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REVIEW ARTICLE

Application of permanganate in the oxidation of micropollutants: a mini review

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Abstract As a green oxidant, permanganate has received considerable attention for the removal of micropollutants in drinking water treatment. To provide a better understanding of the oxidation of organic micropollutants with permanganate, the oxidation kinetics of 32 micropollutants were compiled. The pollutants include algal toxins, endocrine disrupting chemicals (EDCs), and pharmaceuticals. The oxidation kinetics of micropollutants by permanganate were found to be first order with respect to both contaminant and permanganate concentrations from which second-order rate constants (k'') were obtained. Permanganate oxidized the heterocyclic aromatics with vinyl moiety (i.e., microcystins, carbamazepine, and dichlorvos) by the addition of double bonds. For the polycyclic aromatic hydrocarbons (PAHs) with alkyl groups, permanganate attacked the benzylic C-H through abstraction of hydrogen. The mechanism for the oxidation of phenolic EDCs by permanganate was a single electron transfer and aromatic ring cleavage. The presence of background matrices could enhance the oxidation of some phenolic EDCs by permanganate, including phenol, chlorinated phenols, bisphenol A, and trichlosan. The toxicity of dichlorvos solution increased after permanganate oxidation, and the estrogenic activity of bisphnol A/estrone increased significantly at the beginning of permanganate oxidation. Therefore, the toxicity of degradation products or intermediates should be determined in the permanganate oxidation processes to better evaluate the applicability of permanganate. The influence of background ions on the permanganate oxidation process is far from clear and should be elucidated in the future studies to better predict the performance of permanganate oxidation of micropollutants. Moreover, methods should be employed to catalyze the permanganate oxidation process to achieve better removal of micropollutants.

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1 Introduction

Endocrine disrupting compounds (EDCs), algal toxins, and pharmaceuticals have been detected in water supplies and wastewater effluents around the world [1,2]. Although micropollutants are present in water at low to very low concentrations ($ng \cdot L^{-1}$ to $\mu g \cdot L^{-1}$), they exhibit adverse ecological impacts that have raised concern among public and regulatory groups about the fate of such compounds during potable water treatment and human exposure to drinking water [2–5]. In fact, for EDCs, pharmaceuticals, and herbicides, conventional drinking water treatment processes (i.e., coagulation, sedimentation, and filtration) achieve minimal levels of removal [5–7]. To solve the problem, adsorptive and oxidative processes should be applied as safety barriers against micropollutants, because adsorption can remove organic materials directly and oxidation may transform hazardous contaminants into nonhazardous or less toxic compounds [8]. The common chemical oxidants applied during drinking water treatment include ozone, chlorine, chlorine dioxide, chloramines, ferrate(VI), and permanganate. Ozone attacks double bonds, activated aromatic systems, and neutral amines with great specificity, while hydroxyl radicals ($\cdot OH$), which are formed from ozone decomposition in aqueous solutions, randomly attack carbon–hydrogen bonds in organic molecules [9]. Chlorine reacts with the same moieties as ozone but at much lower rates. Chlorine attack leads to less oxidation and more chlorine substitution, generating halogenated organic compounds, such as chloroform, other trihalomethanes (THMs), and haloacetic acids (HAAs) [10]. Both ozone and chlorine unfortunately can react with bromide to form HOBr and generate brominated by-products (especially chlorine reaction) and

bromate (only ozone reaction), which have potentially worse health effects than their chlorinated counterparts [11]. Chlorine dioxide reacts with tertiary amines and activated aromatic systems, and its reactivity is on a scale between ozone and chlorine [12]. The application of chlorine dioxide is regulated at minimal doses to prevent accumulation of chlorite and chlorate as oxidation by-products [13]. Chloramines are undoubtedly poor oxidants but are used frequently as residual disinfectants to minimize total THM (TTHM, four chloro/bromo analogs) formation. Ferrate(VI) can disinfect microorganisms, partially degrade, and/or oxidize the organic and inorganic impurities, and remove suspended/colloidal particulate materials in a single dosing and mixing unit process, due to its unique properties (namely, strong oxidizing potential and simultaneous generation of ferric coagulating species) [14]. Permanganate mainly reacts with double bonds by donating oxygen, but it can also abstract hydride ions, electrons, or hydrogen atoms [15]. Compared to other oxidants, e.g., ozone, chlorine, chlorine dioxide, and potassium ferrate, permanganate is sometimes preferred because of its relatively low cost, ease of handling, effectiveness over a wide pH range, and comparative stability in the subsurface [16]. More importantly, the oxidation of organic matter using permanganate does not lead to the formation of chlorinated or brominated by-products [17]. As an environmentally useful oxidant, permanganate has received considerable attention and been widely used both in portable water treatment for enhancing coagulation and removing micropollutants [17–21] and the remediation of contaminated groundwater [22].

