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## Water treatment chlorination: An updated mechanistic insight review

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**Abstract** Since the 1970s' disinfection by-products (DBPs) detection, the water treatment specialists' main focuses were accorded to the DBPs formation, characterization, regulations and control. Involved stages in disinfection process were at a certain level kept at the side as a black box. This paper is a broad review on chlorination applied in water treatment technology especially in terms of involved mechanisms. Chlorine occasions significant injury to bacterial cells, cell permeability dislocation and nucleic acids and enzymes injury. Hypochlorous acid oxidizes sulfhydryl groups, harms iron-sulfur centers, deactivates nutrient transport, hinders cell respiration, and deteriorates the capacity of cells to keep a sufficient adenylate energy charge to stay viable. All disinfectants are highly efficient killing agents. However, these chemical products are very toxic by their selves. Moreover, they interact with NOM, microorganisms, and algae to produce DBPs which are as well poisonous. Consequently, the use of disinfectants must be avoided upon using physical processes or at least reduced as possible at the lowest level.

**Keywords** Chlorination by-products (CBPs), Disinfection by-products (DBPs), Natural organic matter (NOM), Trihalomethanes (THMs), Membrane processes.

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### 1. Introduction

Disinfection is the demolition of microorganisms able of generating diseases [1-3]. Disinfection is an important and last fence versus human subjection to disease-producing pathogenic microorganisms, comprising viruses, bacteria, and protozoan parasites [4]. Killing microorganisms of potable water [5] is very likely the most crucial preemptive action in human history [6]. Ensuing the finding of the "germ theory" by Louis Pasteur and Robert Koch in the 1880s, chlorination was introduced at the dawn of the 20<sup>th</sup> century to give a supplementary defense opposed to pathogenic microorganisms. The earliest chlorination set-up was established in Middle kerke, Belgium, in 1902. The United States came four years later with the big-scale usage of chlorine for water killing microorganisms in Jersey City [7]. The demolition of pathogens and parasites using disinfection assisted greatly in the decrease of waterborne and food borne diseases [8]. But, in the 1970s, the discovery that chlorination may conduct to the generation of by-products that are toxic or genotoxic to both animals and humans has conducted to a search for securer killing agents [9]. It was as well proved that certain pathogens or parasites are in fact fairly resistant to killing agents and that the conventional guide microorganisms are occasionally not convenient for making sure secure water [10-14]. Moreover than their usage for pathogen and parasite demolition, several of the killing agents (like ozone and chlorine dioxide) are as well used for oxidation of organic matter, iron, and manganese, enhancing filtration



performance, and monitoring biofilm development in water delivery frameworks, and for monitoring taste and odor issues and algal development [15-22].

This review concerns the killing agents most usually utilized in the water treatment industries focusing on their implied mechanisms and disinfection secondary reactions health concerns. It as well treats the physical elimination of pathogens using membranes as a green technology.

## 2. Elements affecting killing microorganisms

Disinfecting water is monitored by numerous elements [7,23].

### 2.1. Kind of disinfectant/biocide

Biocides, comprising disinfectants, apply cidal or impeding influences by interacting with one or more objectives in microbial cells [7,24] (Fig. 1). The objective places comprise the peptidoglycan film, cytoplasmic membrane, outer membrane, structural proteins, thiol groups of enzymes, nucleic acids, viral envelopes, capsids or nucleic acids, and bacterial spore coats [24]. The efficiency of water and wastewater [25] disinfection is function of the kind of chemical product utilized. Several killing agents (such as ozone, chlorine dioxide) are more powerful oxidants than others (like chlorine) [7].

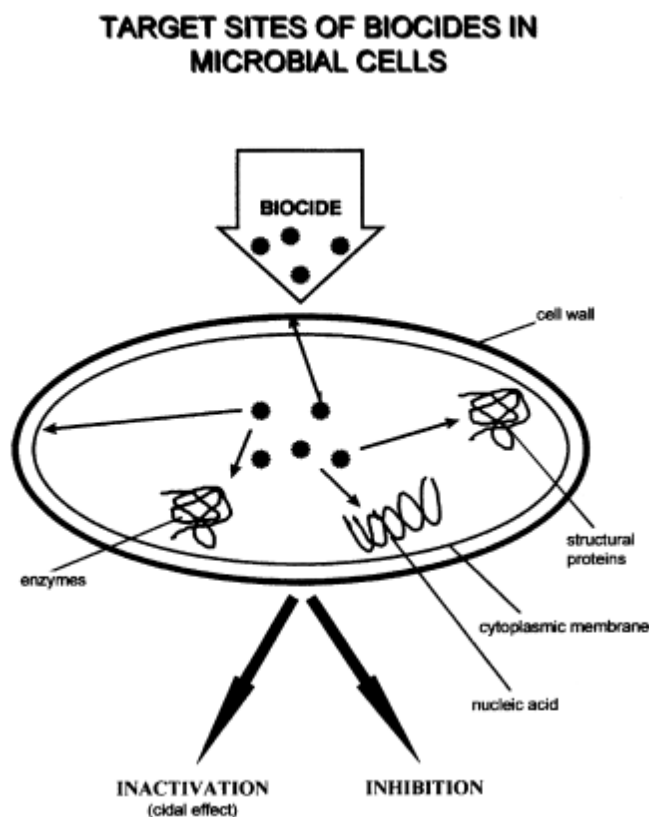


Figure 1: Objectiveplaces of biocides in microbial cells [7].

