UV-C, S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C and H\textsubscript{2}O\textsubscript{2}/UV-C TREATMENT OF AQUEOUS BISPHENOL A RESULTS IN DIFFERENT ACUTE TOXICITY PATTERNS

T. OLMEZ-HANCI\textsuperscript{1}, I. ARSLAN-ALATON\textsuperscript{1}, D. DURSUN\textsuperscript{1}, and B. GENC\textsuperscript{1}

\textsuperscript{1}Istanbul Technical University, Civil Engineering Faculty, Environmental Engineering Department, 34469 Maslak, İstanbul, Turkey
E-mail: tolmez@itu.edu.tr

EXTENDED ABSTRACT

Bisphenol A (2,2-bis(4-hydroxyphenyl)propane), a chemical compound used in the manufacture of polycarbonate, epoxy and polysulfonate resins, can alter the endocrine system, causing adverse effects in the reproductive systems of humans and animals. More recently, the application of advanced oxidation processes (AOPs) has been recommended to treat toxic and/or biorefractory compounds including BPA. AOPs involve the fast and non-selective degradation of organic pollutants by highly reactive free radicals such as HO\textsuperscript{*} and SO\textsubscript{4}\textsuperscript{−}\textsuperscript{*} to more biodegradable oxidation intermediates or non-toxic end products. However, it should be considered that owing to the non-selective and difficult-to-control nature of AOPs, the toxicity of degradation products should be measured during their application. Toxicity assessment is of crucial importance in order to decide whether the treatability of industrial pollutants via AOPs is ecotoxicologically and technically feasible. In the present work, the performance of UV-C treatment and two photochemical AOPs, namely S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C and H\textsubscript{2}O\textsubscript{2}/UV-C treatments, was investigated for the degradation and detoxification of 20 mgL\textsuperscript{−1} (88\textmu M) aqueous BPA solutions. The acute toxicity of BPA and its degradation intermediates was examined by employing \( V. \ fischeri \) and \( P. \ subcapitata \) test protocols. Based on preliminary experiments, optimum reaction conditions were set as 2.5 mM oxidant concentration and an initial pH of 6.5. Experimental results indicated that as compared to HO\textsuperscript{*} and SO\textsubscript{4}\textsuperscript{−}\textsuperscript{*}-radical based treatment, direct UV-C photolysis appeared to be inefficient for the removal of BPA (52%) and TOC (3%) after 60 min irradiation. In parallel to these results, only a minor decrease in the acute toxicity towards the investigated test organisms was observed at the end of 60 min irradiation. On the other hand, BPA removal was complete within 3 min when being subjected to S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C oxidation, accompanied by a rapid mineralization that resulted in 84% TOC removal after 60 min treatment. During the application of H\textsubscript{2}O\textsubscript{2}/UV-C oxidation, complete BPA removal could be achieved after only 4 min; whereas 93% TOC was removed after 50 min treatment. Noteworthy were the acute toxicity profiles observed for \( V. \ fischeri \) during H\textsubscript{2}O\textsubscript{2}/UV-C treatment; a prompt reduction from 73% (relative inhibition caused by 20 mgL\textsuperscript{−1} BPA) to only 14% after 5 min was followed by an abrupt re-increase to 85% after 20 min oxidation, thereafter decreasing to 3-5%, speaking for the formation and subsequent degradation of relatively toxic oxidation products by H\textsubscript{2}O\textsubscript{2}/UV-C treatment. During S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C oxidation of BPA solution, however, a continuous decrease in the acute toxicity towards \( V. \ fischeri \) and \( P. \ subcapitata \) was observed; the relative inhibition decreased to insignificant values after 10 min treatment and did not change thereafter. The above results revealed that although treatment efficiencies were found similar for S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C and H\textsubscript{2}O\textsubscript{2}/UV-C processes, differences in acute toxicity responses suggest variations in reaction pathways and products.