The objectives of this paper are 1) to compile the kinetics data and mechanisms of the oxidation of organic micropollutants using permanganate; 2) to compare the relative removal for some selected micropollutants in different oxidation processes; and 3) to raise concerns about the influence of coexisting ions and natural organic matter on permanganate oxidation and the toxicity of degradation products for future study.

2 Oxidation of pharmaceuticals and endocrine disruptors

In recent years, there has been growing concern about the occurrence of pharmaceuticals and EDCs in the aquatic environment [1,3,23]. Although most pharmaceuticals and EDCs are detected at trace levels ($ng \cdot L^{-1}$–$\mu g \cdot L^{-1}$), concerns have been raised about the effects of their long-term exposure on public health and the aquatic ecology, especially considering the unknown synergistic effects of pharmaceutical and EDCs mixtures [24]. Indeed, hormonal disruption has become a widely recognized mechanism of toxicity, and a number of laboratory studies have shown that exposure to EDCs can impair reproductive function in adults of either sex, leading to irreversible abnormalities or cancer [3,25]. Pomati et al. [24] recently reported that the growth of human embryonic cells was inhibited by a mixture of pharmaceuticals present in water at environmentally relevant concentrations, and Richards et al. reported the adverse effects of pharmaceutical mixtures on aquatic microorgaisms [26]. Although some adverse health effects of pharmaceuticals and EDCs are still unknown, drinking water should be free from these compounds to minimize the risk of unpredictable long-term impacts based on precautionary principles. Hence, it is important to assess water treatment processes with regard to their potential for removing pharmaceuticals and EDCs.

Chemical oxidation processes involving ozone, chlorine, permanganate, and ferrate are commonly employed technologies for the treatment of some pharmaceuticals and EDCs. The removal of some selected pharmaceuticals using ozonation has been investigated [27]. Westerhoff et al. [8] reported that ozone oxidized steroids containing phenolic moieties (estradiol, ethynylestradiol, or estrone) more efficiently than steroids without aromatic or phenolic moieties (androstenedione, progesterone, and testosterone). However, the formation of brominated byproducts including bromated ion (BrO_3^-) and total organic bromine (TOBr) has limited the application of ozonation of pharmaceuticals and EDCs in bromide- and iodide-containing waters [28]. For the reaction of chlorine with some selected pharmaceuticals and EDCs, second-order rate constants could vary over more than five orders of magnitude [29]. Although the apparent rate constants of chlorine reaction with selected pharmaceuticals and EDCs at pH 7 were $> 10^2 \cdot M^{-1} \cdot s^{-1}$, primary products during chlorination could be considered as precursors of DBPs [30]. Hence, the application of chlorine in the removal of pharmaceuticals was limited. In contrast, permanganate and ferrate(VI) may be regarded as attractive oxidizing agents because they are nonhalogenating, and they generate insoluble and environmentally benign reduction products (e.g., $MnO_{2(s)}$ and $Fe(OH)_{3(s)}$) that can be readily removed through sedimentation/filtration [20]. Ferrate(VI) oxidation could be an effective treatment method for the purification of waters, containing some particular antimicrobials and EDCs [31–34]. However, previous studies found that pH 9 was the most favorable condition to obtain the highest removal efficiency for EDCs, which was an obstacle for ferrate(VI) application in the field. In addition, the instability and high cost of ferrate(VI) also limited its use in field treatment practices. Compared with ferrate(VI), permanganate was preferred because of its effectiveness over a wide pH range, comparative stability, and relatively low cost. Permanganate was also an effective oxidant for some EDCs and pharmaceuticals. Hu et al. [20] found that carbamazepine (CBZ) was rapidly oxidized by permanganate, and around 90% CBZ was oxidized by 75 μM permanganate in < 2 min. In addition, some researchers reported that the kinetics and mechanism of EDCs (i.e.,

estrone, triclosan, bisphenol A, and dichlorvos) degradation using permanganate in aqueous solution over a pH range of 5–9 [21,35–37]. So far, however, there are only a few studies on the oxidation of pharmaceuticals and EDCs by permanganate in water treatment processes and environmental remediation, and thus, future work in this field is recommended.

