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Microwave-induced release and degradation of airborne endotoxins from *Escherichia coli* bioaerosol

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**ABSTRACT**

Endotoxins are widely distributed toxins in the outer cell-wall membranes of Gram-negative bacteria and other microorganisms. Chronic exposure to endotoxins can induce and exacerbate airway symptoms and diseases. However, the release and degradation of airborne endotoxins from bioaerosol by microwave (MW) irradiation have not yet been reported. This study investigated the distribution and fate of airborne endotoxins during MW irradiation process, as well as the kinetics and thermodynamics of the degradation of airborne endotoxins. Results showed that MW irradiation induced cell lysis, thus considerably increasing the proportion of cells with ruptured membranes. Furthermore, MW irradiation changed the distribution of airborne endotoxins, sharply decreased the concentration of bound endotoxins from 230 EU/m\(^3\) to 68 EU/m\(^3\), and increased the concentration of free endotoxins from 21 EU/m\(^3\) to 122 EU/m\(^3\). These results indicated that MW irradiation released endotoxins from cells into the atmosphere. MW irradiation likely degraded endotoxins by exerting thermal effects, which achieved a total endotoxin removal efficiency of as high as 35%. Endotoxin degradation was a first-order reaction and required the activation energy of 26.3 kJ/mol.

**1. Introduction**

Bioaerosols are airborne microbial cells, their fragments and particulate matter of any biological origins \([1]\). These small particles are able to affect human health by causing infectious diseases, acute toxic reactions, and allergies \([2]\). Prevention and control measures for bioaerosol have attracted worldwide attention as a result of pandemics of severe acute respiratory syndrome (SARS) and influenza H1N1 viral infections \([3]\).

Microwaves (MWs)-based technologies are attracting more...
Endotoxins are complexes of lipopolysaccharides (LPS) and proteins (Fig. S1 in Suppporting information) and are hazardous biological substances. They are widely distributed in the outer cell-wall membranes of Gram-negative bacteria and other microorganisms [14]. A few previous studies observed the associated waterborne endotoxins release during inactivation of Gram-negative bacteria [15] and intestinal epithelium [16]. These results suggest that the embedded endotoxins can be released into environments after the death of bacteria or cells. Airborne endotoxins are present in the air in two main forms. The first corresponds to pure LPS molecules (free endotoxins). The second form corresponds to LPSs associated with other cell wall components or an intact bacterial cell (bound endotoxins) [17]. Chronic exposure to endotoxins can induce and exacerbate airway symptoms [18], such as asthma, coughing, and respiratory diseases (e.g. toxic pneumonitis, airway obstructions) [19,20]. However, endotoxins release from airborne bacteria under MW irradiation has not been reported.

The degradation of airborne endotoxins has never been reported to the best of our knowledge. Only a few studies have been conducted on the degradation of waterborne endotoxins [21,22]. Conventional oxidative processes employing free chlorine, chloramines, chlorine dioxide, and ozone have been ineffective in waterborne endotoxin degradation [22]. Some AOPs (O₃/H₂O₂ and UV/H₂O₂) were able to effectively oxidize the waterborne endotoxins, which were induced by the hydroxyl radicals [21]. However, the application of AOP technologies is complicated by the extinction of active radicals and inefficient mass transfer process in air. The present study is the first report to present endotoxin degradation in atmospheric environment.

In this study, the distribution and fate of airborne endotoxins, as well as the kinetics (dealing with the rates of inactivation processes) and thermodynamics (dealing with the relations between heat/temperature and inactivation processes) of endotoxin degradation, were investigated. The results of this study may be helpful to provide a new strategy to control airborne endotoxins and to mitigate the environmental impacts of bioaerosols.

2. Materials and methods

2.1. Bioaerosol analysis

A six-stage Andersen sampler (Shanghai Chang Cheng Electronic Technology Co. Ltd., China) was used to obtain bioaerosol samples. A glass petri dish containing 25 ml of nutrient agar medium was placed on each stage of the device, and samples were obtained at a flow rate of 28.3 l/min for 5 min. After sampling, the nutrient agar plates were incubated for 48 h in a constant-temperature incubator at 37 °C. The number of colony-forming units (CFUs) on each nutrient agar plate was manually counted. The number of colonies was corrected by using a positive-hole correction table and then quantified with the following formula [23]:

\[
B = N \times \left( \frac{1}{N} + \frac{1}{N-1} + \cdots + \frac{1}{N-r-1} \right)
\]

where \(P_r\) is the number of colonies after correction in each stage, \(r\) is the actual number of colonies in each stage, and \(N\) is the total number of holes in each stage.