Keywords: Bisphenol A (BPA), Advanced Oxidation Process (AOPs), Acute Toxicity, HO\textsuperscript{*} and SO\textsubscript{4}\textsuperscript{−}\textsuperscript{*} radicals, UV-C, S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C, H\textsubscript{2}O\textsubscript{2}/UV-C treatments.
1. INTRODUCTION

Bisphenol A (2,2-bis(4-hydroxyphenyl)propane; abbreviated as BPA herein), an endocrine disrupting compound (EDC), is widely adopted in the production of epoxy resins and polycarbonate plastics, which are used in various food and drink packaging applications, baby bottles and dental sealants (Staples et al., 1998). It is well accepted that BPA is ubiquitous in the environment, including in surface water and treated drinking water (Umar et al., 2013). In natural water systems, BPA is usually present in lower concentrations (<0.01 to 1.9 µg/L), however, it has been detected at concentrations as high as 10 mg/L in landfill leachate (Staples et al., 1998; Yamamoto et al., 2001). Several studies indicate that BPA might result in adverse health effects, such as human prostate cancer, cardiovascular disease, type 2 diabetes, hormonal imbalance, and liver enzyme abnormalities, in addition to reproduction and developmental effects, neurochemical and behavioral effects (Wetherill et al., 2002; vom Saal and Hughes, 2005). The widespread existence of BPA in the aquatic environment, at low but environmentally relevant levels, implies that conventional treatment technologies are not sufficiently effective for the removal of BPA (Chen et al., 2006). Consequently, advanced remediation techniques have to be developed to effectively remove BPA from contaminated environment including water, wastewater, wastewater sludge, sediments and soils (Mohapatra et al., 2010). One should also keep in mind that, it is not only necessary to assess the degradability or fate of BPA in the environment, but also lower its toxicity and estrogenic activity (Huang and Huang, 2009; Rizzo, 2011). Advanced oxidation processes (AOPs) have received great interest in recent years as complementary methods to conventional water treatment or as alternative treatment strategies for industrial wastewater prior to discharge into sewage or into receiving water bodies (Gultekin and Ince, 2007). There is a growing interest in investigating the use of ultraviolet (UV) irradiation and UV based AOPs for treatment of EDCs (Rosenfeldt and Linden, 2004; Chen et al., 2006; Chen et al., 2007; Huang and Huang, 2009). The effectiveness of direct UV-C photolysis is governed by the absorption spectra of the contaminant and the quantum yield, the addition of hydrogen peroxide (H₂O₂) or persulfate (S₂O₈²⁻) to generate highly active free radicals such as hydroxyl (HO·) and sulfate (SO₄²⁻) often significantly lowers the UV dose required for oxidation as compared to direct photolysis (Rosenfeldt and Linden, 2004).

In the present work, the treatability of (20 mg/L; 88 µM) BPA via UV-C photolysis, S₂O₈²⁻/UV-C and H₂O₂/UV-C photochemical oxidation processes was investigated. The changes in acute toxicity patterns were examined in accordance with the Vibrio fischeri and Pseudokirchneriella subcapitata inhibition tests. Although the initial BPA concentration was high as compared to the concentrations typically detected in water and wastewater, it was necessary to monitor oxidation and mineralization characteristics of BPA. Besides, the results of the present research are expected to be useful to judge whether the treatability and detoxification of BPA via UV-C photolysis and AOPs is feasible.

2. MATERIALS AND METHODS

2.1. Materials

BPA (228 g/mol; C₁₅H₁₆O₄; CAS No: 80-05-7; purity: 99.9%) and potassium persulfate (K₂S₂O₈; purity ≥99.5%) were purchased from Sigma-Aldrich (USA) and used as received. Hydrogen peroxide (H₂O₂; 35% w/w) of analytical grade and acetonitrile of chromatographic grade were obtained from Merck KGaA (Germany). Aqueous BPA solutions were prepared with distilled water. Ultrapure water for the chromatographic measurements was prepared with an Arium 611 UV water purification system (Sartorius AG, Germany). All other chemicals required for analytical and experimental procedures were at least of analytical grade and purchased from Merck (Germany) or Sigma-Aldrich (USA).

2.2. The UV-C photoreactor and experimental procedures

UV-C, S₂O₈²⁻/UV-C and H₂O₂/UV-C treatment experiments were conducted at room temperature (25±2°C) in a 1.9 L-capacity cylindrical batch photoreactor covered with
stainless steel. The UV-C photoreactor setup featuring the UV-C light source (λ\text{max}=253.7 nm) and the procedure of a typical experimental run were previously described elsewhere in more detail (Olmez-Hanci et al., 2011). Samples were taken at regular time intervals for up to 120 min and analyzed for BPA, TOC, S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−} or H\textsubscript{2}O\textsubscript{2} and pH. Control experiments were also conducted to observe BPA degradation in the absence of either S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−} or H\textsubscript{2}O\textsubscript{2}.

