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Research Article

Keywords: Diclofenac sodium, UV/chlorine, Toxicity, Degradation intermediates, RCS, chlorination

Posted Date: June 24th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-475892/v1>

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Version of Record: A version of this preprint was published at Journal of Photochemistry and Photobiology A: Chemistry on November 1st, 2021. See the published version at <https://doi.org/10.1016/j.jphotochem.2021.113667>.

Degradation of diclofenac sodium by the UV/chlorine process: Reaction mechanism, influencing factors and toxicity evaluation

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Abstract

This study examined the reaction mechanism, influencing factors and toxicity of diclofenac sodium (DS) degradation by ultraviolet (UV)/chlorine process. The UV/chlorine was capable of eliminating DS from water. The DS degradation during the UV/chlorine process followed a pseudo-first-order kinetic model that was influenced by chlorine dosage, solution pH, humic acid and bicarbonate concentrations. The free chlorine affects not only DS elimination, but the contribution of various active species as well. Increasing free chlorine dosage from 1 to 7 mg·L⁻¹ increased the first-order rate constant of NaClO, ·OH and reactive chlorine species (RCS) from 0.00063, 0.00328 and 0.00203 min⁻¹ to 0.00233, 0.0101 and 0.0974 min⁻¹, respectively, and increased the contribution of RCS from 8.20% to 75.71%, while the contribution of UV, NaClO, and ·OH were declined from 76.02%, 2.54% and 13.24% to 14.63%, 1.81%, and 7.85%, respectively. The contribution of RCS became increasingly prominence with the increment of free chlorine dosage. The $k_{\text{obs,UV/chlorine,DS}}$ value decreased from 0.0797 to 0.0445 min⁻¹ as pH increased from 5.0 to 8.0. The presence of bicarbonate and natural organic matter both exerted an inhibitory effect on DS degradation. Eleven intermediate products were identified and the degradation pathway involved C-N cleavage, condensation, hydroxylation, and decarboxylation was proposed. The UV/chlorine process effectively reduced acute toxicity and was superior to chlorination. The genotoxicity induced by a chlorinated solution treated by the UV/chlorine process exhibited negative genotoxicity. These results show that the UV/chlorine process is capable for the degradation and detoxification of DS.

Keywords: Diclofenac sodium; UV/chlorine; Toxicity; Degradation intermediates; RCS; chlorination

Introduction

Pharmaceuticals and personal care products (PPCPs) are notable emerging organic contaminants in the aquatic environment due to their frequently occurrence and potential adverse impact on ecosystems and human health (Baalbaki et al. 2016; Deng et al. 2020; Kosma et al. 2014; Padhye et al. 2014; Richardson and Ternes 2014). As can be used as non-selective cyclooxygenase inhibitor and non-steroidal anti-inflammatory agent with antipyretic and analgesic

actions, diclofenac sodium (DS) is one of widely used PPCPs and the annual consumption in China is approximately 1000 metric tons (Halling-Sørensen et al. 1998; Kümmerer 2009; Liu and Wong 2013; Stepanova et al. 2013; Vieno et al. 2007; Zhang et al. 2008). Owing to its broad applications, residuals of DS have been detected widely in sewage water, surface water and drinking water, with concentrations ranging from $\text{ng}\cdot\text{L}^{-1}$ to $\mu\text{g}\cdot\text{L}^{-1}$ (Gómez et al. 2006; Kasprzyk-Hordern et al. 2008; Larsson et al. 2007; Stepanova et al. 2013; Stülten et al. 2008; Ziyilan and Ince 2011). Even in trace concentrations, DS is a potentially toxic metabolite and exerts a negative effect on the growth of terrestrial animals and aquatic organisms, moreover, DS also can promote drug resistance among pathogens and potentially threatening human health (Czech and Oleszczuk 2016; Letzel et al. 2009).

Conventional water and wastewater treatment technologies (e.g., coagulation and sedimentation) cannot effectively destruct DS (Carballa et al. 2004; Joss et al. 2006; Kimura et al. 2007; Stülten et al. 2008; Ternes et al. 2002, 2004; Vieno et al. 2007; Zhang et al. 2008). Therefore, effective technologies must be developed to eliminate DS from water. Many methods such as activated carbon adsorption (Westerhoff et al. 2005), chlorine disinfection (Westerhoff et al. 2005), ultrasonic irradiation (Hartmann et al. 2008), ozonation (O_3) (Rosal et al. 2009; Westerhoff et al. 2005), UV/ O_3 (Justoa et al. 2013), Fenton oxidation (Ravina et al. 2002), UV/Fenton (Bae et al. 2013), UV/ TiO_2 (Rizzo et al. 2009), and UV/ H_2O_2 (Kim et al. 2014) have been investigated for DS removal. UV disinfection is a promising treatment technology for drinking water because it can provide a high level of disinfection without chemical addition. Chlorine is the most commonly used chemical disinfectant with the advantages of low cost, high efficiency and persistence (Liu et al. 2019). The combined UV/chlorine process can yield various highly oxidized radicals such as hydroxyl radicals ($\cdot\text{OH}$), as well as reactive chlorine species (RCS) (e.g. chlorine atom radical ($\text{Cl}\cdot$), dichloride radicals ($\text{Cl}_2\cdot^-$), and hypochlorous acid radical ($\text{ClOH}\cdot^-$)), $\cdot\text{OH}$ or RCS plays a major role in the removal of a wide range of contaminants. Furthermore, the UV/chlorine process has been proved to effectively remove many micropollutants and maintaining residual disinfection capacity in drinking water distribution

systems to provide multiple disinfection barriers (Fang et al. 2014; Jin et al. 2011; Qin et al. 2014; Wang et al. 2017; Xu et al. 2020; Yang and Zhang 2019; Yang et al. 2021; Zhang et al. 2019). The UV/chlorine process has been confirmed to be superior to UV/H₂O₂ process for the degradation of micropollutants (Guo et al. 2018). Consequently, in view of the extensive use of free chlorine and the advantages of UV/chlorine process as an emerging advanced oxidation process (AOP), it is worth examining whether the UV/chlorine process can substantially enhance the DS degradation in comparison to the effectiveness of free chlorine or UV alone, and determined the contributions of different reactive species for DS removal. If so, the UV/chlorine process could be used as a water treatment method to remove DS, especially if such an investigation also showed the ecotoxicity control efficiency of the treatment solution to be satisfactory.

