellitus, it is usually a fungal infection (particularly candidal species such as *C albicans* or *C tropicalis*). Patients without diabetes mellitus rarely have problems. Consider the possibility of MPO deficiency in a case of invasive fungal infection in a patient with no known predisposing immune defects (eg, chemotherapy, corticosteroid treatment) or in a patient with concomitant diabetes mellitus.

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Keywords

myeloperoxidase deficiency, MPO, MPO deficiency, lead toxicity, iron deficiency, diabetes mellitus, renal transplantation, acute myeloid leukemia, AML, chronic myeloid leukemia, CML, polycythemia vera, Hodgkin disease, Hodgkin's disease, refractory megaloblastic anemia, aplastic anemia, myelofibrosis with myeloid metaplasia, myelodysplastic syndrome, *Staphylococcus aureus*, *Serratia*, *Escherichia coli*, *Candida albicans*, *Candida krusei*, *Candida stellatoidea*, *Candida tropicalis*, atherosclerosis

Contributor Information and Disclosures

Author

Maureen M Petersen, MD, Fellow in Allergy and Immunology, Walter Reed Army Medical Center
Maureen M Petersen, MD is a member of the following medical societies: American Academy of Allergy Asthma and Immunology, American Academy of Pediatrics, American College of Allergy, Asthma and Immunology, American Thoracic Society, and Clinical Immunology Society
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Coauthor

Cecilia P Mikita, MD, MPH, Assistant Professor of Pediatrics and Medicine, Uniformed Services University of the Health Sciences; Associate Program Director of Allergy-Immunology Fellowship, Chief of Clinical Services, Staff Allergist/Immunologist, Walter Reed Army Medical Center
Cecilia P Mikita, MD, MPH is a member of the following medical societies: American Academy of Allergy Asthma and Immunology, American Academy of Pediatrics, American College of Allergy, Asthma and Immunology, and Clinical Immunology Society
Disclosure: Nothing to disclose

Javed Sheikh, MD, Assistant Professor of Medicine, Harvard Medical School; Clinical Director, Division of Allergy and Inflammation, Beth Israel Deaconess Medical Center; Clinical Director, Center for Eosinophilic Disorders, Beth Israel Deaconess Medical Center
Javed Sheikh, MD is a member of the following medical societies: American Academy of Allergy Asthma and Immunology and American College of Allergy, Asthma and Immunology
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Medical Editor

C Lucy Park, MD, Director, Allergy and Asthma Center, Associate Professor, Department of Pediatrics, University of Illinois at Chicago
C Lucy Park, MD is a member of the following medical societies: American Academy of Allergy Asthma and Immunology, American Academy of Pediatrics, American Medical Association, Clinical Immunology Society, and Illinois State Medical Society
Disclosure: Nothing to disclose

Pharmacy Editor

Mary L Windle, PharmD, Adjunct Assistant Professor, University of Nebraska Medical Center College of Pharmacy, Pharmacy Editor, eMedicine
Disclosure: Pfizer Inc Stock for Investment from broker recommendation; Avanir Pharma Stock for Investment from broker recommendation

Managing Editor

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David J Valacer, MD is a member of the following medical societies: American Academy of Allergy Asthma and Immunology, American Academy of Pediatrics, American Association for the Advancement of Science, American Thoracic Society, and New York Academy of Sciences
Disclosure: Nothing to disclose

CME Editor

David Pallares, MD, Clinical Assistant Professor, Department of Pediatrics, Division of Allergy and Immunology, University of Louisville
David Pallares, MD is a member of the following medical societies: American Academy of Allergy

Asthma and Immunology

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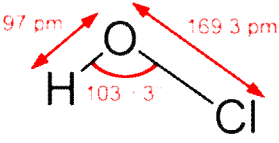
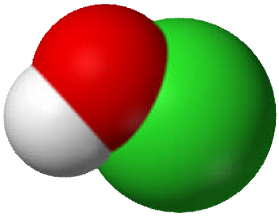
Chief Editor

Harumi Jyonouchi, MD, Associate Professor, Division of Pulmonary Allergy/Immunology and Infectious Diseases, Department of Pediatrics, UMDNJ-New Jersey Medical School