### 2.2. Kind of microorganisms

There is a large difference among the diverse microbial pathogens concerning their resistance to killing agents. Spore-forming bacteria are usually more resistant to killing agents than are vegetative bacteria. Resistance to chemical products changes as well between non-spore-forming bacteria and between strains belonging to the same species. As an illustration, *Legionella pneumophila* is much more resistant to chlorine than is *Escherichia coli*.



Broadly, resistance to disinfection plays along the next sequence: non-spore-forming bacteria < enteric viruses < spore-forming bacteria < protozoan cysts [7].

### 2.3. Disinfectant concentration and residence period

In 1897, Kronig and Paul demonstrated the reliance of disinfection on concentration and residence period. Deactivation of pathogens with killing agent's augments with time and, perfectly, must obey first-order kinetics. Deactivation versus time obeys a straight line when data are plotted on a log-log paper.

$$\frac{N_t}{N_0} = e^{-kt} \quad (1)$$

where  $N_0$  = number of microorganisms at time 0,  $N_t$  = number of microorganisms at time  $t$ ,  $k$  = decay constant ( $\text{time}^{-1}$ ), and  $t$  = time (time) [7].

Nevertheless, as shown in Fig. 2, practical deactivation facts really illustrate a divergence from first-order kinetics [26]. Curve C in Fig. 2 illustrates divergence from first-order kinetics. The tailing off of the curve follows from the survival of a resistant subpopulation inside a heterogeneous population or from safeguard of the pathogens by interfering elements (Fig. 2). Microbial clustering can interpret the "shoulder" of survival curves achieved when uncovering microorganisms to chlorine effect [7].

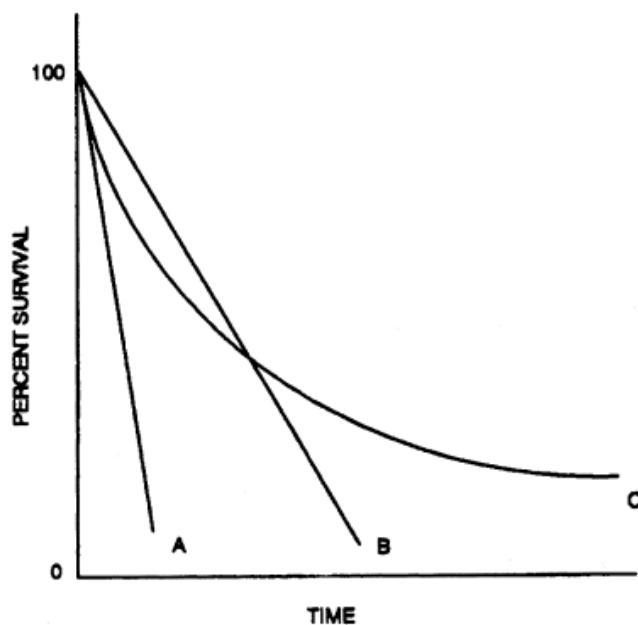


Figure 2: Inactivation curves of microorganisms following disinfection: (a) sensitive homogeneous population; (b) more resistant homogeneous population; (c) heterogeneous population or partially protected by aggregation [7].

Disinfectant performance can be revealed in the form of  $Ct$ ,  $C$  being the killing agent dose, and  $t$  the time needed to deactivate a particular percentage of the population under certain conditions (pH and temperature). The correlation among killing agent level and residence period is provided by the Watson's law [7]:

$$K = C^n t \quad (2)$$

Where  $K$  = constant for a specific microorganism offered to a killing agent under particular conditions,  $C$  = disinfectant dose (mg/L),  $t$  = time needed to destroy a specific percentage of the population (min), and  $n$  = constant as well named the "coefficient of dilution" [7].

While  $t$  is plotted against  $C$  on a double logarithmic paper,  $n$  is the slope of the straightline (see Fig. 3). The amount of  $n$  calculates the value of the killing agent dose or residence period in microorganism deactivation. If  $n < 1$ , the disinfection is more influenced by residence period than by killing agent dose. If  $n > 1$ , the disinfectant dose is the strongest element monitoring disinfection [7]. Nevertheless, the amount of  $n$  is frequently adjacent to unity.