2.3. Analytical procedures

2.3.1. HPLC Analysis
BPA was quantified with an Agilent 1100 Series HPLC equipped with a Diode-Array Detector (DAD; G1315A, Agilent Series) set at 214 nm. A C18 Symmetry column (3.9 mm×150 mm; 5 µm particle size; Waters, USA) was employed as a stationary phase, while the mobile phase was a mixture of acetonitrile/water used at a ratio of 50/50 (v/v). The flow rate and temperature of the column were set as 1.0 mL/min and 25°C, respectively. The instrument detection and quantification limit of BPA for an injection volume of 50 µL was calculated as 70 µg/L and 210 µg/L, respectively.

2.3.2. Toxicity measurements
The acute toxicity towards the photobacterium Vibrio fischeri during application of UV-C photolysis, as well as S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−}/UV-C and H\textsubscript{2}O\textsubscript{2}/UV-C treatment experiments was measured with a commercial assay kit marketed as BioTox™ (Aboatox Oy, Finland) according to ISO 11348-3 (2008). Prior to the assay the pH and salinity of all samples was adjusted to 7.0±0.2 and 2% (w/v), respectively. After mixing 500 µL of untreated and photochemically treated BPA solutions with 500 µL luminescent bacterial suspensions, the light emission after 15 min contact time was measured at a temperature of 15°C. Percent relative inhibition rates were calculated on the basis of the toxicant-free control. A positive control sample was also included for each test and all bioassays were run in triplicate.

The acute toxicity towards the freshwater microalgae Pseudokirchneriella subcapitata (formerly known as Selenastrum capricornutum) was determined using Algaltoxkit F™ (MicroBioTests, Inc.) microbiotests according to ISO 8692 (2012) and the OECD Guideline 201 (2011). The batch tests were prepared by mixing appropriate volumes of sample, growth medium (adjusted to pH 8.0 ± 0.1) and inoculum in the test vessels. The initial cell density was 10000 cells mL\textsuperscript{-1} (±10%). Thereafter, the test vessels were placed in a chamber under continuous white-fluorescent side illumination (~10000 lux), and incubated at 23°C. The cell density of the algal cultures was measured at 24-h intervals over 72 h. In each case, the cell density was calculated from the optical density of the algae in different samples, measured in 10 cm path-length cuvettes at 670 nm, using a Jenway 6300 model spectrophotometer. Specific growth rates (in day\textsuperscript{-1}) were calculated to determine percent growth inhibition rates relative to the blank sample not containing the substrate. During the course of the experiments (72 h) the algal cultures remained in the exponential growth phase. A series of controls containing only growth media and the algal inocula were also prepared. The tests were carried out in triplicate and under axenic conditions.

In order to eliminate their positive effect on the toxicity measurements, any residual/unreacted S\textsubscript{2}O\textsubscript{8}\textsuperscript{2−} and H\textsubscript{2}O\textsubscript{2} remaining in the reaction solutions was removed with sodium thiosulfate (Merck; Germany) and enzyme catalase (made from Micrococcus lysodeikticus; Fluka; Sweden), respectively.

2.3.3. Other measurements
TOC was measured on a Shimadzu V\textsubscript{PCN} analyzer (Japan) equipped with an autosampler by catalytic oxidative combustion at 680°C, using an infrared detector. An Orion (USA) 720+ model pH-meter was used for pH measurements. Residual oxidant concentrations in
the treated samples were traced by employing the iodometric method (Official Methods of Analysis, 1998; Wahba et al., 1959).

3. RESULTS AND DISCUSSION

3.1. UV-C treatment

The direct UV-C photolysis experiment was carried out with 20 mg/L BPA by using the monochromatic UV irradiation source at $\lambda_{\text{max}}=253.7$ nm at the original pH value of the reaction solution. Figure 1 displays changes in BPA-TOC (a) as well as acute toxicity-percent relative inhibition (b) during UV-C photolytic treatment. As is evident in Figure 1(a), direct UV-C photolysis of BPA was incomplete, resulting in an overall removal of 52% after 60 min. Since it is likely that UV-C photolysis will yield degradation intermediates which may possess toxicity, the oxidation efficiency measured in terms of the TOC parameter might also be more important than the removal of the parent compound. As being expected, for a degradation process carried out in the absence of oxidant and/or catalyst, no significant TOC removal was observed throughout 60 min treatment.