2.2. Airborne endotoxin analysis

Airborne endotoxin samples were collected with the AGI-30 impinger. Samples were stored in a sealed pyrogen-free flask at 4 °C for no more than 2 h. The airborne concentrations of the samples were determined using a chromogenic endpoint limulus amebocyte lysate (LAL) assay reagent. The kinetic chromogenic LAL assay is widely applied in airborne endotoxin tests [24,25]. The results were expressed in EU per cubic meter of air (EU/m³). The schematic of LAL method and detailed information for detecting airborne endotoxins can be found in Fig. S2 in the Supporting information.

2.3. Cell viability

Cell viability is commonly evaluated through staining with acridine orange (AO) or ethidium bromide (EB) [26,27]. AO is a fluorescent dye that can cross intact cell membranes and emit green fluorescence when combined with DNA. EB can only cross injured cell membranes and emit orange fluorescence when intercalated into DNA. Samples were collected from the E. coli suspension before and after MW irradiation. AO and EB solutions were added to the samples at a ratio of 100 μg/ml. After 5 min of resting, the treated samples were observed under a fluorescence microscope (IX51 Olympus, Japan). Each test was repeated thrice, and the average value of fluorescence intensity was taken as the final data.

2.4. Cell morphology

Samples were fixed with 2.5% glutaraldehyde solution at 4 °C for 12 h. The fixed samples were washed three times using 0.1 M sodium phosphate buffer (pH 7.2) for 10 min. Then, the samples were dehydrated in a gradient series of ethanol (10 min in each concentration) and then in 100% ethanol for 10 min. Next, the samples were washed using a 1:1 mixture of tertiary butyl alcohol and ethanol to achieve metathesis of ethanol in the cells. The samples were then dried on a critical point dryer. Finally, the dried cell samples were coated with gold and examined through a scanning electron microscopy (SEM).

2.5. Airborne Escherichia coli inactivation by MW irradiation

The schematic of the experimental setup for Escherichia coli. inactivation is shown in Support information (Fig. S3). E. coli suspension was diluted with sterile air after aerosolization using an aerosol generator (ATM226, Topas, Germany) and introduced into the MW irradiation tube in the MW device (M1-L213B, China). The low, middle, and high power levels of the MW device were 130, 169, and 260 W/m³, respectively. The bioaerosol inactivation tests were conducted under a continuous flow operation. The temperature inside the irradiation tube increased and was maintained around 100 °C (Support information, Fig. S4). The diameter and wall thickness of the MW irradiation tube were 100 and 1 mm, respectively. The length of the tube was 30 mm. Airborne samples were discharged from the outlet of the MW device after MW irradiation and collected for further analysis. The air flow rate varied through the MW device to change the airborne E. coli exposure time to MW irradiation. Each measurement for airborne E. coli was repeated thrice, and the average value was taken as the final data.

The inactivation efficiency (E) is defined as the logarithmic order of bacteria concentration decreasing after MW inactivation [28]:

\[
E = \log_{10} \left( \frac{N_0}{N} \right)
\]

where \(N_0\) is the initial number of bacteria, and \(N\) is the number of bacteria after irradiation.
$E = \frac{N_0 - N_t}{N_0}$

(2)

where $N_0$ is the airborne $E. coli$ concentration before MW irradiation, $N_t$ is the airborne $E. coli$ concentration after MW irradiation (irradiation time $t$), and $E$ is the inactivation efficiency.

2.6. Endotoxin release by MW irradiation

Airborne endotoxins may exist in a bound or free state [17,29]. Bound endotoxins remain bound to the bacterial outer membrane, whereas free endotoxins are shed membrane complexes containing phospholipids, proteins, and LPS. Free endotoxin concentrations were detected by filtering samples with a 0.45 μm mixed cellulose ester filter since the intact bacterial $E. coli$ and other cell wall components are generally 0.5–3 μm [30]. Total endotoxins comprise free and bound endotoxins. The total endotoxin concentration was detected following sample pretreatment through ultrasonic extraction (800 W, 20 kHz, 10 min) [25], which directly increases endotoxin concentration by breaking up cells and allowing LPS release. The free, bound, and total endotoxin concentrations of samples before and after MW irradiation were measured. Each measurement for airborne endotoxins was repeated thrice, and the average value was taken as the final data.

