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E. coli: Health Impacts, Exposure Evaluation, and Hazard Reduction

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Abstract

Around one billion persons could not possess access to secured potable water. In developing countries, the largest part of the illnesses remains provoked by pathogens infected water. As a well-known pathogen, *Escherichia coli* is largely employed as an indicator of coliform contamination. This work firstly defines microbiologically *E. coli* bacteria, presents a brief history relating to their first discovery and following contagions, and discusses their clinical characteristics besides their subsistence in nature. A general examination concerning different techniques used for controlling such bacteria is presented. The level of morbidity and mortality changes following the strain and the host's properties. In poor nations, diarrhoeal illness largely conducts to dangerous diseases and dying. In rich nations, even if childhood diarrhoea stays not much serious, contagion with verocytotoxigenic *E. coli* may lead to haemolytic uremic syndrome and thrombiotic thrombo-cytopaenia purpura. Conventional water treatments employ chlorine injection that remains neither an appropriate nor economically feasible method in poor regions. Such competitive techniques may be overcome by a more affordable and off-grid method like a device founded on TiO_2 photoelectrocatalytic disinfection concepts and an advanced hydrodynamic cavitation reactor (ARHCR). Applying photoelectrocatalytic processes in scaled-down and portable equipment authorizes performant water treatment when employing an off-grid point-of-use apparatus. A pilot-scale ARHCR was tested to kill microbes in water, and a fresh probable disinfection route of the ARHCR was suggested comprising hydrodynamical and sonochemical impacts. The ARHCR could be used as an encouraging different or finishing instrument for neutralizing pathogens in wa-

ter, even if more investigation on the disinfection route and scale up remain required.

Subject Areas

Chemical Engineering & Technology

Keywords

Escherichia coli, Water Treatment, Chlorine, Solar Water Disinfection (SODIS), Photoelectrocatalytic Disinfection, Advanced Hydrodynamic Cavitation Reactor (ARHCR)

1. Introduction

Around one billion persons do not possess access to secured potable water and 150 million persons use surface water instead of potable water [1] [2]. In developing countries, a large part of the illnesses stays provoked by pathogens infected water [2]. Thus, furnishing secured potable water remains a huge dare to humans [2]. As a part of the present water treatment techniques, chlorination [3] [4] [5], ozonation [6], and ultraviolet (UV) technology remain frequently employed for killing pathogens and treating water [7] [8]. However, the two first methods form toxic disinfection by-products (DBPs) and the last one suffers from energy consumption [9] [10] [11]. To deal with such challenges, solar water disinfection (SODIS) was proposed as a cost-effective, point-of-use water treatment process, and especially practicable in developing countries [12] [13] [14]. Nonetheless, such a technique needs a comparatively longer period (from many hours to many days) for killing microbes following the intensity of accessible sunlight and water contamination [2]. Consequently, if more important disinfection rates in direct sunlight could be attained, it could certainly be a viable solution to the remaining disinfection techniques accessible [15] [16].

As an encouraging green method for ecological treatment and water remediation, photocatalysis has emerged during the past thirty years [2]. In photocatalytic disinfection technology, semiconductors with an appropriate optical bandgap may be employed as photocatalysts to produce reactive oxygen species (ROSs) in the occurrence of light for demobilizing microbes. Combining photocatalysis and SODIS methods seems to be a plausible concept to increase the kinetics of SODIS [2].

This work aims to focus on *Escherichia coli* especially its health impacts, exposure evaluation, and hazard reduction. This work firstly defines microbiologically *E. coli* bacteria and presents a brief history relating to their first discovery and following contagions. To understand *E. coli*'s behavior, a short description relating to their metabolism and physiology is given. As humankind is concerned by *E. coli* contagion, their clinical characteristics are discussed besides their subsistence in nature. A general examination concerning different techniques used

for controlling such bacteria is finally presented.

2. Microbiological Viewpoint and Natural History

2.1. Microbiological Viewpoint

In most cases, *E. coli* are Gram-negative, rod shaped (2.0 - 6.0 mm in length and 1.1 - 1.5 mm wide bacilli) bacteria with rounded ends (**Figure 1**) [1] [15]. The real form of such microbes could, nonetheless, change from spherical (cocci) cells to elongated or filamentous rods [17] [18]. *E. coli* are non-spore forming, and are generally motile through the action of peritrichous flagella. *E. coli* are facultatively anaerobic and generate gas from fermentation of carbohydrates, as seen by acid and gas formation from lactose at 37˚C and 44˚C. Most *E. coli* produce a positive ortho-nitrophenyl-β-D-galactoside (ONPG) reaction, showing β-galactosidase activity. The methyl red reaction is also positive for *E. coli* showing mixed acid fermentation of glucose; however, the Voges-Proskauer reaction (acetoin production) is negative. *E. coli* generate indole, yet are not able to hydrolyze urea or develop in Møller's KCN broth (depicting an incapability to develop in the existence of cyanide). In addition, formation of hydrogen sulfide (H_2S) is not usually clear when *E. coli* are cultured on triple sugar iron (TSI) agar or Kligler's iron agar (KIA). *E. coli* as well do not induce gelatin liquefaction via gelatinase activity. The majority of strains decarboxylate lysine, utilize sodium acetate, yet do not develop on Simmons' citrate agar, where citrate is the only carbon source [1].

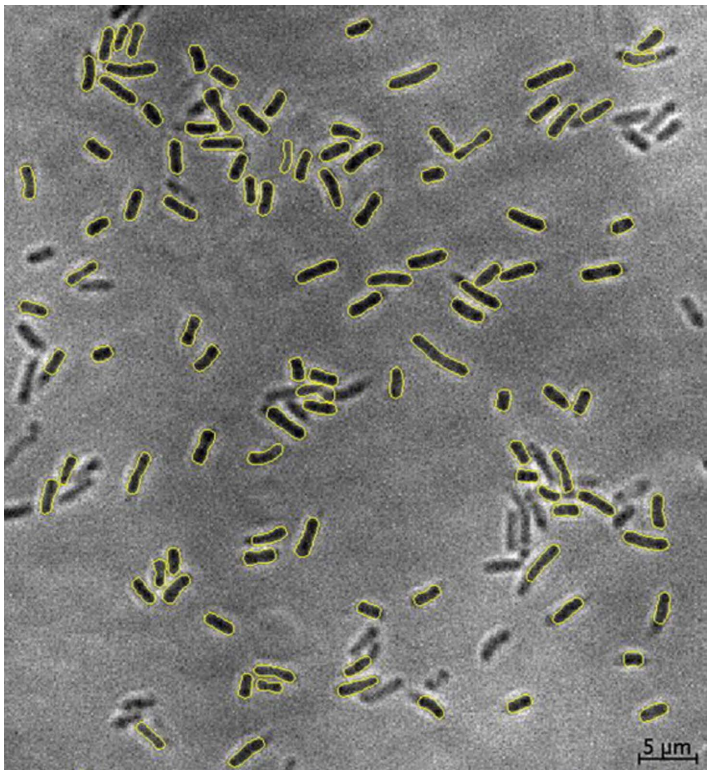

Figure 1. Typical microscope image of *E. coli* cells processed with Microbe Tracker [15].

Numerous *E. coli* cells are capsulated or microcapsulated and such capsules are constituted of acidic polysaccharides [1]. Mucoid strains of *E. coli* generate extracellular slime consisting either of a polysaccharide of certain K antigen specificities, or a usual acid polysaccharide (frequently reported as M antigen) formed of colanic acid [19]. *E. coli* display fimbriae (or pili) of changing structure and antigenic specificity and since such fimbriae are hydrophobic, they furnish host- or organ-specific adhesion features.