Figure 1. Results of the UV-C treatment experiments. Experimental conditions: 20 mg L$^{-1}$; 17 mg L$^{-1}$ TOC; original pH = 6.5; applied UV-C dose: 21 Wh L$^{-1}$

The changes in the TOC content of the oxidation intermediates (TOC$_{\text{int}}$) being calculated as the difference between the total measured TOC and the theoretical TOC of the remaining BPA were also shown in Figure 1(a). As is apparent in the figure, TOC$_{\text{int}}$ gradually increased during the course of UV-C treatment supporting evidence of the formation and subsequent accumulation of degradation intermediates. Changes in the acute toxicity pattern towards the test organisms *V. fischeri* and *P. subcapitata* indicated that the
untreated BPA sample caused an inhibitory effect of 80% and 40%, respectively. Nevertheless, as UV-C treatment progressed, a general reduction in the inhibitory effect of BPA was evident speaking for the fact that at the end of UV-C treatment, the photolysis products of BPA were not more toxic than the original BPA solution.

3.2. \( \text{H}_2\text{O}_2/\text{UV-C treatment} \)

Figure 2 presents BPA and TOC removals (a) as well as changes in percent relative inhibition values towards \textit{V. fischeri} and \textit{P. subcapitata} (b) during BPA treatment with the \( \text{H}_2\text{O}_2/\text{UV-C} \) oxidation process. From Figure 2 (a) it is evident that BPA removal was very fast and complete within the first min of treatment. TOC removal also proceeded rapidly; a gradual decrease that leveled off after 40 min treatment was observed resulting in 91% TOC removal. The observation that TOC abatement practically stopped after 40 min coincided with the treatment time of complete \( \text{H}_2\text{O}_2 \) exhaustion (data not shown). TOC\textit{int} exhibited a parallel trend to the TOC abatement rate and thus decreased rapidly throughout the reaction. This observation indicated that BPA oxidation products were removed efficiently after 40 min \( \text{H}_2\text{O}_2/\text{UV-C} \) treatment corresponding to a UV-C dose of 14 Wh L\(^{-1}\).

![Figure 2. Results of the \( \text{H}_2\text{O}_2/\text{UV-C} \) treatment experiments. Experimental conditions: 20 mg L\(^{-1}\); 17 mg L\(^{-1}\) TOC; 2.5 mM \( \text{H}_2\text{O}_2 \); original pH = 6.5; applied UV-C dose: 21 Wh L\(^{-1}\).](image)

From Figure 2(b), changes in acute toxicity towards \textit{V. fischeri} and \textit{P. subcapitata} can be followed when BPA was subjected to \( \text{H}_2\text{O}_2/\text{UV-C} \) treatment. \textit{V. fischeri} appeared to be very sensitive and reacted rapidly to the photochemical oxidation of BPA; the inhibition rate that was originally 90% increased to 100% after 1 min treatment when BPA was completely degraded to some initial advanced oxidation intermediates. Thereafter, a prompt decrease down to 14% (5 min) was observed, followed by a re-increase to 85% after 20 min.
treatment. Beyond this treatment period, inhibition rates fell down gradually to almost non-toxic levels (≈ 3-5% relative inhibition) after 40 min oxidation due to the ultimate oxidation of BPA degradation products that resulted in over 90% TOC abatement. *P. subcapitata* followed a different toxicity pattern; the inhibitory effect originally being 56% decreased to 35% within 5 min treatment. After 10 min oxidation with the H₂O₂/UV-C process, the acute toxicity value leveled off at around 30% relative inhibition and did not change thereafter. From the observed findings it is apparent that rapid BPA degradation accompanied with efficient TOC elimination decreased the inhibitory effects of BPA and its oxidation products appreciably and in particular *V. fischeri* reacted very sensitively towards changes in the photochemical reaction medium.

### 3.3. S₂O₅²⁻/UV-C treatment

Figure 3 presents percent BPA and TOC removal efficiencies (a) together with the corresponding percent relative inhibition rates towards the test organisms *V. fischeri* and *P. subcapitata* (b) during BPA treatment with the S₂O₅²⁻/UV-C oxidation process. As can be followed from Figure 3(a), BPA removal was complete after 5-10 min photochemical treatment.

![Figure 3](image-url)

**Figure 3.** Results of the S₂O₅²⁻/UV-C treatment experiments. Experimental conditions: 20 mg L⁻¹; 17 mg L⁻¹ TOC; 2.5 mM S₂O₅²⁻; original pH = 6.5; applied UV-C dose: 21 Wh L⁻¹.