Harumi Jyonouchi, MD is a member of the following medical societies: American Academy of Allergy Asthma and Immunology, American Academy of Pediatrics, American Association of Immunologists, American Medical Association, Clinical Immunology Society, New York Academy of Sciences, Society for Experimental Biology and Medicine, Society for Mucosal Immunology, and Society for Pediatric Research

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Hypochlorous acid

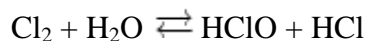
Hypochlorous acid	
	
	
IUPAC name	
Other names	hydrogen hypochlorite hydrogen chlorate(I)
Identifiers	
CAS number	7790-92-3
EC number	232-232-5
SMILES	
Properties	
Molecular formula	HClO

Molar mass	52.46 g/mol
Appearance	colorless aqueous solns
Density	variable
Solubility in water	soluble
Acidity (pK _a)	7.497 ^[1]
Hazards	
Main hazards	oxidizer
Related compounds	
Related compounds	Chlorine Calcium hypochlorite Sodium hypochlorite
Except where noted otherwise, data are given for materials in their standard state (at 25 °C, 100 kPa) Infobox references	

Hypochlorous acid is a weak [acid](#) with the [chemical formula](#) HClO. It forms when [chlorine](#) dissolves in water. It cannot be isolated in pure form due to rapid equilibration with its precursor (see below). HClO is used as a [bleach](#), an [oxidizer](#), a [deodorant](#), and a [disinfectant](#).

Formation

Addition of [chlorine](#) to [water](#) gives both hydrochloric acid (HCl) and **hypochlorous acid**^[2]:



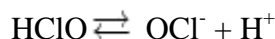
Uses

In [organic synthesis](#), HClO converts [alkenes](#) to [chlorohydrins](#).^[3]

In [biology](#), hypochlorous acid is generated in activated [neutrophils](#) by myeloperoxidase-mediated peroxidation of chloride ions, and contributes to the destruction of [bacteria](#) and this is used in water treatment such as the acid being the active sanitizer in hypochlorite-based swimming pool products.

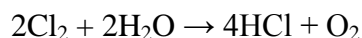
Chemical reactions

In [aqueous](#) solution, hypochlorous acid partially dissociates into the anion *hypochlorite* ClO⁻:



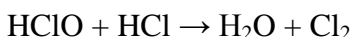
[Salts](#) of hypochlorous acid are also called **hypochlorites**. One of the best-known hypochlorites is [NaOCl](#), the active ingredient in bleach.

In the presence of sunlight, hypochlorous acid decomposes into [hydrochloric acid](#) and [oxygen](#), so this reaction is sometimes seen as:



HClO is considered to be a stronger oxidant than chlorine.

HClO reacts with HCl to form chlorine gas:



Reactivity of HClO with biomolecules

Hypochlorous acid reacts with a wide variety of biomolecules including DNA, RNA,^{[6][7][8][9]} fatty acid groups, cholesterol^{[10][11][12][13][14][15][16][17]} and proteins.^{[2][18][13][19][20][21][22]}

Reaction with protein sulfhydryl groups

Knox et al.^[20] first noted that HClO is a [sulfhydryl](#) inhibitor that, in sufficient quantity, could completely inactivate proteins containing [sulfhydryl groups](#). This is because HClO oxidises [sulfhydryl groups](#), leading to the formation of [disulfide bonds](#)^[23] that can result in crosslinking of [proteins](#). The HClO mechanism of [sulfhydryl](#) oxidation is similar to that of [chloramine](#), and may only be bacteriostatic, because, once the residual chlorine is dissipated, some [sulfhydryl](#) function can be restored.^[19] One [sulfhydryl](#)-containing amino acid can scavenge up to four molecules of HOCl.^[22] Consistent with this, it has been

proposed that [sulfhydryl groups](#) of sulfur-containing [amino acids](#) can be oxidized a total of three times by three HClO molecules, with the fourth reacting with the α -amino group. The first reaction yields [sulfenic acid](#) (R-SOH) then [sulfinic acid](#) (R-SO₂H) and finally R-SO₃H. Each of those intermediates can also condense with another [sulfhydryl group](#), causing cross-linking and aggregation of proteins. [Sulfinic acid](#) and R-SO₃H derivatives are produced only at high molar excesses of HClO, and disulfides are formed primarily at bacteriocidal levels.^[9] Disulfide bonds can also be oxidized by HClO to sulfinic acid.^[23] Because the oxidation of [sulfhydryls](#) and [disulfides](#) evolves [hydrochloric acid](#),^[9] this process results in the depletion HClO.