Many *E. coli* serogroups are familiar and the plurality is non-pathogenic; nonetheless, several groups could provoke dangerous diarrhoeal disease, sometimes with fatal outcome. *E. coli* is of faecal origin and is almost exclusively detected in the digestive tract of warm-blooded animals, especially human beings. Therefore, observation of *E. coli* in drinking water is employed as an indicator of human or animal excreta pollution, and is known as the coliform index [20].

The most famous and well-investigated *E. coli* strain is enterohaemorrhagic (EHEC) *E. coli* O157: H7 [1] [21]. Members of the "O157" serogroup possess the usual somatic (cell surface) O antigen, while the flagellar H antigen is employed to define the specific serotype. *E. coli* O157: H7 is seen as one of the most problematic and pathogenic serotypes, and is frequently synonymously referred to as EHEC. From 1982 to 2002, *E. coli* O157: H7 was notified in 49 states of the USA and related to 73,000 illnesses [22]. Such serotype manifests extended subsistence in water at low temperatures [23] [24]. Subsistence was indeed depicted to expand beyond 8 months in a farm water gutter, and such microbes were then capable to colonize cattle. Importantly, swimming in polluted water has as well led to outbreaks of contagion [25] [26].

Some less frequently faced strains of *E. coli* could be found in nature and drinking water reservoirs, and could as well provoke diarrhoeal illnesses (like dehydrating diarrhoea and traveler's diarrhea [27]) via changing routes [1]. The incubation period for disease depends on strain and this is mostly related to the changing pathogenic routes revealed. In most cases, the incubation period is 1 - 2 days, even so could expand to 5 days. Even if the pathogenic character of *E. coli* has been recognized for a long time, its character as an enteric pathogen has not long ago been reinforced via the manifestation of *E. coli* O157: H7 and the relationship of such strain with haemorrhagic enteritis and haemolytic uremic syndrome (HUS) [28].

E. coli are linked with a set of human infections, following circulation from the intestines of patients who have an underlying problem [1]. As an illustration, urinary tract infections (UTIs) attributed to *E. coli* frequently happen following direct diffusion from the rectum to the urethra. Infections at other body sites commonly grow by haematogenous diffusion (through the blood stream), as typified by appearance of meningitis in young babies. *E. coli* are as well a frequent reason for postoperative wound contagion, where direct infection of the wound (when the bowel was opened) could grow, or indirect infection by faecal infection of patient fingers. *E. coli* can as well infect patients via colonized members of the health care team, as well as other patients [1].

As aforesaid, some strains of *E. coli* provoke diarrhoea after faecal-oral diffusion from humans and animals [1]. The next sections will be dedicated mostly to diarrhoeal contamination emerging from the different pathogenic types of *E. coli* and will define the epidemiology and clinical characteristics as well as virulence factors and their underlying genetic pathways [29].

2.2. Natural History

Escherichia coli was originally discovered in 1885 and called "*Bacterium coli*" [30] by Dr. Theodor Escherich, a German paediatrician [1]. He discovered the bacterium during investigations of the intestinal flora of infants. After that, the bacterium was established to possess pathogenic features implying extraintestinal contamination [1]. Up until 1919, the name "*Bacterium coli*" was largely employed and then Castellani and Chalmers [31] described the genus *Escherichia* and established the type species *E. coli* [1].

There are at least six principal diarrheagenic pathovars of *E. coli* (two different pathovars are related to UTIs and neonatal meningitis) and each type integrates some form of initial attachment to the host cell with following harmful results, either via the elaboration of a toxin, or direct action [32]. Such *E. coli* types comprise the already mentioned enterohaemorrhagic (EHEC), along with enterotoxigenic (ETEC), enteroinvasive (EIEC), enteropathogenic (EPEC), enteroaggregative (EAEC) and diffuse adherent *E. coli* (DAEC). Each specific type provokes diarrhoeal disease by numerous routes and each disease manifests with many clinical symptoms [1].

3. Metabolism and Physiology, and Clinical Characteristics

3.1. Metabolism and Physiology

Most strains of *E. coli* have the potential to ferment lactose, and the occurrence of lactose will as well display a positive ONPG reaction via the activity of *β*-galactosidase [1]. *E. coli* forms indole from the amino acid tryptophan via action of the enzyme tryptophanase, and this is a special property of *E. coli* from other enteric bacteria [33].

E. coli are incapable to hydrolyze urea and as well do not form gelatinase [1]. *E. coli* do not develop in Møller's KCN broth due to development inhibition by cyanide. H_2S generation is usually absent when *E. coli* are grown on TSI and KIA. These media are utilized to reveal fermentation of specific carbohydrates and via integration of sodium thiosulfate and iron allow H_2S detection. *E. coli* does not deaminate phenylalanine, while most strains can decarboxylate lysine and use sodium acetate. *E. coli* do not grow on Simmons' citrate agar, which includes citrate as the sole carbon source. The biochemical properties of the *Escherichia* genus are listed in **Table 1**, and those of *E. coli* are given in **Table 2**.

3.2. Clinical Characteristics

A century ago, Muir and Ritchie [34] were the premier to define the pathogenic

Table 1. Biochemical properties of *Escherichia* genus [1].

Properties	Reaction
Motility	+
MacConkey growth	+
Mannitol fermentation	+, generally gas
Lactose, 37˚C	Acid +, gas +
Lactose, 44˚C	Acid +, gas +
Adonitol	Rarely fermented
Inositol	Rarely fermented
Indole at 37˚C	Generally produced
Indole at 44˚C	Generally produced
Methyl red reaction	+
Voges-Proskauer reaction	−
Urea	No hydrolysis
Phenylalanine deamination	−
Kligler's H_2S (hydrogen sulfide) medium	No blackening
Møller's KCN (potassium cyanide) medium	No growth
Gluconate oxidation	−
Gelatin liquefaction	−
Glutamine acid decarboxylase	+
Lysine decarboxylase	+

Table 2. Biochemical properties of *E. coli* [1].

Properties	Reaction
Gram stain	Negative
Morphology	Straight rods
Motility	+ (peritrichous) some non-motile
Aerobic and anaerobic growth	+
Oxidase	−
Catalase	+
MacConkey growth	+
D-mannitol fermentation	+, generally gas (over 90% of strains)
Lactose, 37˚C	Acid +, gas + (over 90% of strains)
Lactose, 44˚C	Acid +, gas + (over 90% of strains)
D-adonitol	Rarely fermented (over 90% of strains)
Inositol	Rarely fermented

Continued

D-glucose	Acid
Indole at 37˚C	Generally produced
Indole at 44˚C	Generally produced
Methyl red reaction	+ (over 90% of strains)
Voges-Proskauer reaction	− (over 90% of strains)
Urea	No hydrolysis
Phenylalanine deamination	− (over 90% of strains)
H_2S (triple sugar iron) medium	No blackening (over 90% of strains)
KCN (potassium cyanide) medium	No growth
Gelatin liquefaction	− (over 90% of strains)
Glutamine acid decarboxylase	+
Lysine decarboxylase	+ (75% - 89% of strains)

features of *Bacterium coli* related to contaminations of the intestine and urinary tract, some cases of summer diarrhoea (cholera nostras), infantile diarrhoea and food poisoning [1] [34]. The pathogenicity of *Bacterium coli* was defined as: *Bact. coli* is a normal inhabitant of the intestine of man and other animals. In certain circumstances it acquires pathogenicity, and may cause local or general infection. It is a frequent cause of acute and chronic infection of the urinary tract, and may give rise to an acute or chronic cholecystitis" [1].