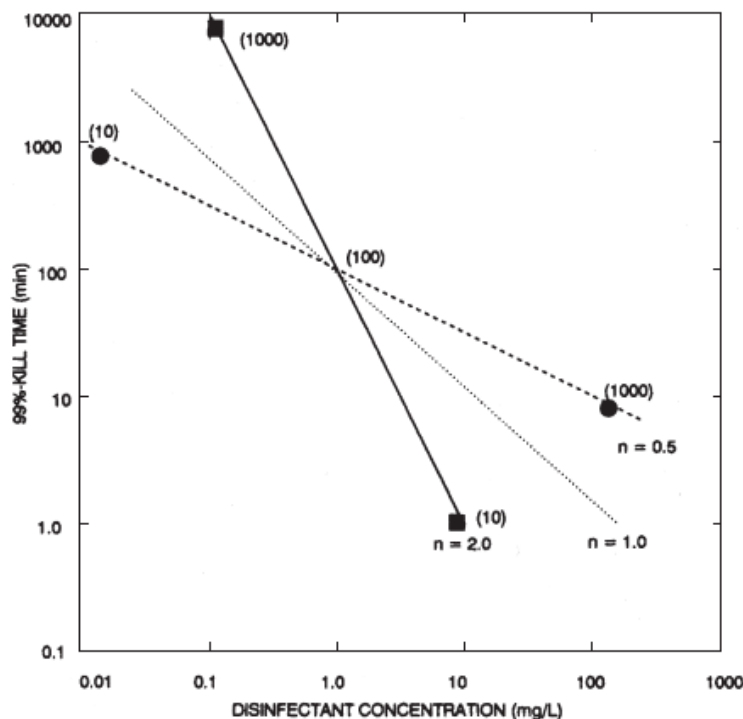


Figure 3: Influence of  $n$  amount on  $Ct$  at different killing agent doses ( $Ct$  values provided in parentheses) [7].

Determination of  $Ct$  may as well consider the temperature and pH degree of the suspending solution. As an illustration, a mathematical model was established to anticipate *Giardia lamblia* cysts deactivation after chlorine injection [7,27].

$$Ct = 0.9847C^{0.1758}pH^{2.7519}T^{-0.1467} \quad (3)$$

Where  $C$  = chlorine dose ( $C \leq 4.23$  mg/L),  $t$  = time to inactivate 99.99% of the cysts, pH interval is 6-8, and  $T$  = temperature interval is 0.5-5.0°C.

$Ct$  values for an interval of pathogens are given in Table 1 [26]. Resistance to chlorine is in the next sequence: protozoan cysts > viruses > non-spore-forming bacteria [7].

Table 1: Microbial deactivation under chlorine:  $Ct$  values (Temperature = 5°C; pH = 6.0) [26].

Chlorine Microorganism	Inactivation		
	Concentration (mg/L)	Time (min)	$Ct$
<i>Escherichia coli</i>	0.1	0.4	0.04
Poliovirus I	1.0	1.7	1.7
<i>Entamoeba histolytica</i> cysts	5.0	18	90
<i>G. lamblia</i> cysts	1.0	50	50
	2.0	40	80
	2.5	100	250
<i>Giardia muris</i> cysts		100	250

An additional manner to indicate the performance of a specific killing agent is the lethality coefficient  $\lambda$  provided by the next equation [7]:

$$\lambda = 4.6/Ct_{\infty} \quad (4)$$

where 4.6 = natural log of 100,  $C$  = remaining dose of killing agent (mg/L), and  $t_{\infty}$  = residence period (min) for 99% deactivation of microorganisms.

The values of  $\lambda$  for demolishing 99% of an interval of microorganisms by ozone in 10 min at 10-15 °C change from 5 for *E. histolytica* to 500 for *E. coli* [7].



#### 2.4. Influence of pH

Concerning killing microorganisms using chlorine, pH manages the quantity of hypochlorous acid (HOCl) and hypochlorite ion (OCl<sup>-</sup>) in solution (see Section 3.1.). HOCl is 80 times more efficient than OCl<sup>-</sup> for *E. coli*. For killing pathogens using chlorine, *Ct* augments with pH. On the contrary, bacterial, viral, and protozoan cyst inactivation by chlorine dioxide is usually more performant at more elevated pH levels [7,23,27].

#### 2.5. Temperature

Pathogen and parasite deactivation augments, in order words *Ct* decreases, as temperature augments.

#### 2.6. Effect of chemical and physical characteristics

Chemical elements interacting with killing microorganisms are inorganic and organic nitrogenous compounds, iron, manganese, and hydrogen sulfide. Dissolved organic matters as well apply a chlorine demand; their presence conducts to diminished killing microorganisms performance [7].