The main objectives of this study were to: (1) investigate the degradation kinetics of DS during the UV/chlorine treatment; (2) reveal the contributions of different reactive species to DS degradation; (3) evaluate different factors (i.e., chlorine dosage, pH, HA, and common anions) on the degradation behaviors; (4) explore the transformation pathways of DS; and (5) assess the changes of acute toxicity and genotoxicity during DS degradation.

Materials and methods

Chemicals and materials

DS ($\geq 99.5\%$) was obtained from Dr. Ehrenstorfer GmbH (Augsburg, Germany). Acetonitrile (HPLC grade) was provided from Merck (Darmstadt, Germany). The chemicals including 4-nitroquinoline-N-oxide, dimethyl sulfoxide, o-nitrophenyl- β -D-galactopyranoside, sodium dodecyl sulfate (SDS) ($\geq 95\%$) and humic acid (technical grade) were purchased from Sigma-Aldrich (Buchs, Switzerland). Nitrobenzene (NB, HPLC grade $> 99\%$) was purchased from CNW (Dusseldorf, Germany). Sodium hypochlorite (NaClO) (active chlorine $\geq 5.2\%$) was acquired from Sinopharm Chemical Reagent Co., Ltd (Shanghai, China). *Salmonella typhimurium* (TA1535/PSK1002) was purchased from Molecular Toxicology Inc. (Boone, NC, USA). The

lyophilized *Vibrio fischeri* (strain NRRL-B-11177) was obtained from Macharey-Nagel (ref. 945 022) (Düren, Germany).

All chemicals used for solutions (NaCl, NaOH, HNO₃, Na₂HPO₄, NaH₂PO₄, etc.) were reagent grade. All solutions were prepared with ultrapure water produced from a Milli-Q® water purification system (Millipore Corp., Billerica, MA, USA).

Experimental setup

All experiments were conducted in a 1-L photochemical reactor (Fig. S1). The ultraviolet radiation was provided by a 4 W low-pressure mercury lamp (working at 254 nm). The incident light irradiance was 3.0 μW·cm⁻² at 17.5 cm from the UV lamp tube, as measured by a UV radiometer (TN-2365, Taina Electronics Co. Ltd., Ningbo, China).

Unless specifically mentioned, experiments were performed in batch mode at a temperature of 25 °C and the initial solution pH was 6.82. Solution pH was adjusted by adding 0.1 mol·L⁻¹ NaOH or HNO₃. A 1-L solution containing 550 μg·L⁻¹ DS was first prepared in the beaker, then a desired dosage of free chlorine (0, 1.0, 3.0, 5.0 or 7.0 mg·L⁻¹) was added and the obtained solution was mixed using the magnetic stirrer. The UV lamp was preheated for 2 min and then initiated the reaction. 5 mL samples were taken out periodically, and the residual free chlorine was quenched immediately by adding an excessive amount of sodium thiosulfate.

Analytical methods

Free chlorine was analyzed using the N,N-diethyl-p-phenylenediamine (DPD) colorimetric method. An AQ4000 portable colorimeter (Thermo Scientific, Massachusetts, USA) was used to measure the concentration of free chlorine.

A high performance liquid chromatography system (LC-20AB, Shimadzu, Kyoto, Japan) equipped with a Shimadzu SPD-M20A diode array detector was employed to analyze the concentrations of DS and NB. The mobile phase solvent used for DS and NB analysis consisted of

pure water and acetonitrile (50% pure water (0.05% trifluoroacetic acid) and 50% acetonitrile for DS, 65% pure water and 35% acetonitrile for NB) at a flow rate of 1.0 mL/min. The inertsil reverse-phase ODS-SP column (250 mm × 4.6 mm × 5 μm; GL Sciences, Inc., Tokyo, Japan) temperature was maintained at 40 °C. The detection wavelength of DS and NB were set at 275 and 262 nm, respectively, and the injected volume was 10 μL. The pH of the solution was measured with a pH/mV Meter (Ohaus, Changzhou, China).

The intermediates was identified using a Waters liquid phase-mass spectrometry system equipped with 2767 sample manager, 515 HPLC pump, 2489 UV/visible light detector and 3100 mass detector. The mass spectrometry conditions were set as follows: desolvation temperature and source temperature were 350 °C and 120 °C, respectively; desolvation gas flow and cone gas flow were 500 L/h and 50 L/h, respectively; capillary voltage was 3000 V and injection volume was 20 μL. Chromatographic conditions were set as follow. The mobile phase was consist of acetonitrile and water (65:35) with a flow rate of 1.0 mL·min⁻¹, the working wavelength was 265 nm, the injection volume was 10 μL. Positive APCI mode was adopted for the identification of both DS and its transformation products.