Nowadays, *E. coli* is rated as a harmless member of the normal microbiota of the human inhabiting the distal end of the intestinal tract [1]. The organism is usually acquired at birth or via the faecal oral route from the mother and also from nature. The serotypes of *E. coli* that provoke contaminations are listed in Table 3.

E. coli remains the most prevalent reason for acute UTIs and urinary tract sepsis [35]. *E. coli* could as well rise neonatal meningitis and sepsis, and as well abscesses in several organ systems. *E. coli* can as well occasion acute enteritis in humans, as well as animals and is a reason for "traveler's diarrhea", dysenteryl-ike disease touching humans and haemorrhagic colitis frequently known as "bloody diarrhea". Numerous oral challenge investigations have been performed with *E. coli* serogroups to evaluate infection doses. The findings of such investigations propose that levels of 10^5 - 10^{10} EPEC organisms, 10^8 - 10^{10} ETEC and 10^8 cells of EIEC have to be ingested to bring about diarrhoea and infection. Such numbers will naturally change with the age and sex of the recipient as well as the acidity of stomach. For EHEC, the infective dose that is apt to provoke contamination is less than 100 [1].

EHEC is the *E. coli* serogroup that is probably of most clinical concern. Such microbes are recognized to provoke HUS, a case that is distinguished by acute renal failure and generally happens in children under the age of 5 years old. An

Table 3. Serogroups and illness associations of *E. coli* [1].

Virulence Type	Serogroup	Disease	Summary of Host Cell Interaction
Enteropathogenic (EPEC)	055 H6, NM 086 H34, NM 0111 H2, H12, NM 0119 H6, NM 0125ac H21 0126 H27, NM 0128 H2, H12 0142 H6	– Enteritis in infants – Traveler's diarrhoea	– EPEC attach to intestinal mucosal cells causing cell structure alterations (attaching and effacing) – EPEC cells invade the mucosal cells
Enterotoxigenic (ETEC)	06 H16 08 H9 011 H27 015 H11 020 NM 025 H42, NM 027 H7 078 H11, H12 0128 H7 0148 H28 0149 H10 0159 H20 0173 NM	– Diarrhoea, vomiting and fever – Traveler's diarrhoea	ETEC adhere to the small intestinal mucosa and produce toxins that act on the mucosal cells
Verocytotoxigenic (VTEC; including enterohaemorrhagic, EHEC)	026 H11, H32, NM 055 H7 0111ab H8, NM 0113 H21 0117 H14 0157 H7	– *Shigella*-like dysentery (stool contain blood and mucus) – Haemolytic uraemic syndrome	EHEC attach to and efface mucosal cells and produce toxin
Enteroinvasive (EIEC)	028ab NM 029 NM 0112ac NM 0124 H30, NM 0136 NM 0143 NM 0144 NM 0152 NM 0159 H2, NM 0164 NM 0167 H4, H5, NM	*Shigella*-like dysentery	EIEC invade cells in the colon and spread laterally, cell to cell
Enteroaggregative (EAEC)	03 H2 015 H18 044 H18 086 NM 077 H18 0111 H21 0127 H2	Persistent diarrhoea in children	EAEC bind in clumps (aggregates) to cells of the small intestine and produce toxins
Diffusely adherent (DAEC)	Not yet established	Childhood diarrhoea	Fimbrial and non-fimbrial adhesions identified

evaluated 10% of those contaminated with EHEC 0157 may develop HUS, haemolytic anaemia or thrombocytopaenia. Around 5% of cases of EHEC develop haemorrhagic colitis, which could progress into HUS, in which mortality rates may be as high as 10%. The pathogenic pathways of the major pathogenic groups of *E. coli* are summarized by Percival and Williams [1].

4. Subsistence in Nature

The normal source of *E. coli* stays in the intestine of humans and other warm-blooded animals [36]. Even if *E. coli* will remain alive in nature, it does not seem to develop and will finally die [37]. Therefore, natural occurrence is considered a sign of faecal contamination [1]. There is proof that *E. coli* may, nevertheless, remain alive and multiply in tropical environments and so its value as an indication of faecal contamination in such regions is less certain [38] [39].

E. coli possesses significance in water bacteriology since it supplies a helpful sign of faecal infection and not on account of its intrinsic pathogenicity [40]. The philosophy is that, if *E. coli* is there, then possibly so could other pathogenic enteric microorganisms [41] [42]. Regardless of worries about the accuracy of *E. coli* as a sign of water safety, it remains the sole species that almost all routine samples are tested for [1] [43] [44].

Situations of *E. coli* pollution linked with infected under-treated water, and especially public potable water, have been communicated [45] [46] [47]. Contaminated cattle on farms are suspected to engender water infection with *E. coli* [48] [49] and irrigation water has been announced to be a pollution origin [50]. Researchers [51] established that *E. coli* O157: H7 was apt to remain alive for prolonged times in commercially bottled mineral water. As an illustration, after seeding water with 10^3 *E. coli* O157: H7 cells/mL and storing samples at 15˚C, about 70 days passed before *E. coli* was not detectable in non-sterile mineral water, 49 days in sterile mineral water and 21 days in sterile distilled deionized water. Scientists [52] investigated subsistence of *E. coli* O157: H7 in well water microcosms utilizing water implied in a waterborne outbreak. Subsistence of the outbreak strain was similar to a wild-type *E. coli* strain under the identical circumstances. *E. coli* 0157 has been found in well water from four different sites in Scotland, UK [53]. Water samples were seeded with a lux-marked *E. coli* O157: H7 strain and stored at 15˚C. Following the water type, such microbes can be observed by culture for at least 65 days (end of monitoring) [1].

4.1. Subsistence in Water and Epidemiology

All enterovirulent *E. coli* are obtained directly or indirectly from human or animal carriers [1]. Danger from potable water, for that reason, only follows faecal pollution of the supply. Considering the vulnerability of *E. coli* to Cl_2 and other killing agents, even if the microbes infect the supply, sufficient chlorination as a rule efficiently eliminates any health hazard [54].

There are no standards for *E. coli* 0157 (EHEC) inside the 1980 European

Drinking Water Inspectorate Directive [1]. In 1997, the Environment Group of the previous Scottish Office of Agriculture, Environment and Fisheries Department authorized the Water Research Council to assume an investigation inspecting present proof for waterborne transmission of *E. coli* 0157 [1]. Such investigation concluded that there was no proof that *E. coli* 0157 was more enduring in nature or more reluctant to water treatment technologies contrasted with non-pathogenic *E. coli* detected in the human gastrointestinal tract [1].

Worries subsist concerning the possible part that biofilms play in saving enterovirulent *E. coli*. The very elevated infectious injections needed for all enterovirulent *E. coli*, other than EHEC, propose that such potential pathway of transmission is improbable as a hazard. The lower infectious injection of EHEC dose possibly augments the danger of contamination from biofilms in water; however, there have been no epidemics or occasional cases of EHEC involved with adequately disinfected water supplies [1].

Fresh investigation has proved that *E. coli* may persist in aquatic environments, even if the elements that participate in subsistence are not well grasped. Higher predominance of *E. coli* 0157 has surely been observed throughout warmer periods [55]. Recreational water exposure, comprising use of swimming pools, has been implied as an origin of *E. coli* O157: H7 [25] [56], yet details concerning *E. coli* O157: H7 in natural waters and potable water stays restricted, which is mainly related to the reality that frequently only small levels (below the sensitivity limits of detection) of such bacterium take place [1].