In raw water, turbidity is constituted of inorganic (such as silt, clay, iron oxides) and organic matter, and also microbial cells. Turbidity is evaluated by estimating light scattering by dispersed matters existing in water. Turbidity interacts with the observation of coliforms in water [7] but may as well decrease the killing microorganisms' performance of chlorine and additional killing agents [28]. The necessity to eliminate turbidity is founded on the certainty that particle-linked microorganisms are more resistant than freely suspended microorganisms to disinfection. The total organic carbon (TOC) related with colloids applies a chlorine demand and therefore interacts with the preservation of a chlorine remaining in water. Microorganisms linked with fecal material, cell debris, or wastewater solid particles are also kept safe from killing. These discoveries are extremely crucial for cities that treat their water only by chlorination. Fig. 4 shows the shielding influence of turbidity for coliform bacteria [7]. It was as well illustrated that the shielding contribution of solid particles in water and wastewater is function of the type and the dimension of the particles. Therefore, cell-linked poliovirus is kept safe from chlorine deactivation, while bentonite or aluminum phosphate does not provide similar preservation to the virus. Viruses and bacterial guides are not safeguarded against ozone deactivation by bentonite. It was observed that solid-linked viruses upon field conditions are more resistant to chlorination than are "free" viruses. Decreasing turbidity to less than 0.1 nephelometric turbidity units (NTU) may be a precautionary action for thwarting the preemptive contribution of solid matter during disinfection [7].

### 3. Chlorine

Until now, chlorine is the most largely utilized killing agent in water and wastewater treatment factories. It is utilized as gas, hypochlorite solution, or onsite-produced hypochlorite. In 2007, it was observed that ~64% of water treatment factories employ chlorine gas [7].

#### 3.1. Chlorine chemistry

Chlorine gas (Cl<sub>2</sub>) added to water hydrolyzes following the next equation:



Chlorine Hypochlorous

gas acid

Hypochlorous acid dissociates in water following the next equation:



Hypochlorous Hypochlorite

acid ion

Fig. 4 illustrates that the fractions of HOCl and OCl<sup>-</sup> are function of the pH of the water. Chlorine, like HOCl or OCl<sup>-</sup> is defined as free available chlorine. HOCl combines with ammonia and organic nitrogen compounds to form chloramines, which are combined available chlorine [7].



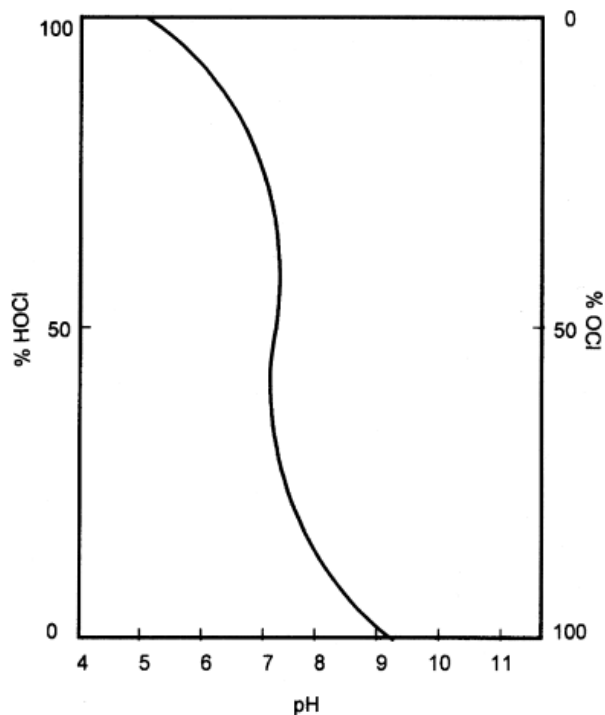


Figure 4: Distribution of HOCl and OCl<sup>-</sup> in water as a function of pH [7].

### 3.2. Chlorine's microorganisms deactivation

Of the three chlorine species (HOCl, OCl<sup>-</sup>, and NH<sub>2</sub>Cl), hypochlorous acid is the most efficient for the deactivation of pathogens in water and wastewater. The existence of interacting compounds in water decreases the killing microorganisms' performance of chlorine; comparatively, elevated doses of chlorine (20-40 mg/L) are needed for acceptable decrease of viruses. In wastewater sewages, no free chlorine species are accessible after a few seconds of residence [7].