Measurement of acute toxicity and genetic toxicity

A Luminometer BioFix®Lumi-10 a portable luminometer (Macherey-Nagel, Düren, Germany) was employed to determine the acute toxicity of water samples through the Microtox® test and lyophilized *Vibrio fischeri* (UNE-EN-ISO 11348-3 2007). The genetic toxicity was examined by SOS/*umu* assay which was performed using *Salmonella typhimurium* and a microplate absorbance reader (Spectramax® M2e, Molecular Devices Corp., San Jose, CA, USA) in a 96-cell microplate. The withdrawn samples were dechlorinated by sodium thiosulfate immediately for acute toxicity

testing. To provide suitable conditions for the bacteria, the pH was controlled between 6.0 and 8.0 and the temperature maintained at 15°C. The acute toxicity was expressed as the relative inhibition ratio of luminescence (%).

The SOS/*umu* assay was performed according to a previously reported method with some modifications (Elisabeth et al. 1998; Li et al. 2018; Oda et al. 1985). In the present study, ampicillin was adopted as an antibacterial agent with a concentration of 50 mg L⁻¹. The incubation time of the mixture of exponentially growing bacterial suspension (300 µL) and water samples (3 µL) was extended to 4.5 h at 37 °C on a reciprocal shaker at 800 rpm. The enzymatic reaction time was set as 20 min at 37 °C on a reciprocating shaker (800 rpm). The β-galactosidase activity (unit) was determined by Eq. (1):

$$\beta\text{-Galactosidase activity (Unit)} = \frac{1000 \times (A_{415} - 1.75 \times A_{570})}{t \times v \times A_{595}} \quad (1)$$

where A_{415} , A_{570} and A_{595} are the absorbances at 415, 570 and 595 nm, respectively, t is the enzyme reaction time (min), and v is the dilution rate of the bacterial suspension.

The genotoxicity in the SOS/*umu* test can be judged by the induction ratio (*IR*):

$$IR = \frac{Unit_{sample}}{Unit_{solvent\ control}} \quad (2)$$

where $Unit_{sample}$ and $Unit_{solvent\ control}$ are the β-galactosidase activity (unit) of water sample and solvent control, respectively.

In general, if the calculated *IR* value exceeds 1.5, the water sample can be considered positive (Jin et al. 2016).

Prior to the SOS/*umu* test, the reacted DS solution was enriched via HC-18 solid extraction column (CNW Technologies, Shanghai, China). The extract was evaporated by nitrogen gas and then re-dissolving in 500 µL dimethyl sulfoxide.

The acute and chronic toxicity of DS and its degradation intermediates on three aquatic organisms (fish, *Daphnia*, and green algae) during the UV/chlorine process were predicted by the ecological structure-activity relationships (ECOSAR, EPA) simulation program (version 1.11) (Li et al. 2019; Zhuang et al. 2019).

Results and discussion

DS degradation

Effect of free chlorine in the UV/chlorine process

The DS degradation kinetics were conducted at different free chlorine dosages (0, 1, 3, 5 and 7 mg·L⁻¹) during the UV/chlorine treatment. Fig. 1 displayed the DS degradation curves as a function of free chlorine concentration (0–7 mg·L⁻¹) during the UV/chlorine process.

Fig. 1 Effect of chlorine on DS degradation in the UV/chlorine process. Conditions: [DS]₀ = 550 µg·L⁻¹, light intensity = 3.0 µW·cm⁻², pH = 6.82.

The degradation of DS followed pseudo-first-order kinetics during the UV/chlorine process, which can be expressed as Eq. (3).

$$-\frac{d[\text{DS}]}{dt} = k_{\text{obs,UV/chlorine,DS}}[\text{DS}]_t \quad (3)$$

where $k_{\text{obs,UV/chlorine,DS}}$ (min⁻¹) represents the pseudo-first-order rate constant.

Based on the slopes of the reaction relationships presented in Fig. 1, $k_{\text{obs,UV/chlorine,DS}}$ values were found to be 0.01883 (0 mg·L⁻¹ free chlorine dosage), 0.02477 (1 mg·L⁻¹), 0.0721 (3 mg·L⁻¹), 0.09017 (5 mg·L⁻¹) and 0.1287 min⁻¹ (7 mg·L⁻¹), respectively. Additionally, $k_{\text{obs,UV/chlorine,DS}}$ exhibited a linear relation with free chlorine dosage ($k_{\text{obs,UV/chlorine,DS}} = 0.01565 + 0.01667[\text{Cl}_2]_0$, $R^2 = 0.9722$, Fig. 1 inset), which linearly magnified from 0.01883 to 0.1287 min⁻¹ as the free chlorine dosage increased from 0 to 7 mg·L⁻¹. $k_{\text{obs,UV/chlorine,DS}}$ was determined to be of the same magnitude

193 as β -cyclocitral and β -ionone during UV irradiation and the UV/chlorine process (Kim et al.
194 2019).