In the Netherlands, *E. coli* O157 was isolated (employing a particular enrichment procedure) in 2.7% of 144 private wells, regardless of such samples satisfying the requested potable water standards [57]. In Canada, after a 2-year investigation realized in the Oldman River watershed, 0.9% of surface water samples (n = 1483) were infected with *E. coli* O157: H7 [49]. In river water, *E. coli* O157: H7 has as well been isolated from the Oldman River Basin in Southern Alberta, Canada [58]. *E. coli* O157 was detected in 33 surface water samples in Baltimore, USA. Nonetheless, *E. coli* 0157 was only observed in low levels of <1 cells per 100 mL of raw water [59].

4.2. Enterotoxigenic (ETEC)

Person to person diffusion of ETEC seems to be scarce and most diffusion of occasional illness is via food and water sources [1]. Because of ETEC, numerous waterborne outbreaks have been registered. One very huge outbreak touched more than 2000 staff and visitors to an American National Park in Oregon in the summer of 1975 [60]. ETEC were separated from 20 (16.7%) of 120 rectal swabs inspected. There was a strong association between disease and potable park water in park staff and visitors ($p < 0.00001$). The only group in which there was no correlation with potable water was one comprising visitors on 7-9 July 1975, when chlorination of the water supply was carefully supervised. Water came from a shallow spring that was polluted by a sewage overflow some 650 m uphill from the spring. The supply was assumed to be chlorinated; however, there was

no methodical control of Cl_2 concentrations throughout the distribution system. Different known outbreak touched 251 passengers and 51 crew on a Mediterranean cruise [61]. ETEC was separated from 13 of 22 passengers and 6 of 13 crew sampled. Faecal coliforms were separated from tap water and potable tap water was the single hazard element related to disease in a case-control study. There were numerous disorders in the ship's water system, comprising probable malfunctioning chlorination and malfunctioning includes letting bilge water into the water containers. Researchers [62] notified three outbreaks of ETEC contamination linked with cruise ships. All three outbreaks were related to consuming drinks with ice cubes on board the ship and two were as well linked with potable unbottled water. Water bunkered in overseas ports was the probable origin of the pollution and this water should be treated before usage [1].

4.3. Enterohaemorrhagic (EHEC)

EHEC are observed in the intestines of numerous animal species, comprising cattle [1]. Contamination of persons may go after direct faecal-oral diffusion from infected animals or other persons, or be linked with pollution of food or water. Several outbreaks have pursued the consumption of beef products, especially undercooked beef burgers or salad products. Numerous outbreaks of EHEC related to recreational water contact and consuming drinking water have been reported. Serotype O157: H7 stays the most regularly announced EHEC strain in Europe and North America and the single strain related to outbreaks of potable water-related illness. Nonetheless, EHEC strains other than O157 are more and more being seen as reasons for outbreaks due to foodborne and person-to-person transmission [1].

The first outbreak of contamination related to *E. coli* O157: H7, which was robustly associated with the consumption of potable water, took place in Burdine Township (Missouri, USA) between 15 December 1989 and 20 January 1990 [46]. Of a population of 3126, a total of 243 persons developed illness and of these, 86 developed bloody diarrhoea, 36 were hospitalized and four died [1]. In a case-control investigation founded on 53 cases, the sole crucial element was that contaminated persons drank more cups of urban water per day than other persons. The water supply to the city came from two deep-ground water sources, and two major water breaks took place on the 23 and 26 December, following the beginning of the outbreak, yet prior to its principal peak.

4.4. Enteroinvasive (EIEC)

Most sickness is suggested to be food- or waterborne, even if individual to individual diffusion as well happens. Nonetheless, at most one outbreak of invasive EIEC because water has been noticed in the literature and this was some 50 years ago [1].

5. Antimicrobial Monitoring

For *E. coli* 0157, the concentrations of chlorine (Cl_2) usually observed in water

have been proved to be enough for its demobilization [63] [64]. Nonetheless, researchers [65] found that Cl_2 (5 mg/L) and ozone (O_3) at (22 - 24 mg/L) possess modest killing impact on *E. coli* O157: H7 [1].

Cheswick *et al.* [66] examined the performance of Cl_2 disinfection for the first time over a range of disinfection conditions employing flow cytometry to furnish new insights into disinfection methods. Demobilization was followed for pure culture bacteria (*E. coli*) and pathogens in real treated water from operational water treatment works (WTWs). A dose dependent increase in demobilization rate (k) was noted for both test matrices, with values of 0.03 - 0.26 and 0.32 - 3.14 L/mg min for the WTW bacteria and *E. coli*, respectively. Following 2 min, *E. coli* was decreased by 2 log for all Cl_2 doses (0.12 - 1.00 mg/L). For the WTW filtrate microbes, following 2 min log reductions were between 0.54 and 1.14 with augmenting Cl_2 injection, reaching between 1.32 and 2.33 after 30 min. A reduction in disinfection performance was detected as temperature decreased from 19˚C to 5˚C for both microbial populations. Concerning chlorination at varying pH (pH 6, 7, 8), membrane demolition was more significant at higher pH. This was not consistent with the higher disinfection performance observed at lower pH when culture based methods are utilized to estimate microbial reductions [66].

With chloramines, *E. coli* O157: H7 is known to possess a CT level (C = disinfectant concentration (mg/L); T = time in min) of around 9.2 mg-min/L. Such value is required to reach a 4 $\log_{10}$ degree of demobilization [67]. Elevated temperatures have been proved to be efficient in killing *E. coli* [68] [69]. Regardless of this, EHEC has been observed to be fit to develop across a wide temperature span [1].

Radiofrequency power and UV light subjection have been notified to block *E. coli* [70]. Scientists [71] depicted that at 12 J/m^2, a 6-log reduction in culturability of *E. coli* O157: H7 happened. Nonetheless, resistant strains of *E. coli* O25: K98: NM requested bigger degrees of UV light for demobilization [1].

Researchers [72] tested potential merits of consecutive disinfection to dominate *E. coli* under circumstances of potable water distribution systems. They treated biofilms developed in polycarbonate and cast-iron reactors with Cl_2, chlorine dioxide (ClO_2) and monochloramine single or in integration with UV. Most important killing took place with the integration treatments with UV. Most importantly, chloramine was proved to be efficient in neutralizing *E. coli* in the effluent, yet not in the biofilm. The influence of Cl_2 on the growth phase of *E. coli*, in matter of its vulnerability to Cl_2, has established Cl_2 to be less performant when *E. coli* is in the stationary phase (juxtaposed with initial lag and exponential growth phase). This has been considered as a significant thought in wastewater treatment technique with changing solids retention times [1] [73].

The growing demand to decrease Cl_2 use and dominate (DBPs) augmented the search of fresh procedures in wastewater disinfection. For example, organic peracids are more and more attracting attention in killing pathogens as an encouraging alternative to Cl_2 and Cl_2-founded chemicals. Pironti *et al.* [74] eva-

luated the antimicrobial characteristics towards *E. coli* and *Staphylococcus aureus* of a fresh organic peracid, permaleic acid (PMA) juxtaposed with the reference peracetic acid (PAA). PMA presented a 10- and 5-fold reduction in the microbial inhibitory concentration level toward *E. coli* and *S. aureus* respectively, juxtaposed to PAA. Trials demonstrated higher performance of PMA concerning wastewater and treated wastewater disinfection at low dosages. PMA was more performant than PAA to avoid the regrowth of planktonic cells of *S. aureus* and *E. coli*. Therefore, PMA may be utilized as a potential alternative to the presently employed disinfection agents [74].