2.1 Kinetics of the oxidation of EDCs/pharmaceuticals by permanganate

Table 1 summarizes the values of the second-order rate constants (k'') found in the literature for permanganate oxidation of pharmaceuticals/EDCs, including antibacterial, pesticides, substituted phenols, and polycyclic aromatic hydrocarbons (PAHs). The oxidation of contaminants by permanganate was second-order overall and first order with respect to both contaminant and permanganate concentrations. With respect to the variability in k" within and across the families of micropollutants, a general observation that can be made from Table 1 is that a number of micropollutants react with permanganate nearly as fast or faster than the chlorinated ethenes. This comparison is potentially significant because the chlorinated ethenes have been remediated successfully with permanganate in field-scale applications of in situ chemical oxidation (ISCO) [42]. Therefore, it follows that the EDCs and pharmaceuticals, shown in Table 1, are possible candidates for ISCO with permanganate. To ensure successful removal of micropollutants with permanganate in field-scale applications, other factors, such as the influence of background matrices or the potential to form toxic oxidation products, need to be evaluated.

Compared with other oxidants, permanganate is fairly effective in the treatment of certain micropollutants (i.e., CBZ) under neutral condition. The reaction kinetics of CBZ with permanganate followed a generalized second-order rate law, with apparent rate constants of $3.0(\pm0.3) \times 10^2\ M^{-1}\cdot s^{-1}$ at pH 7 and 25°C [20]. Huber et al. [43] reported that both ClO_2 ($k'' < 0.015\ M^{-1}\cdot s^{-1}$ at pH 7, 20°C) and Cl_2 ($k'' < 1.5\ M^{-1}\cdot s^{-1}$; at pH 7, 20°C) react very slowly with CBZ. Hu et al. [20] showed that the second-order rate constant for ferrate(VI) was just $70(\pm3)\ M^{-1}s^{-1}$ at pH 7 and 25°C. Although O_3 ($k'' \approx 3 \times 10^5\ M^{-1}\cdot s^{-1}$) and ·OH ($k'' \approx 8.8 \times 10^9\ M^{-1}\cdot s^{-1}$) oxidize CBZ much more rapidly than permanganate, the higher reactivity of these oxidants is counterbalanced by their shorter lifetimes in natural waters. The half-lives for O_3 and ·OH in water with typical concentration of dissolved organic matter are estimated to be a few minutes and a few microseconds, respectively [44].

2.2 Mechanisms of the oxidation of pharmaceuticals/EDCs by permanganate

The generally accepted mechanism of the oxidation of chlorinated ethenes with permanganate is electrophilic attack by permanganate on the carbon-carbon double bond, with formation of cyclic hypomanganate diester as a reaction intermediate [45]. For the oxidation of CBZ by permanganate, Hu et al. [20] proposed a reaction pathway similar to the oxidation of chlorinated ethenes by permanganate, which was initiated by electrophilic attack at the olefinic double bond on the central heterocyclic ring by permanganate. Electrophilic attack by permanganate led to the ring-opening of CBZ and a series of organic oxidation products through cyclization and hydrolysis reactions detected by LC-MS/MS. Liu et al. [21] reported that the degradation of dichlorvos was also initiated by the attack of permanganate on the C = C moiety through a transition state. The cyclic ester decomposed quickly and led to the formation of dimethyl phosphate (DMP). DMP could be further oxidized to monomethyl phosphate (MMP), with a relatively low-rate constant.