2.7. Airborne endotoxins degradation tests

Pure endotoxin (analytical reagent) was used in the degradation tests. The endotoxin degradation tests were conducted under a continuous flow operation (Support information, Fig. S3). Both the kinetic and thermodynamic of airborne endotoxin degradation were studied in this study. The kinetic of endotoxins degradation was conducted at a constant temperature of 100 °C. The residual concentration of airborne endotoxin were detected according to the above method (Section 2.2). The thermodynamic tests were conducted under different temperatures (40–200 °C) given the same irradiation time (20 s) and initial airborne endotoxin concentration (251 EU/m³). Each test for airborne endotoxin concentration was repeated thrice.

3. Results and discussion

3.1. Bioaerosol inactivation performance by MW irradiation

Fig. 1 shows the survival airborne $E. coli$ under various operating conditions during MW irradiation. A control experiment without MW irradiation showed no antibacterial effect (Support information, Fig. S4), indicating that MW has a role in inactivation. MW irradiation exhibited a rapid reduction of airborne $E. coli$, and the highest removal efficiency ($E$, calculated by Eq. (2)) could reached 4.1-log reduction in 20 s (Fig. 1A). Previous studies on MW application in waterborne microbe disinfection showed that several minutes were often required to achieve satisfactory inactivation performance [31,32]. In this study, the fast inactivation performance under short exposure time compared with previous reports on waterborne cases, which adds to the data on the potential suitability of MW as an anti-bioaerosol technology. Increasing the irradiation time (Fig. 1A) and input energy density (Fig. 1B) of MW can promote the inactivation of airborne bacteria. The experimental data in Fig. 1 demonstrated a linear decrease in survival logarithmic $E. coli$ concentrations with irradiation time. These results suggested that the inactivation reactions of airborne $E. coli$ followed the first-order kinetics. Such microbial inactivation rate laws or kinetic models were often reported in previous studies [33].

The inactivation performance of $E. coli$ bioaerosol, in this study, can reach 4-log, which is more efficient than that of waterborne exposure to MW irradiation (1-log) under similar experimental conditions [9]. Some other literatures reported higher inactivation performance in liquid phase using longer exposure time (several minutes or hours) and larger input energy [32]. Although MW can be strongly absorbed by water molecular, most of energy is converted to increase water temperature instead of inactivating waterborne microorganisms. Therefore, the results presented herein suggest superior inactivation performance of airborne microorganisms using shorter exposure time and expended less energy.

MW can increase temperature in the air over time (Support information, Fig. S5). High temperatures can cause denaturation and cell membrane damage [34], as well as alter enzymatic activities by disrupting weak bonds in active protein forms [35]. Moreover, MW irradiation exerts nonthermal effects on cell viability by interfering with cell signaling pathways, generating current within cells [36], and producing reactive oxygen species such as ‘OH [37]. Our previous studies indicated that a significant difference between MW irradiation and sole thermal treatment (Support information, Fig. S6). These results indicated that the two kinds of mechanisms played certain roles in airborne inactivation, but non-thermal effects should be the primary inactivation mechanism for airborne bacteria at temperatures around 100 °C.

3.2. Cell morphology and membrane damage

Fig. 2 shows the fluorescence of cells stained with AO/EB before and after MW irradiation. Green fluorescence, which is emitted by AO dye (Fig. 2A and B), represents intact cells. The intensity of green fluorescence decreased (Fig. 2E) after MW irradiation because a great proportion of $E. coli$ cells were inactivated by MW irradiation. However, the intensity of orange fluorescence (Fig. 2C and D), which represents injured cells, significantly increased (Fig. 2F). This result indicated that MW exposure significantly increased the proportion of cells with ruptured membranes. Similar finding was observed by Wu et al. [8] that the microwave-irradiated $P. fluorescens$ cells apparently had damaged membranes.