Even if pulsed UV (PUV) technique is adopted commercially for disinfection inside the food packaging industry, it is not used in the water/wastewater field [75]. Fitzhenry *et al.* [75] estimated the performance of PUV for disinfecting water disinfection below flow-through conditions. They employed *E. coli*, *S. aureus* and *Listeria innocua* to examine the capacity for photoreactivation and/or dark repair post PUV flow-through disinfection. Bacterial demobilization via flow-through PUV is a function of energy output with *E. coli* showing greatest sensitivity to PUV application (5.3 $\log_{10}$ demobilization following application at 1539 mJ/cm^2, output in UV range < 300 nm); *L. innocua* presented the highest PUV resistance (3.0 $\log_{10}$ demobilization following application at 1539 mJ/cm^2, output in UV range < 300 nm) below identical treatment circumstances. Greater photoreactivation took place at lower PUV outputs for both *S. aureus* and *E. coli* following flow-through PUV treatment. Therefore, exposure of inactivated bacteria to natural light, immediately post flow-through PUV treatment, must be averted to decrease photoreactivation. The LPUV proved demobilization of all microbes below the limit of detection (1 colony-forming unit (CFU)/mL) and inhibited the presence of photoreactivation.

He *et al.* [76] suggested a solar-light-driven magnetic photocatalyst, reduced-graphene-oxide/Fe,N-TiO_2/Fe_3O_4@SiO_2 (RGOFeNTFS), for the photocatalytic disinfection of various strains of bacteria: gram-negative *E. coli* and *Salmonella typhimurium*, and gram-positive *Enterococcus faecalis* (**Figure 2**). Gram-positive *E. faecalis* was observed to be more vulnerable to photocatalytic disinfection and showed a higher leakage of intracellular components than the two gram-negative bacteria. The interactions between the bacteria and RGOFeNTFS were examined for Zeta potential, hydrophilicity and scanning electron microscopy (SEM). The opposite surface charges of the bacteria (negative Zeta potential) and RGOFeNTFS (positive Zeta potential) contribute to their interactions. With a more negative Zeta potential (than *E. coli* and *E. faecalis*), *S. typhimurium* interacts more strongly with RGOFeNTFS and is mostly attacked by $^{\bullet}OH$ near the photocatalyst surface. With less negative Zeta potentials, *E. coli* and *E. faecalis* interact less strongly with RGOFeNTFS, and compete for the dominant reactive species ($^{\bullet}O_2^-$) in the bulk solution. Thus, the co-existence of bacteria significantly inhibits the photocatalytic disinfection of *E. coli* and *E. faecalis*, but insignificantly for *S. typhimurium*. Furthermore, photocatalytic disinfection employing RGOFeNTFS is

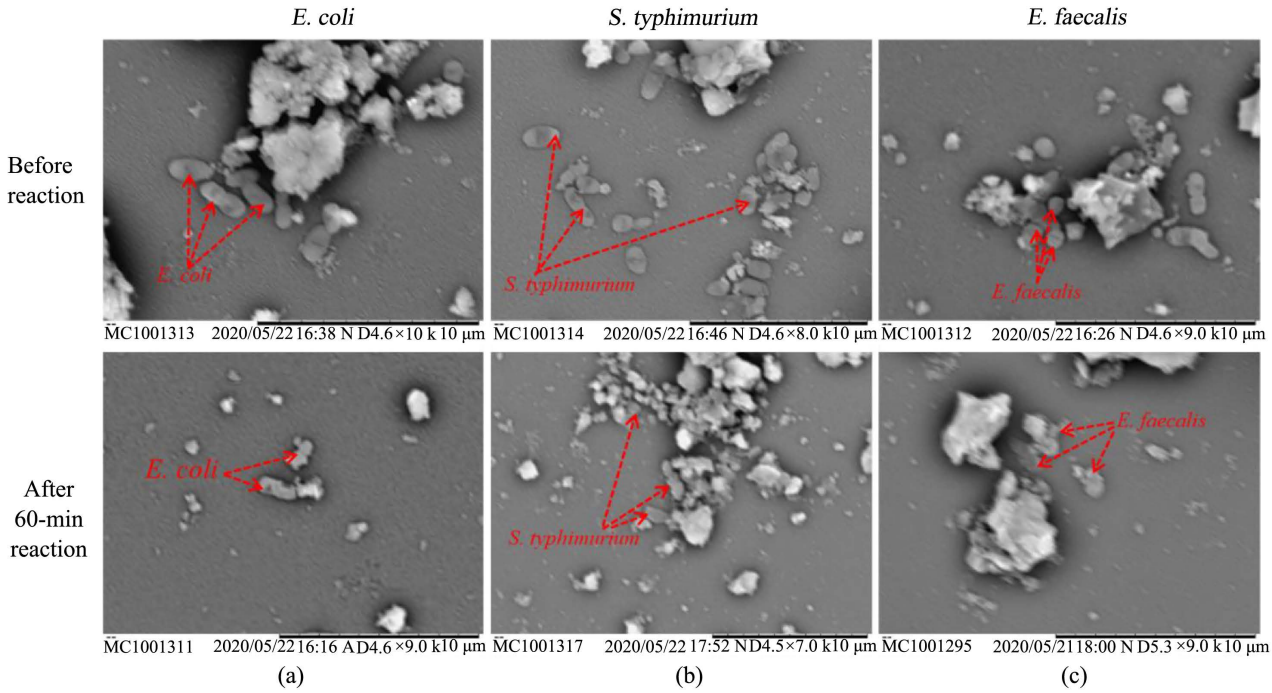

Figure 2. Scanning electron microscopy (SEM) images of (a) *E. coli*; (b) *S. typhimurium*; (c) *E. faecalis* after 60 min of photocatalytic disinfection using RGOFeNTFS [76].

promising for dealing with real sewage, and various bacteria are killed simultaneously [76].

Suggesting narrow-band mercury-free light sources, like light emitting diodes (LEDs) and excilamps, has motivated investigation on killing pathogens via dual-wavelength light radiation [77]. Indeed, dual-wavelength light radiation is considered as a developed instrument for improving microbial demobilization in water in view of potential synergistic effect. Matafonova and Batoev [77] focused on its pathways under dual-wavelength light exposure and discussed some related references in terms of yes-or-no synergy. They suggested three fundamental demobilization pathways, which work in the estimated spectrum ranges I (190 - 254 nm), II (250 - 320 nm) and III (300 - 405 nm) and furnish a synergistic effect when combined. Such pathways implicate proteins damage and deoxyribonucleic acid (DNA) repair suppression (I), direct and indirect DNA damage (II) and generation of ROSs via endogenous photosensitizers (III), like porphyrins and flavins (**Figure 3**). A synergy under dual-wavelength light irradiation simultaneously or sequentially takes place if coupling two wavelengths of different ranges (I + II, I + III, II + III) in order to trigger various demobilization pathways. New progresses of dual-wavelength light strategy in photodynamic therapy can be applied for water disinfection. They open perspectives for using the sources of near-UV and visible radiation and making the disinfection techniques more energy- and cost-effective. In such context, the synergistically efficient dual-wavelength combinations II + III and the combinations within the extended to 700 nm range III (near-UV + VIS) seem to be encouraging for