In the case of phenols and PAHs, there have not been any detailed mechanistic studies under environmentally relevant conditions. One mechanism that has been suggested for the oxidation of phenols by permanganate was single electron transfer [46,47]. Both quinones and products suggesting ring cleavage were observed in reactions of permanganate with different substituted phenols [39]. Abe et al. [48] proposed that permanganate directly cleaves the aromatic rings of phenolic compounds to form organic acids and inorganic carbon, since few decomposition products with aromatic rings were observed in the permanganate oxidation of phenol, bisphenol A, and 4-*t*-butyl-phenol. With respect to the PAHs with alkyl groups (i.e., methylnaphthalene), the mechanism might be similar to that applicable to toluene, ethylbenzene, and xylenes. Permanganate attacked the benzylic C-H bond, followed by the cleavage of benzylic C-H bond, and the observed products were correspondingly benzoic acids, aldehydes, alcohols, or ketones [49,50].

3 Oxidation of algal toxins

The widespread occurrence of cyanotoxins in water resources and drinking waters throughout the world has led to not only to livestock deaths but also to several cases of human hepatoenteritis and even deaths [51]. Among the cyanotoxins, microcystin-LR (MC-LR), which originates from the cyanobacteria *Microcystis*, *Anabaena*, *Planktothrix*, *Nostoc*, and *Anabaenopsis*, is the most common in water [52]. To minimize public exposure to microcystins (MCs), the World Health Organization (WHO) has set a provisional guideline value of $1\ \mu g\cdot L^{-1}$ for MC-LR in drinking water [53]. Cylindrospermopsin (CYN) is often associated with blooms of *Cylindrospermopsis*, *Anabaena*, and *Aphanizomenon* and is also common in occurrence [54]. The WHO has responded by proposing a $1\ \mu g\cdot L^{-1}$ guideline for CYN, due to its hepatotoxicity, cytotoxicity, and genotoxicity. Less common in occurrence, the

Table 1 Summary of second-order rate constants (k'') for compounds of EDCs and pharmaceuticals in the Mn(VII) oxidation processes

micropollutants	pH	T/°C	$k''/(M^{-1}\cdot S^{-1})$	Ref.
substituted phenols				
phenol	7.2	16	35.4	[38]
2-chlorophenol	7.0	25	74.3	[16]
3-chlorophenol	7.0	25	13.4	[16]
4-chlorophenol	7.2	16	59.2	[38]
2,4-dichlorophenol	7.2	16	43.8	[38]
	7.0	25	142	[16]
2,6-dichlorophenol	7.2	16	31.9	[38]
2,4,6-trichlorophenol	7.0	25	120	[16]
estrone	5.8	25	4150	[35]
m-cresol	7.0	25	98	[16]
p-cresol	7.0	25	237	[16]
2-nitrophenol	7.0	25	0.243	[16]
	6.8	35	0.305	[39]
3-nitrophenol	6.8	35	0.87	[39]
4-nitrophenol	6.8	35	0.0978	[39]
	7.0	25	0.037	[16]
2,4-dinitrophenol	7.0	25	0.0643	[16]
bisphenol A	7.3	19	72	[37]
pesticides				
aldicarb	7.0	25	2.4	[16]
dichlorovos	7.0	25	15.7	[16]
	7.0	25	25.2	[21]
PAHs				
1-Methylnaphthalene	not buffered	22	0.014	[40]
2-Methylnaphthalene	not buffered	22	0.018	[40]
acenaphthalene	not buffered	22	0.21	[40]
carbazole	not buffered	22	0.44	[40]
chrysene	not buffered	22	0.012	[40]
fluorene	not buffered	22	0.43	[40]
naphthalene	not buffered	22	0.011	[40]
phenanthrene	not buffered	22	0.42	[40]
Phamarceuticals/Antibacterials				
carbamazepine	7.0	25	300	[20]
triclosan	7.0	23	130	[36]
Chlorinated Ethylenes*				
PCE	7.0	25	0.051	[41]
	7.0	25	0.0458	[16]
TCE	7.0	25	1.19	[41]
	7.0	25	0.76	[16]

Note: * The rate constants of chlorinated ethylenes with permanganate were listed here just for comparison.

neurotoxin anatoxin-a (ANTX) still requires further toxicity studies to establish a guideline value. It can be generated by *Anabaena*, *Oscillatoria*, and *Aphanizomenon* [55]. As the WHO progresses with provisional drinking water guidelines for cyanotoxins, efficient treatment strategies are necessary to prevent cyanotoxins from reaching consumers [17].