195 The values of k_{obs} during treatment with free chlorine (3 mg·L⁻¹) and UV were determined
196 to be 0.00075 and 0.01883 min⁻¹, respectively, while a much higher k_{obs} was obtained to be 0.0721
197 min⁻¹ for the UV/chlorine process (3 mg·L⁻¹). The UV/chlorine treatment resulted in a much
198 higher k_{obs} which was 96.13 and 3.83 times higher than that for chlorine and UV alone,
199 respectively. The hybrid UV irradiation and free chlorine process exerts a synergistic effect and
200 accelerates DS degradation. DS can be degraded by ·OH, which can be generated during UV
201 irradiation (Wols et al. 2015a). Free chlorine has a slower reaction rate with organic compounds,
202 and a lower removal of DS has been observed during free chlorine oxidation (Rigobello et al.
203 2013; Sharma 2008; Soufan et al. 2012). Both HOCl and OCl⁻ are presented in free chlorine
204 solution, which can generate ·OH and ·Cl under UV light (Kovacic et al. 2016). Furthermore,
205 these radicals can induce a series of chain reactions that lead to the generation of additional
206 radicals, for example, ·OH, ·O⁻, and RCS such as Cl· and Cl₂·, ·OCl, etc. The mechanism of the
207 generated active radicals is shown in Eqs. (4)–(15) (Benitez et al. 2015; Bolton 2010; Chuang et al.
208 2017; Fang et al. 2014; Feng et al. 2007; Guo et al. 2017; Kim et al. 2016; Weidauer et al. 2016;
209 Wu et al. 2016; Zhu et al. 2017). The species generated in the UV/chlorine process play an crucial
210 role in DS degradation.

DS degradation can be ascribed to UV, chlorination, radicals ($\cdot\text{OH}$, $\cdot\text{O}^-$, and RCS such as $\text{Cl}\cdot$ and $\text{Cl}_2\cdot^-$, $\cdot\text{OCl}$.) that were induced through free chlorine irradiated by UV light. The $\cdot\text{O}^-$ has a low reactivity with organic matter, and the contribution of $\cdot\text{O}^-$ during the UV/chlorine reaction is often neglected or underestimated (Fang et al. 2014; Kim et al. 2020; Nikraves et al. 2020). Therefore, the DS degradation by the UV/chlorine process was simply described by Eq. (16):

$$k_{\text{obs, UV/chlorine, DS}} = k_{\text{obs, UV, DS}} + k_{\text{obs, chlorine, DS}} + k_{\cdot\text{OH, DS}}[\cdot\text{OH}]_{\text{UV/chlorine}} + k_{\text{obs, RCS}} \quad (16)$$

where $k_{\text{obs, UV/chlorine, DS}}$ (min^{-1}) represents the determined pseudo-first-order reaction rate constant during the UV/chlorine process, and $k_{\text{obs, UV, DS}}$ (min^{-1}) represents the pseudo-first-order reaction rate constant for UV photodegradation, $k_{\text{obs, chlorine, DS}}$ (min^{-1}) represents the pseudo-first-order reaction rate constant for free chlorine oxidation, $k_{\cdot\text{OH, DS}}$ ($\text{M}^{-1}\text{s}^{-1}$) represents the second order kinetic rate constant for $\cdot\text{OH}$ and DS, $[\cdot\text{OH}]_{\text{UV/chlorine}}$ (M) represents the steady-state concentration of $\cdot\text{OH}$ during the UV/chlorine process, and $k_{\text{obs, RCS}}$ (min^{-1}) represents the pseudo-first-order reaction rate constant for RCS during the UV/chlorine process.

The effect of free chlorine on the contribution of diverse radicals for DS degradation deserves

further investigation. $k_{\text{obs,UV/chlorine,DS}}$, $k_{\text{obs,UV,DS}}$ and $k_{\text{obs,chlorine,DS}}$ can be determined through the UV/chlorine treatment, direct UV photolysis and free chlorine oxidation, respectively. Furthermore, $k_{\text{OH,DS}}$ ($\text{M}^{-1}\text{s}^{-1}$) can be determined by UV/ H_2O_2 oxidation as described in Text S1. The determined second-order rate constant of DS with $\cdot\text{OH}$ ($k_{\text{OH,AAP}}$) is $2.97 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$.

Nitrobenzene (NB) can react with $\cdot\text{OH}$ exclusively with a second-order rate constant of $3.9 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$, and it does not react with other radicals (Fang et al. 2014; Ji et al. 2017). Because of this inherent property, NB was often chosen as a radical probe compound for calculating the contribution of $\cdot\text{OH}$ in AOPs. The NB degradation by the UV/chlorine process was simply described by Eq. (17):

$$k_{\text{obs, UV/chlorine, NB}} = k_{\text{obs, UV, NB}} + k_{\text{obs, chlorine, NB}} + k_{\text{OH, NB}}[\cdot\text{OH}]_{\text{UV/chlorine}} \quad (17)$$

$[\cdot\text{OH}]_{\text{UV/chlorine}}$ can be determined at different free chlorine dosage during the UV/chlorine process as detailed in Text S2. The values of $[\cdot\text{OH}]_{\text{UV/chlorine}}$ were determined to be 6.62×10^{-11} ($1 \text{ mg}\cdot\text{L}^{-1}$), 1.12×10^{-10} ($3 \text{ mg}\cdot\text{L}^{-1}$), 1.55×10^{-10} ($5 \text{ mg}\cdot\text{L}^{-1}$) and 2.03×10^{-10} ($7 \text{ mg}\cdot\text{L}^{-1}$), respectively. Then the pseudo-first-order reaction rate constant ($k_{\text{OH,DS}}[\cdot\text{OH}]_{\text{UV/chlorine}}$) during the UV/chlorine process can be obtained by the product of $[\cdot\text{OH}]_{\text{UV/chlorine}}$ and the calculated $k_{\text{OH,DS}}$. Accordingly, by applying Eq. (16), the values of $k_{\text{obs,RCS}}$ (min^{-1}) can be determined (detailed calculation method is presented in Supporting Information (Text S3)). The contribution of various radical species can be calculated (Fig. 2).