Fig. 1. Airborne Escherichia coli inactivation by microwave (MW) irradiation. Changes in $E. coli$ survival concentrations under various (A) irradiation time and (B) input volume energy density. The dots represent experimental data for airborne tests. And the dashed lines are the fitted curves. (Error bar presents standard deviation, n presents numbers of samples, n = 3).
membrane envelope as observed in the SEM images. Previous researches showed that MW can alter enzymatic activities by disrupting weak bonds in active protein forms [38], and produce reactive oxygen species (such as $\cdot$OH) [37], which was used to damage membrane envelope. The SEM images of *E. coli* cells before and after MW irradiation (Fig. 3) confirmed that the cells were damaged and ruptured after MW irradiation. Given that *E. coli* is a Gram-negative bacterium, which possess a thinner membrane layer than Gram-positive bacterium, its cell membrane is easily damaged. The results obtained in this study provided evidence that airborne bacterial cells are lysed during MW irradiation, suggesting that cell membrane damage is a crucial mechanism of bacterial inactivation through MW irradiation. Similar results were reported by Woo et al [39]. It was found that untreated *E. coli* cells had a smooth surface, while most of the microwave-radiated cells exhibited severe destruction. The surfaces of the microwave-heated cells were damaged and had become rough and swollen.

### 3.3. Airborne endotoxin release during MW irradiation

Endotoxin release after cell lysis was further characterized. Fig. 4 shows concentrations of different endotoxin forms. Before MW irradiation, the total endotoxin concentration was as high as 251 EU/m$^3$, and mainly comprised bound endotoxins, which are derived from *E. coli* cell membranes. After MW irradiation, the bound endotoxin concentration sharply decreased from 230 EU/m$^3$ to 68 EU/m$^3$. However, the free endotoxin concentration increased from 21 EU/m$^3$ to 122 EU/m$^3$. This result was expected because cell membrane rupture released endotoxins into the atmosphere. Endotoxins change from a bound to a free form in the atmosphere.

The leakages of intracellular ions (K$^+$ and Ca$^{2+}$), DNA, and proteins from cells exposed to MW irradiation were also detected in this study.
lipid A is unable to activate the enzymes necessary for triggering the LAL reaction when the LPS molecule is still embedded in the cell membrane [17]. Although ultrasonic extraction technique was used to break up cells and release free LPS into the reaction medium in this study, the LAL method would underestimate the actual amount of endotoxins in air samples. Because the free LPS can agglutinate and form structures with higher molecular weight in aqueous solutions [42], which cannot be detected by LAL assay. Rylander et al. reported that the toxicity of inhaled endotoxin is high when the endotoxins are bound [43]. Therefore, the actual potential risk of endotoxins may be more significant than the experimental data. The effects of limitation on measured results still need further studies in future.

3.4. Airobrne endotoxin degradation by MW irradiation

3.4.1. Degradation of airborne endotoxin and the corresponding mechanism

Fig. 5 compares the airborne endotoxin concentrations under different temperatures. The results showed that MW irradiation at 40 ºC could not degrade airborne endotoxins, because the energy of a MW photon is about 10−5 eV (2.45 GHz), which is much lower than the covalent bonds energy of C–C (3.82 eV) and C–H (4.51 eV). However, MW irradiation at 100 ºC and sole thermal heating conditions presented remarkable and similar degradation performance. This result suggested that thermal effect was likely to be the dominant mechanism for airborne endotoxins degradation. The present study proved that MW irradiation likely degrades endotoxins by exerting thermal effects, which is an effective alternative method for airborne endotoxins removal.

Control of the released endotoxin during microbes inactivation is another major concern, which is known to have toxic and pyrogenic effects. Degradation of this complex compound in water phase was first reported by Sunada et al. [44]. Some oxidative processes generally used for disinfection employing free chlorine, ozone and chlorine dioxide have been ineffective in waterborne endotoxin degradation [22]. These processes suffer from the limitations of harsh conditions with excess of chlorine used in the process and unsatisfactory results. In this study, microwave assisted heating demonstrated a rapid and effective degradation of airborne endotoxins. Although the maximum removal efficiency of 35% was obtained in this study, high removal performance would be expected given longer reaction time.