The application of conventional water treatment technologies (coagulation, flocculation/sedimentation, and filtration) has been reported to be effective for the removal

of cyanobacterial cells but ineffective for the removal of extracellular MCs [56]. Chemical oxidation is a possible option as a safe barrier against cyanotoxins in order to reduce the public health risk. Investigations using ozone [57], chlorine [58], and UV photolysis in the presence of TiO_2 [59] have shown that MC-LR is readily oxidized to nontoxic degradation products under appropriate conditions. Monochloramine and chlorine dioxide, however, are not suitable oxidants for the degradation of MCs in drinking water treatment processes [58,60] because many carcinogenic substances, such as trihalomethane [61] and other mutagens [62], may be produced in the process of chlorination. Hall et al. [63] reported that permanganate was a possible method for removing dissolved MC-LR in waters with low oxidant demand and must be applied before sedimentation/filtration in order to control the final manganese concentrations. In addition, the permanganate dose must be controlled since permanganate applied at high doses might cause cell lysis and toxin release in raw water containing algal cells [64,65]. Therefore, the safe strategy for applying permanganate as a preoxidant in drinking water treatment is to remove the dissolved toxins without cell rupture [66].

3.1 Kinetics of algal toxin oxidation by permanganate

The second-order rate constants for the reactions of MC-LR, -RR, and -YR with potassium permanganate were determined at pH 6.2–8.2 and temperature 10°C–25°C [17]. The reaction of permanganate with MCs was second order overall and first order with respect to both permanganate and toxin. The second-order rate constant for the reaction of MC-LR with permanganate at pH 7 and 20°C was $357.2\ M^{-1}\cdot s^{-1}$. The influence of pH on the oxidation process was not appreciable, and the activation energy was $28.8\ kJ\cdot mol^{-1}$. Slightly higher reactivity with permanganate was found for MC-RR ($418.0\ M^{-1}\cdot s^{-1}$) and MC-YR ($405.9\ M^{-1}\cdot s^{-1}$).

Rodríguez et al. [19] examined the kinetics of the oxidation of CYN with permanganate and found that the oxidation of CYN with permanganate was a rather slow process with the rate constant $< 1\ M^{-1}\cdot s^{-1}$. Therefore, the permanganate dose required for CYN oxidation is very high and not applicable in waterworks. Permanganate is a selective oxidant, which reacts mainly through addition to double bonds. For this reason, permanganate is a good option for the oxidation of MC-LR, with a rate constant at pH 7 and 20°C of $357.2\ M^{-1}\cdot s^{-1}$ [17]. The oxidation of ANTX by permanganate is even faster, and the rate constant is $2.5 \times 10^4\ M^{-1}\cdot s^{-1}$ at pH 7 [52]. Chen et al. [67] investigated the reaction kinetics of the oxidation of MCRR by permanganate. Their experimental results indicated that the reaction was second order overall and first order with respect to both permanganate and MCRR, and the rate constant was $544.2\ M^{-1}\cdot s^{-1}$ at pH 6.7 and 25°C. The reaction kinetics of the oxidation of cyanotoxin with permanganate reported in literature is summarized in Table 2.

Rodríguez et al. [52] compiled a kinetic database for the oxidative treatment of three cyanotoxins: MC-LR, CYN, and ANTX with ozone, chlorine, chlorine dioxide, and permanganate. Overall, permanganate can effectively oxidize ANTX ($k'' = 2.3 \times 10^4\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C) and MC-LR ($k'' = 357\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C), while chlorine will oxidize CYN ($k'' = 490\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C) and MC-LR ($k'' = 33\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C), and ozone is capable of oxidizing all three toxins at the highest rates (MC-LR: $k'' = 4.1 \times 10^5\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C; CYN: $k'' = 3.4 \times 10^5\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C; ANTX: $k'' = 6.4 \times 10^5\ M^{-1}\cdot s^{-1}$ at pH 8, 20°C). In addition, Hou et al. [68] compared the performances of permanganate preoxidation and prechlorination for algal toxin removal in full-scale experiments and found that the concentration of algal toxin in the effluent subject to permanganate preoxidation was lower.