Fig. 2 Contributions of RCS, chlorination, UV and $\cdot\text{OH}$ to DS degradation by the UV/chlorine process under different chlorine dosage. Conditions: $[\text{DS}]_0 = 550 \text{ }\mu\text{g}\cdot\text{L}^{-1}$, light irradiance = $3.0 \text{ }\mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 1.0, 3.0, 5.0 \text{ and } 7.0 \text{ mg}\cdot\text{L}^{-1}$, $\text{pH} = 6.82$.

As can be seen in Fig. 2, RCS played a prominence role in DS degradation when the dosage of free chlorine was greater than 3 mg·L⁻¹. RCS accounted for 65.14-75.71% of the DS elimination during the UV/chlorine process, and the contribution increased with the increase of free chlorine dosage. However, UV photolysis plays an dominant role on DS degradation when the free chlorine dosage was 1 mg·L⁻¹, and the corresponding contribution even reached up to 76.02%. It's worth noting that ·OH play an indistinctive role in DS degradation (7.85-13.24%). Similar results have been observed during the degradation of diatrizoate (Hu et al. 2018), and 1H-benzotriazole (Lee et al. 2019) in the UV/chlorine process, in which the degradation of targeted pollutant was mainly ascribed to the generated RCS rather than chlorination. The free chlorine addition may influence not only DS elimination, but the contribution of various active species. Increasing free chlorine dosage from 1 to 7 mg·L⁻¹ increased the first-order rate constant of NaClO, ·OH and RCS from 0.00063, 0.00328 and 0.00203 min⁻¹ to 0.00233 (increased by 2.69 times), 0.0101 (increased by 2.08 times) and 0.0974 min⁻¹ (increased by 46.98 times), respectively, and increased the contribution of RCS from 8.20% to 75.71%, while the contributions of UV, NaClO, and ·OH were declined from 76.02%, 2.54% and 13.24% to 14.63%, 1.81% and 7.85%, respectively. The contribution of RCS becomes increasingly prominence with the increase of free chlorine dosage.

Effect of solution pH

The existing form of chlorine (HOCl or OCl⁻) is affected by solution pH, which also impacts the photon absorption efficiency during the photolysis of chlorine (Watts and Linden, 2007). The influence of pH (pH 5–8) on DS degradation was investigated during the UV/chlorine process and the results were presented in Fig. 3.

The DS degradation followed pseudo-first-order kinetics in all investigated pH conditions.

The k_{obs} value gradually decreased from 0.0797 min^{-1} at pH 5.0 to 0.0445 min^{-1} at pH 8.0. This result showed that acidic conditions were more effective for DS degradation than neutral and alkaline conditions.

Fig. 3 Effect of pH on DS degradation in the UV/chlorine process. Conditions: $[\text{DS}]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light irradiance = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, and $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$.

Solution pH can affect the formation of radicals and the existing form of DS. HOCl and OCl^- possess different quantum yield coefficient of 1.45 and $0.97 \text{ mol Einstein}^{-1}(254 \text{ nm})$, respectively (Eqs. (6) – (7)) (Feng et al. 2007; Ye et al. 2017). Thus, pH affects the generation of $\text{HO}\cdot$ and $\text{Cl}\cdot$ by their influence on the quantum yield of chlorine photolysis. As the pH gradually increases, the partition coefficient of HClO decreases and the proportion of ClO^- increases. Correspondingly, the formation of $\text{HO}\cdot$ and $\text{Cl}\cdot$ decreases, and ClO^- produces $\text{O}\cdot$ rather than $\text{HO}\cdot$ through photolysis (Eq. (7)). On the other hand, ClO^- is the dominant form of chlorine in alkaline conditions and is more competitive with $\cdot\text{OH}$ and $\cdot\text{Cl}$ than HClO (the dominant chlorine species under acidic conditions). The rate constants of OCl^- with $\cdot\text{OH}$ ($8.8 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$) and $\cdot\text{Cl}$ ($8.2 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$) are higher than those of HOCl with $\cdot\text{OH}$ ($2.0 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$) and $\cdot\text{Cl}$ ($3.0 \times 10^9 \text{ M}^{-1}\text{s}^{-1}$). Thus, the reaction rate constants between $\cdot\text{OH}$ or $\cdot\text{Cl}$ and HOCl or OCl^- both reach 10^9 magnitudes, which indicates that OCl^- can be treated as radical scavenger. Moreover, under alkaline conditions a large amount of OH^- also quickly react with $\text{HO}\cdot$ and $\text{Cl}\cdot$ (Eqs. (14) and (15)), which consume the generated $\text{HO}\cdot$ and RCS and result in a reduction of DS removal.

Previous studies have suggested that the degradation of organic pollutants, especially ionizable organic pollutants, was highly pH-dependent (Cheng et al. 2015; Saïen and Khezrianjoo 2008; Stewart et al. 2008). DS is a weakly acidic compound and its pK_a is 4.0 at 25°C (Ahuja et al. 2006), so it deprotonates in pH range of 5–8, which results in a reduction of DS removal.

Effect of natural organic matter

The effect of natural organic matter (NOM) on DS degradation in the UV/chlorine process

was investigated using commercially prepared humic acid at different concentrations (Fig. 4).

Fig. 4 Effect of NOM concentration on DS degradation in the UV/chlorine process. Conditions: $[DS]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light irradiance = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, pH = 6.82.

As shown in Fig. 4, the k_{obs} value decreased from 0.0721 min^{-1} to 0.0223 min^{-1} as NOM concentration increased from 0 to $7.00 \text{ mg}\cdot\text{L}^{-1}$. The results demonstrated that the presence of NOM hindered DS elimination during the UV/chlorine process.