3.4.2. Kinetic of airborne endotoxin degradation

Because the degradation of airborne endotoxin was dependent on tempreatures, the kinetic test of endotoxins degradation was conducted at a constant temperature of 100 ºC (Fig. 6). The natural logarithm of the residual concentration of endotoxins (ln c) was plotted against the time (t) to yield the degradation kinetic curve. Generally, when endotoxins degradation is a first-order reaction, its kinetics are expressed as follows:

\[
\ln c = \ln c_0 - kt
\]

where \( k \) is a reaction constant, \( t \) is reaction time, \( c \) and \( c_0 \) is the endotoxin concentration at time of \( t \) and 0, respectively. From the curve, the slope of the decline was determined by the least-squares method. By using the value of the slope, the rate constant of bacterial destruction \( (k) \) was calculated by the Eq. (3).

Fig. 6 shows that endotoxin concentration and reaction time are linearly related. This result indicated that the endotoxin degradation followed a first-order reaction, which has a rate law in which the sum of the exponents is equal to 1. Recall that an integrated rate law gives the relationship between endotoxin concentration and time. Eq. (4) predicts that the concentration of airborne endotoxin will decrease in a smooth exponential curve over time. Knowing the rate constant for the decay of airborne endotoxin enables engineers to design reactors to achieve the desired inactivation efficiency. The degradation rate
constants for airborne endotoxin was calculated as 0.008 s⁻¹. This result implies that the rate of bioaerosol inactivation is very high, whereas the endotoxin degradation rate is much slower. Some other literatures reported the degradation of endotoxins in water phase using AOPs (O₂/H₂O₂ and UV/H₂O₂) [21] and photocatalysis [33] technologies. Longer exposure time (several minutes or hours) compared with this study were often used to degrade the low concentrations of endotoxins (less than 0.5 EU/l) [21,33].

3.4.3. Thermodynamic of airborne endotoxin degradation

The endotoxin removal performance under different temperatures were tested. The results (Fig. 7A) indicated that the endotoxin removal performance was dependent on temperatures. For example, endotoxins were not degraded under the temperature of 40°C. When the temperature increased to 200°C, however, endotoxin removal efficiency increased to nearly 35%. Hence endotoxin degradation is likely caused by the thermal effects of MW irradiation.

The reaction constants under each temperature (Fig. 7A) were calculated and plotted in Fig. 7B. When ln(k) is plotted as a function of 1/T, the slope is equal to -Ea/R. The activation energy (Ea) of a reaction represents the minimum energy needed to form an activated complex between reactants. Here, the activation energy of endotoxin degradation in this study was calculated as 26.3 kJ/mol through the Arrhenius equation. Values of Ea for reaction in liquid phase range from 4 to 125 kJ/mol [45]. The degradation of airborne endotoxins with relative low Ea values, which is in the low range of most reactions in liquid phase, indicates a lower energy barrier for endotoxin degradation in gas phase as compared to the reactions in liquid phase.

$$k = Ae^{-Ea/RT}$$ (5)

$$\ln k = \ln A + \left(\frac{-Ea}{R} \right) \frac{1}{T}$$ (6)

where k is the reaction constant, T is the absolute temperature (in kelvins), A is the pre-exponential factor, Ea is the activation energy of the reaction, and R is the universal gas constant.

4. Conclusions

In summary, this study investigated the release and degradation of airborne endotoxins from E. coli bioaerosol under MW irradiation. Airborne bacterial cells are lysed during MW irradiation. Cell lysis significantly increases the proportion of cells with ruptured membranes. MW irradiation sharply decreased bound endotoxin concentration from 230 EU/m³ to 68 EU/m³ and increased free endotoxin concentration from 21 EU/m³ to 122 EU/m³ because endotoxins are released from bacterial cells into the atmosphere. MW irradiation likely degrades endotoxins by exerting thermal effects and can achieve an endotoxin removal efficiency of as high as 35%. The analysis of the kinetics and thermodynamics of endotoxin removal showed that endotoxin degradation follows a first-order reaction and requires the activation energy of 26.3 kJ/mol. The results of this study mainly focus on a particular Gram-negative bacteria of E. coli. And further investigations about the airborne endotoxins associated with other microbes still need to be conducted.

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Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.jhazmat.2018.11.088.

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