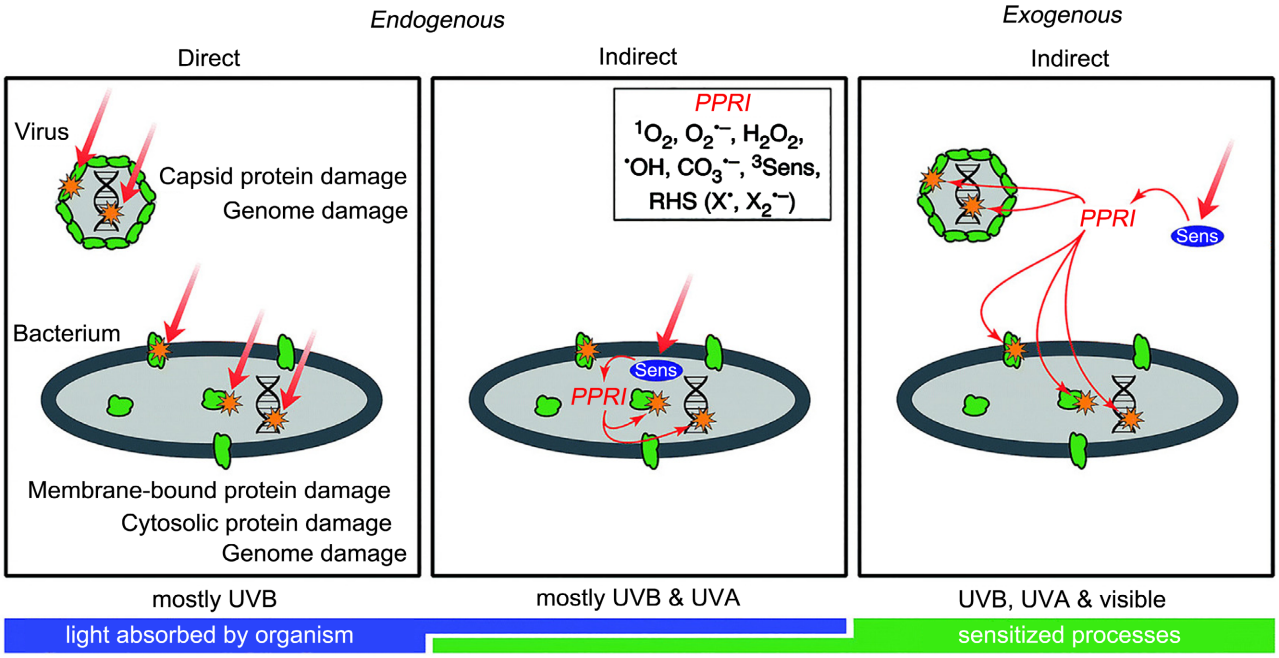

Figure 3. Conceptual model of sunlight inactivation pathways in viruses and bacteria. For direct mechanisms, the photon is absorbed by a chromophore at the site of damage (orange star). For indirect pathways, the photon is absorbed by a sensitizer (Sens), and damage (orange star) occurs at a different site. Green shapes represent proteins. PPRI = photo-produced reactive intermediates [77].

developing fresh advanced oxidation processes for disinfection of real turbid waters [77].

In rural communities, developing communities with low quality centralized water distribution, portable water purification devices are requested to furnish potable water. As mentioned above, filtration, UV light, or chemical injections present tools to eliminate microorganisms from water. Montenegro-Ayo *et al.* [78] suggested a small portable photoelectric point-of-use device that uses a commercial teacup from which TiO_2 nanotube photoanodes were formed *in-situ*. With a small rechargeable battery powered 365 nm LED, the apparatus was found to reach 5-log demobilization of *E. coli* in 10 s and 2.6-log of *Legionella* in 60 s of treatment in model water samples (**Figure 4**). Dealing with natural water attained a 1-log bacteria demobilization after 30 s thanks to matrix effects.

For killing pathogens, hydrodynamic cavitation seems to be an encouraging technology. Sun *et al.* [79] investigated the disinfection properties of an advanced hydrodynamic cavitation reactor (ARHCR) in pilot scale. They examined the impacts of different flow rates (1.4 - 2.6 m^3/h) and rotational speeds (2600 - 4200 rpm) on killing *E. coli*. A disinfection rate of 100% was attained in only 4 min for 15 L of simulated effluent under 4200 rpm and 1.4 m^3/h, with energy efficiency at 0.0499 kWh/L. The morphological changes in *E. coli* were studied by SEM (**Figure 5**). The ARHCR may conduct to serious cleavage and surface damages to *E. coli*. They suggested a likely damage route of the ARHCR, comprising both the hydrodynamical and sonochemical impacts (**Figure 6**).

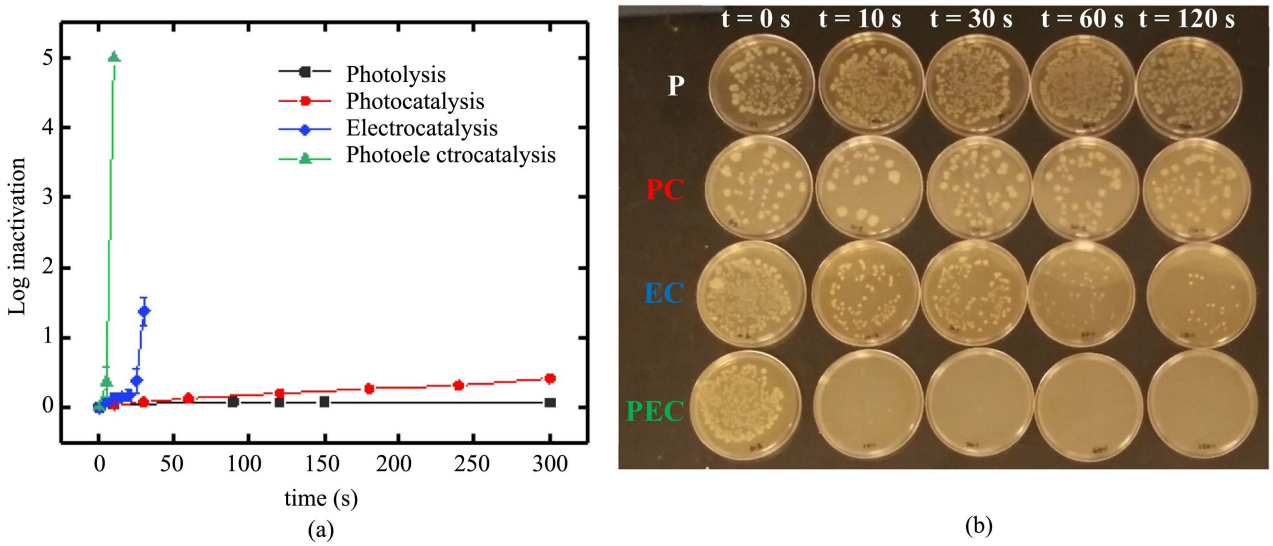

Figure 4. *E. coli* inactivation utilizing the portable photoelectric point-of-use device in (■) photolysis, (●) photocatalysis, (◆) electrocatalysis, and (▲) photoelectrocatalysis mode. (a) Logarithm of demobilization and (b) the plating assays to evaluate *E. coli* CFUs. The initial *E. coli* concentration was 1×10^5 CFU/mL [78].