NOM can react with $\cdot\text{OH}$ ($2.5\times 10^{-4} \text{ mg L}^{-1}\text{s}^{-1}$), $\cdot\text{Cl}$ ($1.3\times 10^{-4} \text{ mg L}^{-1}\text{s}^{-1}$) and $\text{ClO}\cdot$ ($1.3\times 10^{-4} \text{ mg L}^{-1}\text{s}^{-1}$) and generally deemed to be a radical scavenger (Fang et al. 2014; Guo et al. 2017; Lee et al. 2007). Furthermore, NOM exerts an internal filtering effect during UV photolysis and prevents some or all of the UV light from reaching the target pollutant. Thus, increasing NOM concentrations reduces the contributions made by free chlorine, $\text{HO}\cdot$ and RCS. Nevertheless, other researchers have reported that NOM can be activated under UV irradiation to produce solvated electrons, $\text{HO}\cdot$, $^1\text{O}_2$ and reactive triplet states as reactive species (Aguer et al. 1999; Zhang and Hsu-Kim 2010).

Due to the characteristics of humic acid, NOM can affect DS degradation in two different ways, either accelerating the reaction by stimulating production of $\text{HO}\cdot$ or inhibiting the degradation by competing with other matrix components for absorption of the radiation. In this study, the inhibitory effect outweighed the promoting effect and NOM significantly inhibited DS degradation. Similar results also have been reported during the elimination of diuron (Xiang et al. 2018), iopamidol (Zhao et al. 2019), and microcystin-LR (Zhang et al. 2019) by the UV/chlorine process.

Effect of bicarbonate in the UV/chlorine process

Bicarbonate (HCO_3^-) occurred in surface water and groundwater commonly acts as an radical scavenger in AOP systems (De Laat et al. 2011; Ma and Graham 2000). In this study, various levels of HCO_3^- (0–100 $\text{mg}\cdot\text{L}^{-1}$) were introduced to investigate the influence of HCO_3^- on DS degradation (Fig. 5).

Fig. 5 Effect of HCO_3^- on DS degradation in the UV/chlorine process. Conditions: $[\text{DS}]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light irradiance = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, pH = 6.82.

As can be seen in Fig. 5, HCO_3^- exerted an inhibitory effect on DS degradation during the UV/chlorine process. The k_{obs} value obviously decreased from 0.07181 min^{-1} to 0.04657 min^{-1} as the HCO_3^- concentration increased from 0 to 100 $\text{mg}\cdot\text{L}^{-1}$, and the k_{app} value decreased by 35.14%. It is worthnoting that the introduction of HCO_3^- mainly affects the intermediate reaction process and the removal efficiencies of DS after 60 min still maintain high value (> 96%).

HCO_3^- can react with $\cdot\text{Cl}$ and $\text{HO}\cdot$ to generate carbonate radicals ($\text{CO}_3^{\cdot-}$) with the second order rate constants of $2.2 \times 10^8 \text{ M}^{-1}\text{s}^{-1}$ and $8.5 \times 10^6 \text{ M}^{-1}\text{s}^{-1}$, respectively (Buxton et al. 1988; Matthew and Anastasio 2006). Thus, HCO_3^- competes for $\cdot\text{Cl}$ and $\cdot\text{HO}$, and thus decreasing DS removal.

The generated $\cdot\text{OH}$ can be consumed by HCO_3^- to form $\text{CO}_3^{\cdot-}$. Although $\text{CO}_3^{\cdot-}$ is a weaker radical and owns lower oxidizing capacity compared to $\text{HO}\cdot$ and $\text{Cl}\cdot$, it still degrades a variety of organic pollutants with rate constants in the order 10^6 – $10^7 \text{ M}^{-1}\text{s}^{-1}$ (Guo et al. 2018; Wols et al. 2015b). This result were similar to those of atrazine and trimethoprim degraded by UV/chlorine process (Kong et al. 2016; Wu et al. 2016).

Identification of intermediates and proposed pathways

To obtain the detailed information about DS degradation in the UV/chlorine process, the intermediates were analyzed using liquid chromatography-mass spectrometry and eleven intermediate products were identified (Table 1, Fig. S2).

Table 1 LC/MS data in ESI-negative mode for the DS intermediates and their proposed structures

Based on the detected intermediates, a possible degradation pathway for DS was put forward (Fig. 6). The condensation reaction, C-N cleavage, hydroxylation, and decarboxylation of the phenylacetic acid group were identified as the main degradation processes. 2,4-dichloroaniline and 2-hydroxyphenylacetic acid were considered as common transformation products obtained from the cleavage of C-N bond of DS (Cheng et al. 2015; Pérez-Estrada et al. 2005; Vogna et al. 2004). Under the attack of RCS, 2,4,6-trichloroaniline and 2,5-dihydroxyphenyl acetate may be further reaction products.

The aromatic ring of DS can be easily attacked by $\cdot\text{OH}$ (Khabbaz and Entezari, 2017). The formation of carboxymethyl-2-(2,6-dichloro-phenylmino)-4-oxo-pentanedioic acid and hydroxyl-DS can be observed. The latter has been reported often by manganese oxide oxidation and chlorine dioxide oxidations (Huguet et al. 2013; Wang et al. 2014). Notably, hydroxyl-DS will further react with DS to produce [2-(2,6-Dichloro-4-{2-[2-(2,6-dichloro-phenylamino)-phenyl]-acetoxy}-phenylamino)-phenyl]-acetic acid in the condensation reaction. The attack of chlorine on the aromatic ring could lead to the generation of monochloro DS derivatives, as illustrated in other researches (Miyamoto et al. 1997; Quintana et al. 2010; Soufan et al. 2012).