Chlorine, particularly HOCl, is usually completely performant in deactivating pathogenic and indicator bacteria. Water treatment with  $\leq 1$  mg/L for  $\sim 30$  min is usually performant in importantly decreasing bacterial numbers. As an illustration, *Campylobacter jejuni* shows more than 99% deactivation in the existence of 0.1 mg/L free chlorine (residence period = 5 min) [7]. In spite of the fact that there is large change in the resistance of enteric viruses to chlorine, enteric viruses are usually more resistant to chlorine than are vegetative bacteria. That demonstrates why viruses are often observed in chlorinated secondarily treated sewages. Chloramines are much less performant than is free residual chlorine ( $\sim 50$  times less performant) regarding viral deactivation. Protozoan cysts (such as *G. lamblia*, *E. histolytica*, *Naegleria gruberi*) are more resistant to chlorine than are both bacteria and viruses. In the existence of HOCl at pH 6, the *Ct* value for *E. coli* is 0.04 in comparison with a *Ct* value of 1.05 for poliovirus type 1 and a *Ct* value of 80 for *G. lamblia*.

*Cryptosporidium* oocysts are very resistant to killing microorganisms; however, they may be deactivated by ammonia at levels detected in natural conditions. A chlorine or monochloramine dose of 80 mg/L is required to produce 90% deactivation after 90-min residence period. This parasite is not totally deactivated in a 3% solution of sodium hypochlorite, and the oocysts may continue to exist during 3-4 months in 2.5% potassium dichromate solution. The *Ct* value for *Cryptosporidium* is in the 1000s, as illustrated in Fig. 5 [7]. Therefore, this parasite would be very resistant to disinfection as achieved in water and wastewater treatment factories [7].



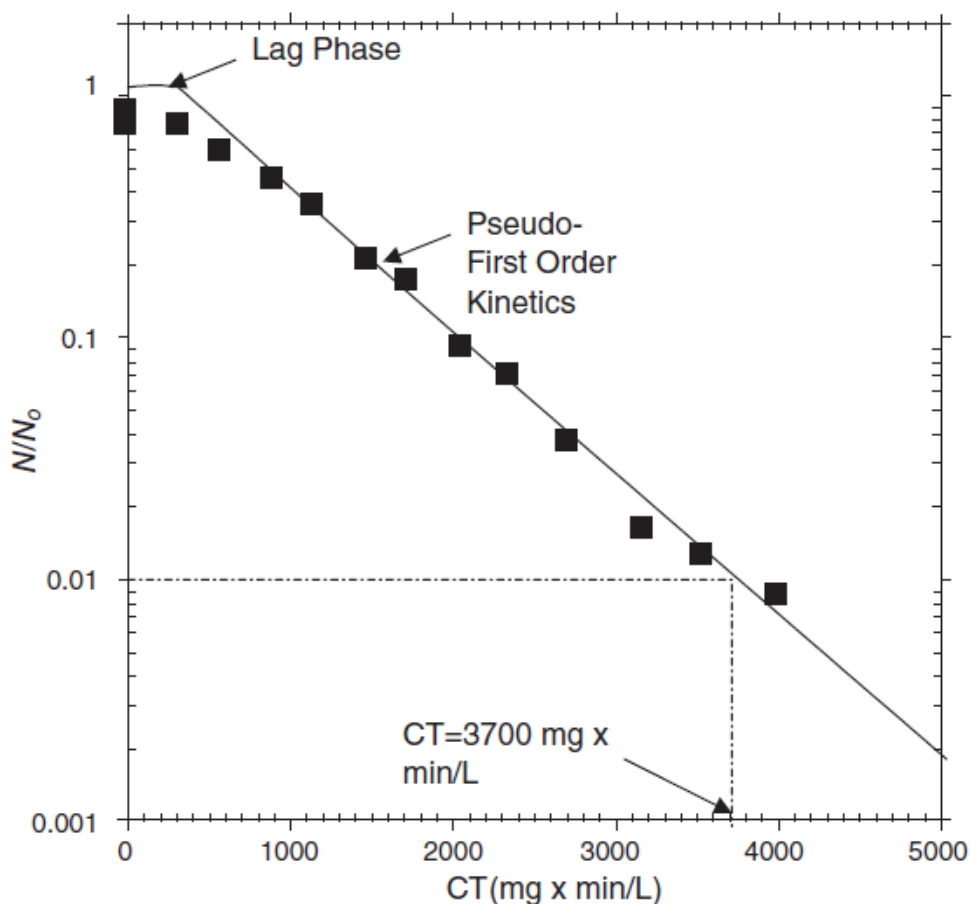


Figure 5: Primary deactivation of *Cryptosporidium parvum* oocysts upon free chlorine at pH 6 and 20°C [7].

### 3.3. Cell damage under chlorine

Physical means (such as heat, freezing, sunlight) and chemical products (chlorine, sub-lethal doses of heavy metals) may produce damage to bacterial cells. Damage generated by ecological elements may conduct to cell size decrease, harm to cell membranes, and also modified cell physiology and virulence [7].

Chlorine and copper seem to generate important damage to coliform bacteria in potable water. The damaged bacteria miss to develop in the existence of eclectic elements (such as sodium lauryl sulfate, sodium deoxycholate) conventionally integrated in growth media conceived for the isolation of indicator and pathogenic bacteria. Nevertheless, chlorine- and copper- damaged microorganisms (like enterotoxigenic *E. coli* [29]) keep their capacity for enterotoxin generation and are capable to recuperate in the small intestine of animals, maintaining their pathogenicity [7].