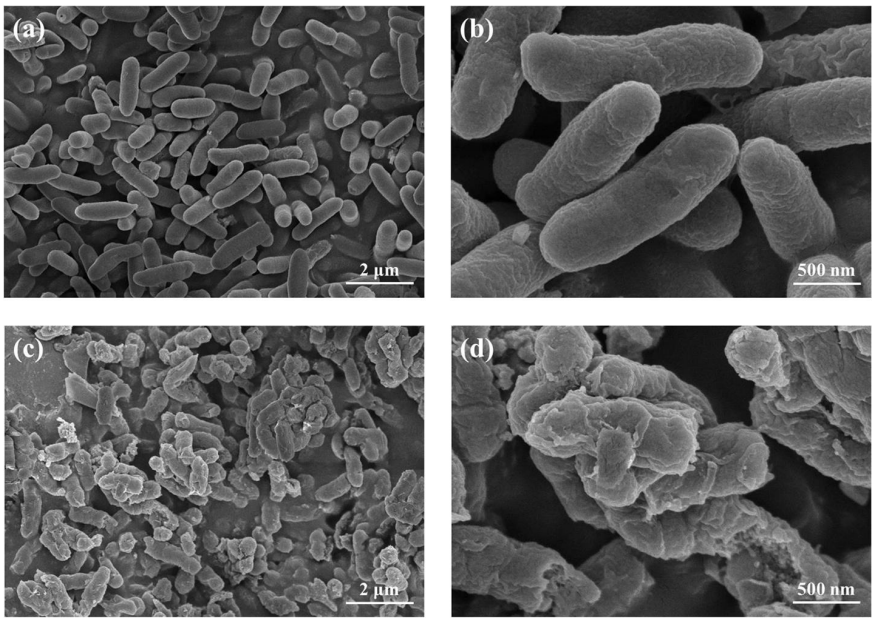

Figure 5. Scanning electron micrographs of *E. coli* cells before ((a) 0 min (×10,000) and (b) 0 min (×40,000)) and after ((c) 10 min (×10,000) and (d) 10 min (×40,000)) the ARHCR treatment at 3800 rpm and 1.4 m^3/h [79].

Antibiotic-resistant bacteria (ARB) form a dangerous threat to public health [80]. As a low energy consumption and environmentally-friendly technology, electrochemistry seems to be appropriate for killing ARB. Liu *et al.* [80] investigated the suitability of electrochemical disinfection (ED) for demobilizing ARB (*E. coli* K-12 LE392 resistant to kanamycin, tetracycline, and ampicillin) and the regrowth probability of the treated ARB. They depicted that 5.12-log ARB reduction was reached within 30 min of applying molybdenum carbide as the anode

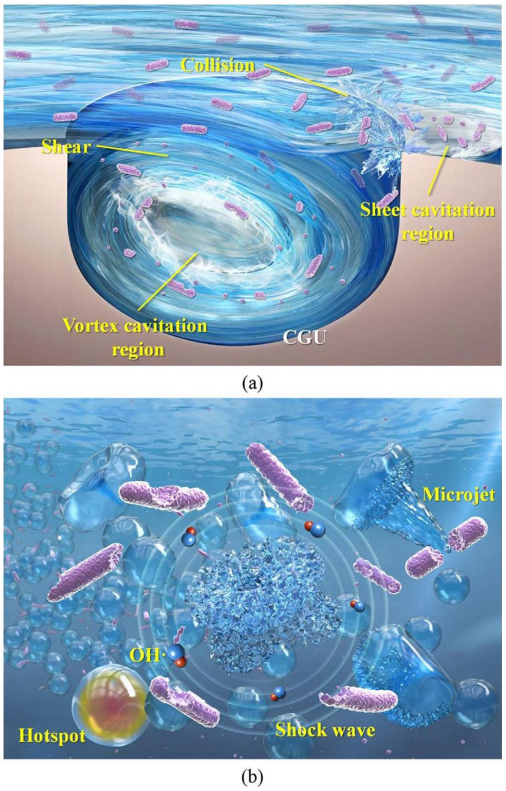

Figure 6. Likely disinfection mechanism of the ARHCR: (a) hydrodynamical and (b) sonochemical effects [79].

and cathode material under a voltage of 2.0 V. No ARB regrowth was noted in the cathode chamber after 60 min of incubation in unselective broth, showing that the technique in the cathode chamber was more efficient in demobilizing ARB permanently. The pathways underlying the ARB demobilization were verified founded on intercellular ROSs measurement, membrane integrity detection, and genetic damage assessment. Higher ROSs generation and membrane permeability were detected in the cathode and anode groups ($p < 0.001$) compared to the control group (0 V). Furthermore, the DNA was more likely to be damaged throughout the ED application. Such investigation establishes once again that ED is an encouraging process for disinfecting water to avert the diffusion of ARB (**Figure 7**).

Xia *et al.* [81] presented a new process founded on piezoelectric catalytic persulfate (PS) activation for water advanced disinfection. They synthesized and used silver modified barium titanate (Ag-BTO) as a piezoelectric catalyst to activate PS under ultrasonic (US) vibration for demobilizing *E. coli*. The suggested US/Ag-BTO/PS method reached a 6.2 log demobilization within 5 min and 20 min, respectively, for *E. coli* at culturable state and viable but nonculturable state (**Figure 8**). The important performance of *E. coli* reduction was related to the successive formation of hydroxyl radicals and sulfate radicals via PS activation by piezo-catalytically formed electrons and superoxide radicals. A synergism between ultrasonication and radical oxidation was observed by employing the

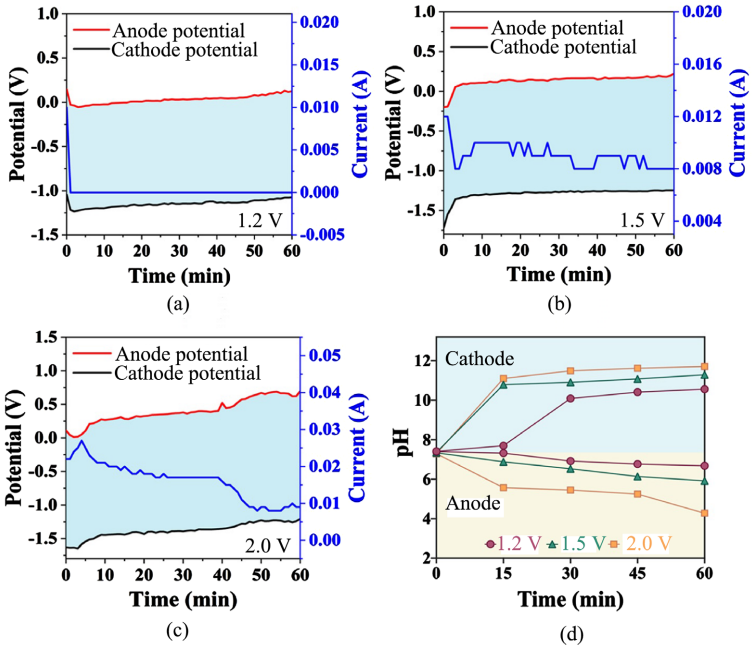

Figure 7. Variations of the current, potential and pH levels in the two-chamber apparatus below various voltages (1.2, 1.5, and 2.0 V) throughout electrochemical disinfection (ED) with Mo_2C employed as the anode and cathode. (a): Current and potential below 1.2 V voltage; (b): Current and potential below 1.5 V voltage; (c): Current and potential below 2.0 V voltage; (d): pH levels in the two-chamber apparatus below various voltages (1.2, 1.5, and 2.0 V) [80].

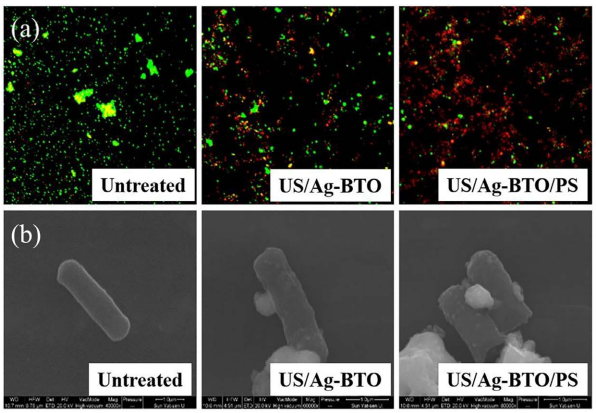

Figure 8. (a) Fluorescence microscopic images and (b) Scanning electron microscopy (SEM) images of *E. coli* K-12 untreated *E. coli* and after the treatment in the US/Ag-BTO and US/Ag-BTO/PS systems for 5 min.