Reaction with protein amino groups

Hypochlorous acid reacts readily with amino acids that have [amino group](#) side-chains, with the chlorine from HClO displacing a hydrogen, resulting in an organic chloramine.^[24] Chlorinated [amino acids](#) rapidly decompose, but [protein](#) chloramines are longer-lived and retain some oxidative capacity.^{[22][5]} Thomas et al.^[5] concluded from their results that most organic chloramines decayed by internal rearrangement and that fewer available [NH₂](#) groups promoted attack on the [peptide bond](#), resulting in cleavage of the [protein](#). McKenna and Davies^[25] found that 10 mM or greater HClO is necessary to fragment proteins in vivo. Consistent with these results, it was later proposed that the chloramine undergoes a molecular rearrangement, releasing [HCl](#) and [ammonia](#) to form an [amide](#).^[26] The [amide group](#) can further react with another [amino group](#) to form a [Schiff base](#), causing cross-linking and aggregation of proteins.^[13]

Reaction with DNA and Nucleotides

Hypochlorous acid reacts slowly with DNA and RNA as well as all nucleotides in vitro.^{[7][27]} [GMP](#) is the most reactive because HClO reacts with both the heterocyclic NH group and the amino group. In similar manner, [TMP](#) with only a heterocyclic NH group that is reactive with HClO is the second-most reactive. [AMP](#) and [CMP](#), which have only a slowly reactive amino group are less reactive with HClO.^[27] [UMP](#) has been reported to be reactive only at a very slow rate.^{[6][7]} The heterocyclic NH groups are more reactive than amino groups, and their secondary chloramines are able to donate the chlorine.^[9] These reactions likely interfere with DNA base pairing, and, consistent with this, Prütz^[27] has reported a decrease in viscosity of DNA exposed to HClO similar to that seen with heat denaturation. The sugar moieties are unreactive and the DNA backbone is not broken.^[27] NADH can react with chlorinated TMP and UMP as well as HClO. This reaction can regenerate UMP and TMP and results in the 5-hydroxy derivative of NADH. The reaction with TMP or UMP is slowly reversible to regenerate HClO. A second slower reaction that results in cleavage of the pyridine ring occurs when excess HClO is present. NAD⁺ is inert to HClO.^{[27][9]}

Reaction with lipids

Hypochlorous acid reacts with [unsaturated bonds](#) in [lipids](#), but not [saturated bonds](#), and the [OCl⁻](#) ion does not participate in this reaction. This reaction occurs by [hydrolysis](#) with

addition of [chlorine](#) to one of the carbons and a [hydroxyl](#) to the other. The resulting compound is a chlorhydrin.^[10] The polar [chlorine](#) disrupts [lipid bilayers](#) and could increase permeability.^[11] When chlorhydrin formation occurs in [lipid bilayers](#) of red blood cells, increased permeability occurs. Disruption could occur if enough chlorhydrin is formed.^{[10][16]} The addition of preformed chlorhydrins to [red blood cells](#) can affect permeability as well.^[12] [Cholesterol](#) chlorhydrins have also been observed,^{[11][14]} but do not greatly affect permeability, and it is believed that [Cl₂](#) is responsible for this reaction.^[14]

Mode of disinfectant action

[Escherichia coli](#) exposed to hypochlorous acid lose [viability](#) in less than 100 ms due to inactivation of many vital systems.^{[2][28][29][30][31]} Hypochlorous acid has a reported [LD₅₀](#) of 0.0104 ppm - 0.156 ppm^[32] and 2.6 ppm caused 100% growth inhibition in 5 minutes.^[25] However it should be noted that the concentration required for bactericidal activity is also highly dependent on bacterial concentration.^[20]