However, the possible chlorination products of mono- and dichlorinated DS were not detected in this study.

Previous research found that decarboxyl-DS can be generated from the decarboxylation of the phenylacetic acid group and transformed directly to an aldehyde group (Bartels et al. 2007; Pérez-Estrada et al. 2005). The formation of 2,6-dichloro-N-(2-methylphenyl) benzenamine, 1,7-dichloro-8-aldehyde-9H-carbazole and 1-chloro-8-methyl-9H-carbazole have not been reported in previous studies. Thus, this is the first study to define these three intermediate products, which may derive from decarboxyl-DCF via chlorination and cyclization reactions.

2,4,6-trichloroaniline, chlorophenol and other small molecule organic acids were ultimately formed and all of them underwent further reactions to eventually form H₂O, HCl and CO₂. It should be noting that the proposed pathway included only a part of the intermediates from DS degradation. Moreover, the unidentified disinfection byproducts were also likely generated during the UV/chlorine treatment. 2,4,6-trichloroaniline is a well known toxic compound, but the eco-toxicity of mono- and dichlorinated DS is still unclear (Bedner and MacCrehan 2006; Wang et al. 2019).

Fig. 6 Proposed pathway of DS degradation during the UV/chlorine process.

Toxicity assessment

Due to the production of various intermediates, it is essential to investigate the toxicity characteristics of the identified intermediates during the UV/chlorine process. The acute toxicity was defined as the half lethal concentration (LC₅₀) in fish and water fleas (*Daphnia spp.*) and the half effective concentration (EC₅₀) in green algae, and the chronic value (ChV) was expressed as

the geometric mean of the no observed effect concentration (NOEC) and the lowest observed effect concentration (LOEC). The ecotoxicity was simulated by ECOSAR program and summarized in Table S1. According to the toxicity assessment levels of the globally harmonized classification and labeling system for chemicals (Nations U 2019) (Table S2), the acute and ChV toxicity levels of DS and its degradation intermediates were determined and presented in Fig. 7 (a) and (b).

The values of $LC_{50,96hr}$ (fish), $LC_{50,96hr}$ (water fleas) and $EC_{50,48hr}$ (green algae) for DS were 37.655, 25.754 and 41.414 $mg \cdot L^{-1}$, respectively. DS itself appears to exert harmful acute effect on daphnia, fish, and green algae, while have a toxic chronic effect on daphnia and fish. The degradation intermediates m/z 619, m/z 589, m/z 126, and m/z 250 present higher acute toxicity and chronic toxicity. Conclusively, the intermediates generated during the UV/chlorine process appear to present aquatic toxicity, in contrast to the parent compound. However, m/z 362 may be completely harmless, the acute toxicity and chronic toxicity of m/z 193 are lower than that of DS, which can result in the reduction in ecotoxicity of the reacted solution.

Toxicity assessment of the individual DS and its degradation intermediates cannot reflect the whole toxic effect change of the reacted solution, which also influenced by other factors, such as concentration, interaction of the degradation intermediates. Therefore, the SOS/*umu* genotoxicity test and an acute toxicity test using luminescent bacteria (*V. fischeri*) were conducted to evaluate the toxicity of the resulted solution after treated by the UV/chlorine process. Before performing the Microtox[®] test and the SOS/*umu* genotoxicity test, $Na_2S_2O_3$ was introduced into the water sample to quench any residual free chlorine and the results were shown in Fig. 7(c) and (d).

As presented in Fig. 7(d), during UV irradiation, the acute toxicity (luminescence inhibition

rate) of the reaction solution initially decreased from 18% (0 min) to 9% (5 min), but afterwards increased and reached a maximum inhibition rate of 26% at 30 min and finally decreased to 25% at 75 min. The slight increase of acute toxicity suggested that UV alone was not effective for reducing the acute toxicity of the reacted solution. This result was well consistent with previous finding in which DS solution was treated by chlorine dioxide (Wang et al. 2014).

During the UV/chlorine process, the luminescence inhibition rates were higher than those resulting from UV treatment alone in the early stage of the reaction (i.e., at sampling times of 5 min, 10 min and 30 min) except at 20 min (10%). However, the inhibition rate gradually decreased from 31.1% to 8% with prolonging the reaction time from 30 min to 75 min. The UV/chlorine treatment was superior to UV irradiation for the control of acute toxicity. As shown in Fig. 7(d), the relative inhibition rate after the UV/chlorine treatment decreased to 8% at 90 min, which was much lower than that of 25% achieved by UV irradiation alone.

Fig. 7 (a) Acute toxicity and (b) Chronic toxicity of DS and its degradation products; (c) Variation of genotoxicity during DS degradation by the UV/chlorine process; (d) Variation of acute toxicity during DS degradation by the UV and the UV/chlorine processes. Conditions: $[DS]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light intensity = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, and pH = 6.82.

Fig. 7(c) showed the genotoxicity change induced by the chlorinated solution treated by the UV/chlorine process increased from 375.44 units (0 min) to a maximum β -galactosidase activity of 624.43 units (45 min). The corresponding removal efficiency at 45 min was 96.3%. The result of SOS/*umu* tests revealed that the intermediates generated during the UV/chlorine process showed a higher genotoxicity than DS itself. As the treatment time extended beyond 45 min, the

β -galactosidase activity gradually decreased and reached a minimum value of 400 units (90 min).