US/Ag-BTO/PS method for *E. coli* demobilization. The ultrasonication disrupted cell membrane of *E. coli*, accelerated the permeation of the radicals and enhanced the following inner metabolic dysfunction and enzyme oxidation by radicals [81].

Concerning *E. coli* diarrhoeal contagions, fluid and electrolyte correction is obligatory [1]. Extraintestinal *E. coli* contagions are, yet, treated with antibiotics. If such procedure of treatment is used, it must be admitted that *E. coli* reveal in-

trinsic resistance to benzylpenicillin. *E. coli* remain usually vulnerable to the antibiotics (ampicillin, tetracycline, aminoglycosides, trimethoprim and the cephalosporins). Nonetheless, due to the broad diffusion proof of antibiotic resistance being acquired by plasmid transfer, there is growing numbers of *E. coli* resistant to streptomycin and tetracycline. For such cause, antibiograms must be realized, mostly for epidemiological objectives. Employing antibiotics in dealing with *E. coli* 0157 remains controversial because of the trouble of growing numbers of *E. coli* 0157 with antibiotic resistance. Most important is the augmenting propagation of extended-spectrum β-lactamase (ESBL) producing *E. coli* [82]. ESBL generating *E. coli* show resistance to the majority of the *b*-lactam antibiotics, comprising penicillins, monobactams and most cephalosporins. The explanation for this stays the occurrence of plasmid-encoded enzymes, which hydrolyze the β-lactam antibiotics [1]. Such microbes are more and more involved in provoking hospital, as well as community-acquired contagions and when existing, tend to be treated with carbapenems, since these remain active against many ESBL-*E. coli* [83]. However, resistance of *E. coli* to carbapenems is as well as emerging [84] [85] [86].

Antibiotics that have historically been active against *E. coli* have comprised sulfonamides, tetracyclines, aminoglycosides (comprising gentamicin and amikacin), chloramphenicol, semi-synthetic penicillins, cephalosporins, β-lactamase-inhibitor combinations, carbapenems and fluoroquinolones [87] [88]. Notwithstanding all such antibiotics being standard therapies for *E. coli*, there has been much worry concerning the fast expansion of resistance [89] [90]. Fundamental pathways of resistance against such antibiotics have comprised exclusion and efflux of the agents from the bacterial cell, acquisition of resistance genes comprising those that generate enzymes fit to decompose β-lactamas, carbapenemases, and aminoglycoside. As aforesaid, extended-spectrum β-lactamases (ESBLs) are plasmid-mediated enzymes that may decompose recent analogues of cephalosporins. It has been assessed in the UK that of all 10% of *E. coli* bacteraemias are linked with ESBL strains and 90% of these are CTX-M type [91].

Relating to antibiotic resistance amongst environmental isolates, scientists [92] fixed the minimum inhibitory concentrations for 241 *E. coli* isolates recuperated from water, sediment and biofilms in an intensive agricultural watershed (Elk Creek, British Columbia, Canada) between 2005 and 2007. Such isolates had an elevated frequency of resistance to tetracycline, ampicillin, streptomycin and nalidixic acid [1].

6. Conclusions

This work firstly defined microbiologically *E. coli* bacteria and presented a brief history relating to their first discovery and following contagions. To understand *E. coli*'s behavior, a short description relating to their metabolism and physiology is given. As humankind is concerned by *E. coli* contagion, their clinical characteristics are discussed besides their subsistence in nature. A general examina-

tion concerning different techniques used for controlling such bacteria is finally presented. The main points drawn from this work may be listed as below:

1) In terms of health effects (*i.e.*, the occurrence of disease, degree of morbidity and mortality): a) the pathogenic *E. coli* serotypes are categorized following their route of producing symptoms (The contagious dose of most pathogenic *E. coli* is elevated, ranging from 10^5 to 10^{10} organisms) [1]. b) The six groups are enteropathogenic (EPEC), enterotoxigenic (ETEC), verocytotoxigenic (VTEC; comprising enterohaemorrhagic, EHEC), enteroinvasive (EIEC), enteroaggregative (EAEC) and diffusely adherent. c) All pathogenic *E. coli* provoke diarrhea at different levels of gravity. d) EPEC: traveler's diarrhoea, enteritis in infants; ETEC: traveler's diarrhoea, diarrhoea, vomiting and fever; VTEC: *Shigella*-like dysentery (stools with blood and mucus) and haemolytic uremic syndrome (HUS); diffusely adherent: diarrhoea in children. e) *E. coli* induces urinary tract infections (UTIs) and may provoke sepsis and meningitis in neonates. f) Pathogenic *E. coli* strains lead to the majority of childhood diarrhoea. g) The level of morbidity and mortality changes following the strain and the host's properties. In poor countries, diarrhoeal illness is much more probably to conduct to the dangerous disease and death. In rich countries, even if childhood diarrhoea is less serious, contagion with verocytotoxigenic *E. coli* may lead to HUS and thrombiotic thrombocytopaenia purpura. Such circumstances could create acute kidney failure and death [1].

2) Conventional water treatments employ high energy-intense mercury lamps or chlorine injection, which remain neither a feasible nor economically viable methods for vulnerable populations in developing areas [78]. Such competitive techniques may be overcome by a more affordable and off-grid method like a device that is founded on TiO_2 photoelectrocatalytic disinfection concepts. Applying photoelectrocatalytic processes in scaled-down and portable devices authorizes performant water disinfection when using an off-grid point-of-use system. Employing LED sources guarantees working with low energy consumption. This furnishes interesting alternatives to traditional disinfection treatments [78].

3) A pilot-scale ARHCR was tested for water disinfection, and a new probable disinfection route of the ARHCR was proposed comprising hydrodynamical and sonochemical effects. The ARHCR could be used as an encouraging alternative or complementary tool for water disinfection as well as other process intensifications. More research on the disinfection route, structural optimization, and scale up remains required in the future [79].

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Conflicts of Interest

The authors declare no conflicts of interest.

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Abbreviation

ARB	Antibiotic-Resistant Bacteria
ARHCR	Advanced Hydrodynamic Cavitation Reactor
CFU	Colony-Forming Unit
DAEC	Diffuse adherent *E. coli*
DBPs	Disinfection by-Products
DNA	Deoxyribonucleic Acid
EAEC	Enteroaggregative *E. coli*
ED	Electrochemical Disinfection
EHEC	Enterohaemorrhagic *E. coli* O157: H7
EIEC	Enteroinvasive *E. coli*
EPEC	Enteropathogenic *E. coli*
ESBL	Extended-spectrum β-Lactamase
ETEC	Enterotoxigenic *E. coli*
HUS	Haemolytic Uremic Syndrome
k	Demobilization rate (k)
KIA	Kligler's Iron Agar
LEDs	Light Emitting Diodes
ONPG	Ortho-Nitrophenyl-β-D-Galactoside
PAA	Peracetic Acid
PMA	Permaleic Acid
PPRI	Photo-Produced Reactive Intermediate
PUV	Pulsed Ultraviolet
RGOFeNTFS	Reduced-Graphene-Oxide/Fe,N-TiO_2/Fe_3O_4@SiO_2
ROSs	Reactive Oxygen Species
SEM	Scanning Electron Microscopy
SODIS	Solar Water Disinfection
TSI	Triple Sugar iron
US	Ultrasonic
UTIs	Urinary Tract Infections
UV	Ultraviolet
VTEC	Verocytotoxigenic *E. coli*
WTW	Water Treatment Works