This discovery proposes that cells damaged upon chlorine application remain possess a possible health importance. Damage under chlorine may touch a large range of pathogens, comprising enterotoxigenic *E. coli*, *Salmonella typhimurium*, *Yersinia enterocolitica* and *Shigella* spp. The amplitude of damage upon chlorine is function of the kind of pathogen implicated [7].

### 3.4. Free chlorine's cidal effect enhancement

The deadly activity of free chlorine may be increased by introducing salts such as KCl, NaCl, or CsCl into solution. Following chlorination, viruses are more efficiently deactivated in potable water (like Cincinnati potable water) than in purified water (Fig. 6). The mechanism of the enhancement influence of salts is not totally comprehended [7].



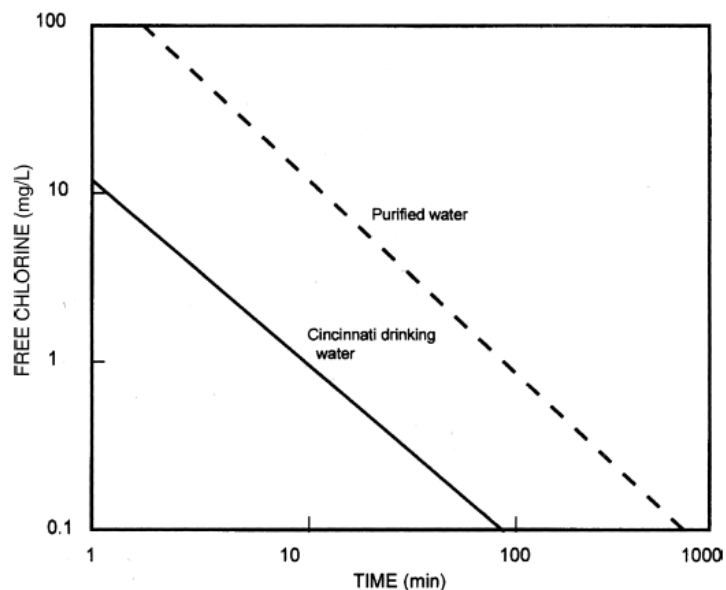


Figure 6: Relative rates of deactivation of 99.99% of poliovirus type 1 at 5°C under free chlorine at pH 9.0 in purified water and in Cincinnati drinking water [7].

The killing microorganisms' capacity of chlorine may well be improved in the existence of heavy metals. The deactivation rate of pathogenic (like *L. pneumophila*) bacteria and viruses (such as poliovirus) is augmented when free chlorine is ameliorated with electrolytically formed copper and silver (400 and 40 mg/L, respectively) [7] (Fig. 7). This process was as well established for indicator bacteria and *Naegleria fowleri* amoeba in water. This phenomenon does not, nevertheless, totally remove enteric viruses of medical significance like hepatitis A virus or human rotavirus [7]. Acclimation of *S. typhimurium* to acid as well improves its responsiveness to hypochlorous acid; however, the actual implementation of this discovery to water and waste water treatment persists uncertain.

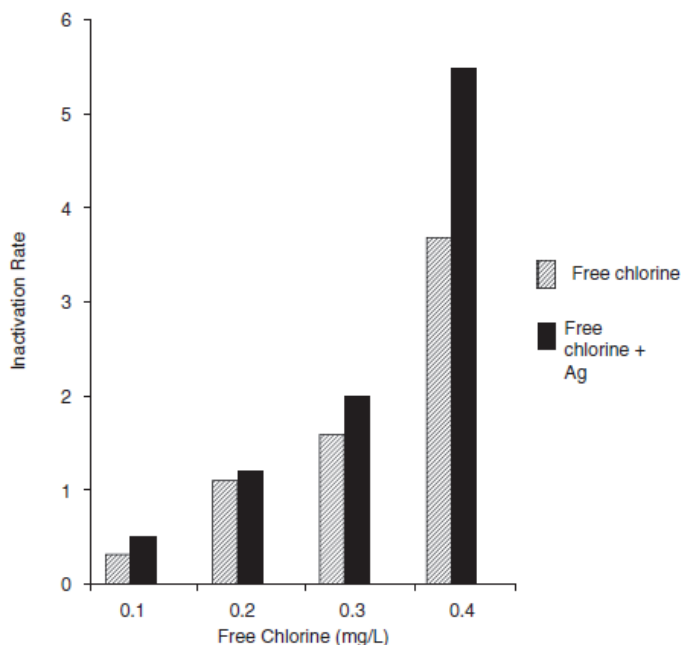


Figure 7: Deactivation of *L. pneumophila* by application to electrolytically formed copper and silver and/or different concentrations of free chlorine [7].