As in the case of H₂O₂/UV-C oxidation, TOC gradually decreased and TOC removal practically stopped after 40 min treatment. The overall TOC removal efficiency of 89-90% was already reached at this time period. Residual S₂O₅²⁻ concentrations were also measured during S₂O₅²⁻/UV-C oxidation of BPA (data not shown) and followed a similar trend to TOC removal patterns; after 50 min photochemical oxidation, 94% of the originally
present $\text{S}_2\text{O}_8^2-$ was consumed for BPA treatment and the photochemical oxidation process stopped after this treatment time. TOC$_{int}$ also exhibited a parallel trend to the TOC abatement rate and thus decreased rapidly throughout the reaction. Figure 3(b) shows percent relative inhibition rates towards $V. \text{fischeri}$ and $P. \text{subcapitata}$ during $\text{S}_2\text{O}_8^2-/\text{UV-C}$ treatment of BPA. This time, the responses of $V. \text{fischeri}$ and $P. \text{subcapitata}$ were relatively close to each other. In both cases, the toxic effect of BPA solution rapidly decreased and leveled off after 10 min $\text{S}_2\text{O}_8^2-/\text{UV-C}$ treatment; the inhibition rate was reduced down to only 2% after 10 min treatment and did not change thereafter for the test carried out with $V. \text{fischeri}$. The same acute toxicity pattern was evidenced for $P. \text{subcapitata}$; the inhibitory effect decreased rapidly to 11% after 2-3 min treatment and reached its lowest value of around 9% relative inhibition after 10 min $\text{S}_2\text{O}_8^2-/\text{UV-C}$ treatment. The discrepancies between toxicity patterns being observed for the studied photochemical oxidation processes could be attributable to the different reaction mechanisms involved resulting in the intermediacy of different oxidation products.

4. CONCLUSIONS
The present work aimed at investigating the treatability of aqueous bisphenol A (BPA) solution employing the UV-C, $\text{H}_2\text{O}_2/\text{UV-C}$ and $\text{S}_2\text{O}_8^2-/\text{UV-C}$ processes with special emphasis placed on the inhibitory effect of BPA and its degradation products. The acute toxicity of BPA and its degradation products was measured by using the photobacteria $Vibrio \text{fischeri}$ and the freshwater algae $Pseudokirchneriella \text{subcapitata}$ test protocols. The following conclusions could be drawn from the experimental results of the study;

- UV-C treatment of BPA solution resulted in poor BPA degradation rates and insignificant TOC removals. If complete BPA removal and effective oxidation is targeted, UV-C photolysis appears to be a kinetically inefficient process. Hence, advanced oxidation processes have to be applied for the elimination of BPA and its organic carbon content from water and wastewater samples.
- $\text{H}_2\text{O}_2/\text{UV-C}$ treatment on the other hand was capable of rapid and complete BPA degradation accompanied with TOC removals exceeding 90%. Acute toxicity tests indicated that $Vibrio \text{fischeri}$ was very sensitive to photochemically induced changes in the reaction solution. As long as the TOC content of BPA was transformed to various oxidation intermediates, the $Vibrio \text{fischeri}$ toxic response kept on fluctuating, whereas the inhibitory effect of BPA on $Pseudokirchneriella \text{subcapitata}$ originally being less than that on $Vibrio \text{fischeri}$ gradually decreased and leveled off when the reaction stopped due to $\text{H}_2\text{O}_2$ exhaustion and/or mineralization.
- $\text{S}_2\text{O}_8^2-/\text{UV-C}$ treatment also brought about rapid and complete BPA degradation together with fast and high TOC removal efficiencies. However, unlike in the case of $\text{H}_2\text{O}_2/\text{UV-C}$ oxidation, the acute toxicity pattern on $Vibrio \text{fischeri}$ and $Pseudokirchneriella \text{subcapitata}$ exhibited a similar behaviour; the inhibitory effect of BPA decreased gradually and did not change further when the reaction stopped and oxidation was practically complete.

The similarities in treatment performances (BPA and TOC removal kinetics) and differences in acute toxicity patterns (of $Vibrio \text{fischeri}$ and $Pseudokirchneriella \text{subcapitata}$) being observed for the studied photochemical oxidation processes are interesting and speculatively a consequence of the different oxidation mechanisms involved resulting in the intermediacy of different degradation products.

REFERENCES