3.2 Mechanisms of Algal toxins oxidation by permanganate

Lawton and Robertson [69] proposed that the decomposition of MC and removal of toxicity by permanganate resulted from the attack of unsaturated bonds in the Adda moiety (3-amino-9-methoxy-2,4,8-trimethyl-phenyldeca-4(E), 6(E)-dienoic acid). To support this hypothesis, Rodríguez et al. [17] determined the stoichiometry of the MC-LR/permanganate reaction experimentally. The result obtained was that 2 mol of permanganate were consumed per mole of MC-LR reacted, which supported the hypothesis of permanganate attack on the two double bonds of the Adda group. To further support the hypothesis of permanganate attack of the Adda moiety, the oxidation of a model compound (sorbic acid) was investigated whose chemical structure was similar to the Adda group. An average rate constant value and activation energy for the reaction between permanganate and sorbic acid at 20°C were consistent with those previously obtained for MC-LR, supporting the idea of permanganate attack on the conjugate double bond of the Adda group.

4 Discussion

4.1 Influence of background ions on permanganate oxidation of micropollutants

To test the accuracy of the kinetics model for predicting micropollutant removal in utility source waters, an examination should be made of the influence of important nontarget water constituents on the permanganate reactivity with target compounds. Hu et al. [20] reported that high concentrations of most water constituents, including 10 mM Ca^{2+}, Mg^{2+}, HCO_3^-, NH_4^+, and NO_3^-, 20 $mg\cdot L^{-1}$ $FeOOH_{(s)}$ and $SiO_{2(s)}$, and 10–20 $mg\cdot L^{-1}$ of NOM, had no

Table 2 Summary of second-order rate constants (k'') for the reaction of permanganate with cyanotoxins

algal toxin	$[toxin]_0$/μM	$[KMnO_4]_0$/μM	pH	T/°C	ionic strength/M	k''/($M^{-1}\cdot S^{-1}$)	Ref.
microcystin-LR (MC-LR)	2.4	28.5	7.2	20	0.01		
	1.8	29.8	7.2	20	0.01		
	1.3	30	7.2	20	0.01		
	0.9	30.7	7.2	20	0.01	357	
	2.4	35.6	7.2	20	0.01		
	2.4	45.1	7.2	20	0.01		
	2.4	61.1	7.2	20	0.01		
	2.3	30	6.2	20	0.01	355	[17]
	2.3	30	8.2	20	0.01	361	
	2.4	29	7.2	10	0.01	236	
	2.4	29	7.2	15	0.01	299	
	2.4	29	7.2	25	0.01	440	
MC-RR	1.9–2.5	30	7.2	20	0.01	418	
MC-YR	1.9–2.5	30	7.2	25	0.01	406	
MC-RR	4.22	31.6	5.0	25		620	
	4.22	31.6	6.7	25		544	
	4.22	31.6	9.0	25		484	
	4.22	31.6	6.7	15		404	[67]
	4.22	31.6	6.7	20		469	
	4.22	31.6	6.7	30		594	
anatoxin-a (ANTX)	1	0-9.5	6.8	20		23000	[52]
cylindro-spermopsin (CYN)	4.81–6.13	1380-3160	7.0	20		0.3	[19]

significant effect on CBZ-permanganate reaction rates. In contrast to the substances described above, the presence of three reduced species (RS), Fe^{2+}, Mn^{2+}, and HS^-, decreased the apparent rate of CBZ oxidation substantially. RS are typically present in natural water at only very low concentrations, and thus, the kinetic model based on the kinetic parameters obtained in laboratory solutions could be used to estimate CBZ removal during permanganate treatment processes at water utilities. For the elimination of MCs by permanganate, the determined rate constants in Milli-Q water can be applied to predict the elimination of MCs during permanganate oxidation in natural waters [17], and it seemed that the influence of nontarget compounds could be neglected. However, Jiang et al. [36,47] demonstrated that some ligands, including phosphate, pyrophosphate, EDTA, and HA, could accelerate permanganate oxidation of triclosan, bisphenol A, as well as phenol and 2,4-dichlorophenol, while these ligands had no influence on the oxidation of carbamazepine and methyl p-tolyl sulfoxide by permanganate. This was mainly attributed to the effects of identified Mn(III) complexes, which would otherwise autodecompose spontaneously in the absence of ligands. He et al. [70] reported that high nominal molecular weight fractions of humic acid could enhance phenol oxidation by permanganate significantly at pH 7. Humic acids of different origins affected the oxidation of phenol and substituted phenols by permanganate to different extents, depending on the properties of HA as well as the position and amount of substitutes on the benzene ring [71]. It was also reported that the oxidation rate of bisphenol A and estrone using permanganate in tap water or filtered natural water background was significantly higher than that in ultrapure water system [35,37]. Therefore, the role of background matrices of real waters in the permanganate oxidation processes is far from clear and should be elucidated in the future studies, especially the influence of RS and ligands (i.e., Mn^{2+}, Fe^{2+}, and humic substances).