Deactivation of *Cryptosporidium* oocysts upon free chlorine may as well be improved using ozone pre-treatment. A 4- to 6- fold augmentation in free chlorine efficiency was achieved with ozone pre-treatment. The successive deactivation employing ozone accompanied by free chlorine was illustrated to augment the deactivation of *Salmonella*, bacterial phage, poliovirus type 1, and parasites such as *Cryptosporidium* and *Giardia* [7].

### 3.5. Chlorine effect mechanisms

Chlorine occasions significant injury to bacterial cells.

#### 3.5.1. Cell permeability dislocation

Free chlorine damages the safety of the bacterial cell envelope, therefore conducting to mislaying of cell permeability and to the dislocation of other cell roles [7]. Subjection to chlorine conducts to an escape of proteins, ribonucleic acid (RNA) [30], and deoxyribonucleic acid (DNA) [31]. Cell demise is the consequence of freeing of TOC and UV-absorbing materials, reduction in potassium intake, and decrease in protein and DNA fabrication. Permeability dislocation was as well involved as the reason of injury of chlorine to bacterial spores.

#### 3.5.2. Nucleic acids and enzymes injury

Chlorine as well harms bacterial nucleic acids and enzymes (like catalase, dehydrogenases). As a result of decreased catalase action is hindering by the cumulative hydrogen peroxide. Complete genome analysis of *Staphylococcus aureus* uncovered to HOCl has indicated that the killing agent conducts to inhibition of the transcription of genes monitoring cell membrane synthesis, protein synthesis, membrane transport, and primary metabolism. Nevertheless, HOCl causes genes encoding for virulence factors in *S. aureus* [7].

#### 3.5.3. Additional influences

Hypochlorous acid oxidizes sulfhydryl groups, harms iron-sulfur centers, deactivates nutrient transport, hinders cell respiration, and deteriorates the capacity of cells to keep a sufficient adenylate energy charge to stay viable [7].

#### 3.5.4. Influences on viruses

The manner of operation of chlorine on viruses can be a function of the kind of viruses. Nucleic acid deterioration is the first manner of deactivation for bacterial phage f2 or poliovirus type 1. The protein coat seems to be the objective place for other kinds of viruses (like rotaviruses) [7].

### 3.6. Chlorine and chlorination by-products (CBPs) toxicology

In 1974, Bellaret al. [32], in the United States, and Rook [33,34], in the Netherlands, earliest discovered four trihalomethanes (THMs) in water succeeding chlorination: chloroform, dichlorobromomethane, monochlorodibromomethane, and bromoform [7,35].

#### 3.6.1. CBPs formation

CBPs are produced succeeding the reaction of chlorine with precursors such as natural organic matter (NOM), mostly humic and fulvic acids [36-38], and microorganisms like algal cells (such as diatoms) [36] as well as cyanobacteria and their extracellular products. There is an acceptable correlation among THM formation potential (THMFP) and TOC in water (Fig. 8). THMs comprise chloroform ( $\text{CHCl}_3$ ), bromodichloromethane ( $\text{CHBrCl}_2$ ), dibromochloromethane ( $\text{CHBr}_2\text{Cl}$ ), bromoform ( $\text{CHBr}_3$ ), haloacetic acids (HAAs) (like monochloroacetic acid, monobromoacetic acid, dichloroacetic acid, dibromoacetic acid, trichloroacetic acid), and halocetonitriles. There are nine HAA species, even if only five are currently regulated [7].

#### 3.6.2. CBPs health consequences

The toxicology of chlorine and its by-products is obviously crucial because it is evaluated that 79% of the U.S. population is uncovered to chlorine. There is proof of a combination among chlorination of potable water and augmented hazard of bladder, kidney, and colorectal cancers. This combination is more powerful for consumers who have been uncovered to chlorinated water for more than 15 years [7]. However, touching bladder cancer, human epidemiological surveys have illustrated a quantifiable influence of disinfection by-products (DBPs) [39-40].