The initial value of *IR* was negative and showed no mutagenicity. However, the value of *IR* was 2.54 (> 1.5) at 45 min and exhibited obvious positive genotoxicity. Thereafter, the value of *IR* decreased to 1.33 at 90 min, which indicated that the treated solution did not possess any positive genotoxicity. It is noteworthy that the *IR* value for the reaction solution was higher than that for the parent compound. Combined effects of the byproducts might strengthen the genotoxicity of the resulted solution (Scheurell et al. 2009).

These observations clearly revealed that some intermediates possessing higher toxicity than DS itself were formed during the degradation process. The asynchronous elimination of DS and detoxification suggested that some chlorinated intermediates contributed to toxicity of the reaction solution in the UV/chlorine process even when DS has been effectively eliminated. The toxicity changes were primarily attributed to the concentration variation of the intermediate products. Nevertheless, the intermediate products were further degraded in the UV/chlorine process as the reaction progressed, thereby decreasing both acute toxicity and genotoxicity. The acute toxicity and genotoxicity of the reaction solution depends on not only the characteristics of the intermediate products, but also their concentrations. Due to a lack of standard materials, this issue could not be addressed in this research and will conduct an in-depth study on this issue.

Conclusions

Rapid and complete elimination of DS from water can be achieved by the UV/chlorine process. The degradation of DS can be well described using a pseudo-first-order model. The $k_{\text{obs,UV/chlorine,DS}}$ exhibits distinct dependence on solution pH and free chlorine dosage. The addition of free chlorine affects not only DS elimination, but the contribution of various active species as

well. Low solution pH is beneficial to DS degradation, while the presence of bicarbonate and NOM retard the DS removal. The degradation of DS is mainly achieved by the attack of reactive species. The relative contributions of $\cdot\text{OH}$ and RCS for DS elimination are dependent on free chlorine dosage. During the UV/chlorine treatment, DS can be converted to 11 by-products. The DS degradation pathway involves the C-N cleavage, condensation, hydroxylation, and decarboxylation. The UV/chlorine process is superior to the use of chlorine alone for the reduction of acute toxicity. The results of this study provide some valuable information for the application of the UV/chlorine process in water treatment plants.

Acknowledgments

This study was supported in part by the National Natural Science Foundation of China (Grant No. 51878582, 51978618, 41801219), the Natural Science Foundation of Fujian Province of China (No. 2018J01526, 2019J01849), New Century Excellent Talents in Fujian Province University (JA14227), The grants from Xiamen University of Technology (XPDKT19026).

Declaration of interests

- Ethics approval and consent to participate

Not applicable

- Consent for publication

Not applicable

- Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author on reasonable request.

- Competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

●Funding

This study was supported in part by the National Natural Science Foundation of China (Grant No. 51878582, 51978618, 41801219), the Natural Science Foundation of Fujian Province of China (No. 2018J01526, 2019J01849), New Century Excellent Talents in Fujian Province University (JA14227),The grants from Xiamen University of Technology (XPDKT19026).

●Authors' contributions

Qingsong Li: Funding acquisition, Resources, Writing - review & editing. Chengran Lai: Data Curation, Writing - Original Draft. Jianwei Yu: Supervision, Writing - Review & Editing. Jingyu Luo: Data Curation. Jing Deng: Funding acquisition, Resources, Writing - review & editing. Guoxin Li: Methodology, Validation. Weizhu Chen: Methodology, Validation. Boqiang Li: Resources.Guoyuan Chen:Methodology, Validation.

●Acknowledgements

This study was supported in part by the National Natural Science Foundation of China (Grant No. 51878582, 51978618, 41801219), the Natural Science Foundation of Fujian Province of China (No. 2018J01526, 2019J01849), New Century Excellent Talents in Fujian Province University (JA14227),The grants from Xiamen University of Technology (XPDKT19026).

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Figures

Figure 1

Effect of chlorine on DS degradation in the UV/chlorine process. Conditions: $[DS]_0 = 550 \mu g \cdot L^{-1}$, light intensity = $3.0 \mu W \cdot cm^{-2}$, pH = 6.82.

Figure 2

Contributions of RCS, chlorination, UV and $\cdot OH$ to DS degradation by the UV/chlorine process under different chlorine dosage . Conditions: $[DS]_0 = 550 \mu g \cdot L^{-1}$, light irradiance = $3.0 \mu W \cdot cm^{-2}$, $[Cl_2]_0 = 1.0 \sim 3.0, 5.0$ and $7.0 mg \cdot L^{-1}$, pH = 6.82.

Figure 3

Effect of pH on DS degradation in the UV/chlorine process. Conditions: $[DS]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light intensity $= 3.0 \mu\text{W}\cdot\text{cm}^{-2}$, and $[Cl_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$.

Figure 4

Effect of natural organic matter (NOM) concentration on DS degradation in the UV/chlorine process. Conditions: $[\text{DS}]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light intensity = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, $\text{pH} = 6.82$.

Figure 5

Effect of HCO_3^- on DS degradation in the UV/chlorine process. Conditions: $[\text{DS}]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light intensity = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, pH = 6.82.

Figure 6

Proposed pathway of DS degradation during the UV/chlorine process.

Figure 7

(a) Acute toxicity and (b) Chronic toxicity of DS and its degradation products (c) Variation of genotoxicity during DS degradation by the UV/chlorine process; (d) Variation of acute toxicity during DS degradation by the UV and the UV/chlorine processes. Conditions: $[DS]_0 = 550 \mu\text{g}\cdot\text{L}^{-1}$, light intensity = $3.0 \mu\text{W}\cdot\text{cm}^{-2}$, $[\text{Cl}_2]_0 = 3.0 \text{ mg}\cdot\text{L}^{-1}$, and pH = 6.82.

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