Inhibition of glucose oxidation

In 1948, Knox et al.^[20] proposed the idea that inhibition of [glucose](#) oxidation is a major factor in the bacteriocidal nature of chlorine solutions. He proposed that the active agent or agents diffuse across the cytoplasmic membrane to inactivate key [sulfhydryl](#)-containing [enzymes](#) in the [glycolytic pathway](#). This group was also the first to note that chlorine solutions (HOCl) inhibit [sulfhydryl enzymes](#). Later studies have shown that, at bacteriocidal levels, the [cytosol](#) components do not react with HOCl.^[1] In agreement with this, McFeters and Camper^[33] found that [aldolase](#), an [enzyme](#) that Knox et al.^[20] proposes would be inactivated, was unaffected by HOCl *in vivo*. It has been further shown that loss of [sulfhydryls](#) does not correlate with inactivation.^[19] That leaves the question concerning what causes inhibition of [glucose](#) oxidation. The discovery that HOCl blocks induction of [β-galactosidase](#) by added [lactose](#)^[34] led to a possible answer to this question. The uptake of radiolabeled substrates by both ATP hydrolysis and proton co-transport may be blocked by exposure to HOCl preceding loss of viability.^[1] From this observation, it proposed that HOCl blocks uptake of nutrients by inactivating transport proteins.^{[1][18][35][33]} The question of loss of glucose oxidation has been further explored in terms of loss of respiration. Venkobachar et al.^[36] found that succinic dehydrogenase was inhibited *in vitro* by HOCl, which led to the investigation of the possibility that disruption of [electron transport](#) could be the cause of bacterial inactivation. Albrich et al.^[6] subsequently found that HOCl destroys [cytochromes](#) and [iron-sulfur clusters](#) and observed that oxygen uptake is abolished by HOCl and adenine nucleotides are lost. Also observed was, that irreversible oxidation of [cytochromes](#) paralleled the loss of respiratory activity. One way of addressing the loss of oxygen uptake was by studying the effects of HOCl on succinate dependent [electron transport](#).^[37] Rosen et al.^[31] found that levels of reductable [cytochromes](#) in HOCl-treated cells were normal, and these cells were unable to reduce them. Succinate dehydrogenase was also inhibited by HOCl, stopping the flow of electrons to oxygen. Later studies^[29] revealed that Ubiquinol oxidase activity ceases first, and the still-active [cytochromes](#) reduce the remaining quinone. The [cytochromes](#)

then pass the [electrons](#) to [oxygen](#), which explains why the [cytochromes](#) cannot be reoxidized, as observed by Rosen et al.^[31] However, this line of inquiry was ended when Albrich et al.^[2] found that cellular inactivation precedes loss of respiration by using a flow mixing system that allowed evaluation of viability on much smaller time scales.

This group found that cells capable of respiring could not divide after exposure to HOCl.

Depletion of adenine nucleotides

Having eliminated loss of respiration Albrich et al.^[2] proposes that the cause of death may be due to metabolic dysfunction caused by depletion of adenine nucleotides. Barrette et al.^[34] studied the loss of adenine nucleotides by studying the energy charge of HOCl-exposed cells and found that cells exposed to HOCl were unable to step up their energy charge after addition of nutrients. The conclusion was that exposed cells have lost the ability to regulate their adenylate pool, based on the fact that metabolite uptake was only 45% deficient after exposure to HOCl and the observation that HOCl causes intracellular ATP hydrolysis. Also confirmed was that, at bacteriocidal levels of HOCl, cytosolic components are unaffected. So it was proposed that modification of some membrane-bound protein results in extensive ATP hydrolysis, and this, coupled with the cells inability to remove AMP from the cytosol, depresses metabolic function. One protein involved in loss of ability to regenerate ATP has been found to be [ATP synthetase](#).^[18] Much of this research on respiration reconfirms the observation that relevant bacteriocidal reactions take place at the cell membrane.^{[34][18][38]}

Inhibition of DNA replication

Recently it has been proposed that bacterial inactivation by HOCl is the result of inhibition of [DNA](#) replication. When bacteria are exposed to HOCl, there is a precipitous decline in [DNA synthesis](#) that precedes inhibition of [protein](#) synthesis, and closely parallels loss of viability.^{[25][39]} During bacterial genome replication, the [origin of replication](#) (oriC in *E. coli*) binds to proteins that are associated with the cell membrane, and it was observed that HOCl treatment decreases the affinity of extracted membranes for oriC, and this decreased affinity also parallels loss of viability. A study by Rosen et al.^[40] compared the rate of HOCl inhibition of DNA replication of plasmids with different replication origins and found that certain plasmids exhibited a delay in the inhibition of replication when compared to plasmids containing oriC. Rosen's group proposed that inactivation of membrane proteins involved in DNA replication are the mechanism of action of HOCl.

External links

- [National Pollutant Inventory - Chlorine](#)
- [- how bleach kills germs -esbati](#)

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