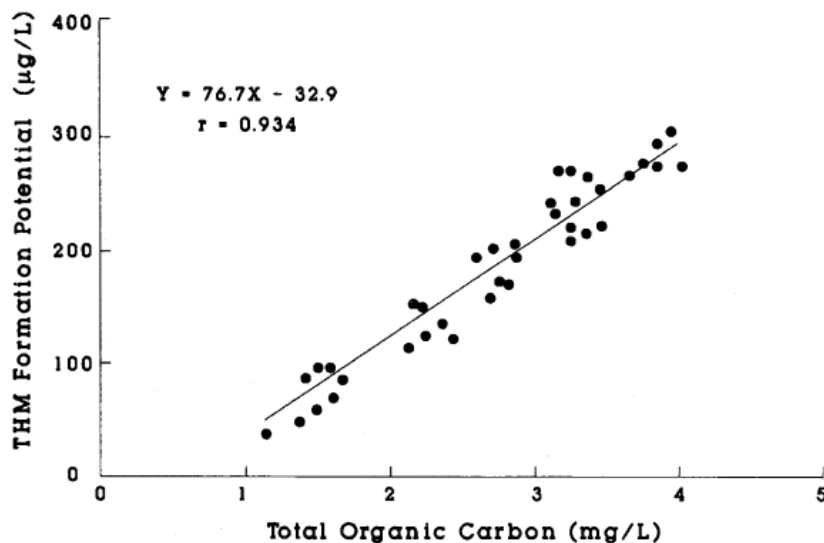


Figure 8: Correlation among THM formation potential (THMFP) and total organic carbon (TOC) [7].

### 3.6.3. Monitoring CBPs

Several proposed manners for decreasing or monitoring CBPs (if chlorination is used as disinfection process) or DBPs (if other disinfectants than chlorine are used) in water and wastewater are mentioned below:

#### a) Reducing CBPs precursors before chlorination

Organic matters (mainly NOM, comprising humic substances [38,41,42], and algae and their extracellular products [43]) levels may be decreased by some processes such as enhanced coagulation [21,44-49] granular activated carbon, or membrane filtration [50]. An integration of coagulation [51,52], ozonation [53], and biofiltration may efficiently decrease THMs and HAAs formation potential. NOM, the major precursor of DBPs, may as well be decreased by iron oxide-coated filtration media, water softening, or nanofiltration. Even if pre-chlorination augments the capacity for THM generation in water treatment factories, it does not appear to possess an important effect on HAAs production [7].

#### b) CBPs elimination in the water treatment factory

HAAs and THMs and chlorinated furanones concentration may as well be decreased upon adsorption on activated carbon and biodegradation or by biodegradation in sand filters and biofilms in water distribution pipes. THMs may as well be biodegraded via co-metabolism by mixed nitrifier cultures. In general, an increase in the number of halogen atoms decreases biodegradation but enhances adsorption [7].

## 4. Conclusion

The main important points drawn from this review may be listed as:

- (1). In a general manner, disinfectants apply cidal or impeding actions by interacting with one or more objectives in microbial cells: the peptidoglycan film, cytoplasmic membrane, outer membrane, structural proteins, thiol groups of enzymes, nucleic acids, viral envelopes, capsids or nucleic acids, and bacterial spore coats.
- (2). Chlorine occasions significant injury to bacterial cells, cell permeability dislocation and nucleic acids and enzymes injury. Hypochlorous acid oxidizes sulfhydryl groups, harms iron-sulfur centers, deactivates nutrient transport, hinders cell respiration, and deteriorates the capacity of cells to keep a sufficient adenylate energy charge to stay viable.
- (3). All disinfectants (chlorine, chloramine, ozone, UV, etc.) are very efficient in killing microorganisms; but, these killing agents are highly toxic by their selves. Moreover, they interact with NOM, microorganisms, and algae to produce DBPs which are as well poisonous. Consequently, the use of disinfectants must be avoided or at least reduced as possible at the lowest level.



### Acknowledgement

The author wishes to acknowledge G. Bitton [7]; his very excellent book was widely cited in this review paper.

### Abbreviations

$C$	Disinfectant (chlorine) dose (mg/L)
$C$	Remaining dose of killing agent (mg/L)
CBPs	Chlorination by-products
$\text{CHBrCl}_2$	Bromodichloromethane
$\text{CHBr}_2\text{Cl}$	Dibromochloromethane
$\text{CHBr}_3$	Bromoform
$\text{CHCl}_3$	Chloroform
$Ct$	Product $C \times t$ ( $\text{mg L}^{-1} \text{ min}$ )
DBPs	Disinfection by-products
DNA	Deoxyribonucleic acid
HAAs	Haloacetic acids
$\text{HOCl}$	Hypochlorous acid
$K$	Constant for a specific microorganism offered to a killing agent ( $\text{mg}^n \text{ L}^{-n} \text{ min}$ )
$k$	Decay constant ( $\text{time}^{-1}$ )
NOM	Natural organic matter
$n$	Constant ("coefficient of dilution")
$N_0$	Number of microorganisms at time 0
$N_t$	Number of microorganisms at time $t$
NTU	Nephelometric turbidity units
$\text{OCI}^-$	Hypochorite ion
RNA	Ribonucleic acid
$t$	Time (min)
THMs	Trihalomethanes
THMFP	Trihalomethanes formation potential
TOC	Total organic carbon
$t_\infty$	Residence period for 99% deactivation of microorganisms (min)

### Greek Letters

$\lambda$	Lethality coefficient ( $\text{mg}^{-1} \text{ L min}^{-1}$ )
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