4.2 Toxicity of degradation products generated in the process of micropollutants oxidation by permanganate

It is necessary to assess the permanganate mediated incomplete oxidation of pharmaceuticals and EDCs to organic byproducts rather than mineralization to inorganic products, and the toxicity of organic products. Rodríguez et al. [72] investigated the toxicity of degradation products using protein phosphatase 1 inhibition assay (PPIA) during the oxidation of MCs with permanganate and revealed that protein phosphatase 1 (PP1) inhibition emerged only from intact MC, while the oxidation products were nontoxic. Nevertheless, Liu et al. [21] found that the toxicity of

degradation products in the oxidation of dichlorvos by permanganate were much more acute than dichlorvos by a bacterial luminescence test. Yang [37] investigated the toxicity of degradation products using a yeast two-hybrid assay during the oxidation of bisphenol A with permanganate and revealed that the estrogenic activity increased significantly at the beginning of the reaction but finally disappeared after 90 min. Shao et al. [35] also observed an increase in the estrogenic activities in the initial 15 min of estrone's reaction with permanganate and then a gradual decrease. They found that estrogenicity was removed by 73.8% within 30 min of reaction, higher than the removal of estrone within the same duration. These results indicated the following: (1) the toxicity of degradation products or intermediates should be determined in the permanganate oxidation processes, and the increased toxicity may hinder the application of permanganate in drinking water treatment for removing EDCs and pharmaceuticals. (2) Enough contact time must be guaranteed in order to remove estrone to avoid the increase of estrogenicity during permanganate oxidation. (3) The degradation products of EDCs and pharmaceuticals by permanganate oxidation should be determined to better understand the mechanisms of EDCs/pharmaceuticals oxidation by permanganate.

5 Conclusions

The oxidation of numerous organic micropollutants with permanganate has been investigated in the field of water treatment processes and environmental remediation. However, the kinetics, reaction products, and mechanisms of oxidation of certain micropollutants (i.e., algal toxins, pharmaceuticals, and EDCs) by permanganate are not yet available. Hence, further study of the oxidation of these pollutants by permanganate is necessary.

For the reaction kinetics of permanganate with micropollutants under neutral conditions, the kinetics was found to be first order with respect to both contaminant and permanganate concentrations, and second-order rate constants varied over more than four orders of magnitude. Addition and oxidation reactions of permanganate with organic compounds were reported in the literature. For the micropollutants with conjugate double bonds (i.e., MCs, CBZ, and dichlorvos), the oxidation of micropollutants was an electrophilic attack by permanganate on the carbon-carbon double bond, with formation initially of a cyclic hypomanganate diester. For the PAHs with alkyl groups, permanganate attacked the benzylic C-H bond in the aqueous solution. One mechanism that has been suggested for the oxidation of phenols by permanganate is single electron transfer.

However, some gaps in the knowledge of permanganate oxidation are also identified. For example, the influence of coexisting ions and natural organic matter should be clarified to better predict the performance of permanganate oxidation of micropollutants in actual practice and the transformation of these pollutants in the environment. In addition, due to the incomplete oxidation of micropollutants using permanganate, the toxicity of reaction organic products should be investigated in future studies to ensure the safety of permanganate application. Finally, compared with other oxidants, such as ozone, the reaction rate of certain target compounds with permanganate is relatively low and, thus, studies on the catalytic oxidation of micropollutants using permanganate are valuable.

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