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Dioxins and Furans: Emerging Contaminants of Air

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<http://dx.doi.org/10.5772/intechopen.80680>

Abstract

Dioxins and furans are classified highly contaminating toxic chemicals having serious effect on human health. This chapter begins with a brief summary on the formation, occurrence and toxicity of polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzofurans (PCDFs) with their potential health effects, mitigation measures of these harmful compounds. Depending on position of chlorine atoms on aromatic rings, about 210 chemically different PCDD/PCDFs known as “congener” are present in the environment. The expected biological activity of PCDD/PCDFs is expressed relatively to the activity of 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD) using a common metric. TCDD is the most biologically potent among all the congeners. Toxic level of a mixture of PCDD/PCDFs is therefore expressed in TCDD toxicity equivalents or TEQs. There are two mechanisms for the formation of dioxins and furans, one from precursors and other by de novo synthesis. PCDD/PCDFs followed a mechanism which uses macromolecule carbon and chlorine in fly ash to form dioxins at low temperature. There are various sources of the formation of PCDD/PCDFs like hospital waste incinerators, industrial combustion and burning of domestic waste. Dioxins and furans have very harmful effects on the human health causing cancer, diabetes, neurotoxicity, immunotoxicity and chloracne. It has been experienced that hybrid method secure a sustainable future for the incinerators and PCDD/PCDFs removal technologies. This chapter will help the researchers and practitioners for better understanding and decision making for future research to establish a sustainable PCDD/PCDFs free life.

Keywords: dioxin, furan, contaminant, toxicity, incineration, mitigation

1. Introduction

Emerging contaminants are those which were not considered as such as in previous times, but exist in the environment on global level. They are common derivatives of municipal,

agricultural and industrial sources and pathways. A considerable rise has been observed in terms of emission of air pollutants in the atmosphere [1, 2]. Environmental quality may be at risk by developmental activities as imbalance in the composition of air. Persistent organic pollutants (POPs) are regarded as very harmful compounds because they are resistant to various factors of biochemical and photolytic degradation. POPs are persistent to soils, sediments, and air for several decades [3]. Having high toxicity and long persistency in the environment they accumulate in the fatty tissues of humans and animals resulting into many behavioral, reproductive and developmental changes [4, 5]. POPs have gained global attention due to their transportation over long distances from the source. Various researchers proved that persistent organic pollutants (POPs) are dangerous compounds due to their persistent, bio accumulative and toxic characteristics. POPs include pesticides, polychlorinated biphenyls, polycyclic aromatic hydrocarbons (PAHs), polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), and groups of brominated flame retardants [6]. In Stockholm convention on POPs it was decided to control the concentration of the dirty dozen in the environment. PCDDs and PCDFs are members of dirty dozen which are classified as toxic and carcinogenic, found in very small amounts in the environment. The major issue of PCDDs/PCDFs is due to their extreme persistency in the environment causing toxicity and cancer to living organisms and can potentially cause cancer. Dioxin and furans are family of chlorinated hydrocarbon compound which are categorized into three main classes as: polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and dioxin-like polychlorinated biphenyl (DL-PCBs). PCDDs and PCDFs are produced from different anthropogenic activities like forest fires, domestic and hospital waste incineration [7]. They are by products of the synthesis or combustion of chlorine based compounds that include some of the most toxic chemical substrates.

Polychlorinated dibenzo-p-dioxins (PCDD), polychlorinated dibenzofurans (PCDF) are produced accidentally due to inadequate combustion, as well as during the manufacturing and formulation of chlorinated pesticides and other substances. They are also emitted from the incineration of hospital, municipal and hazardous waste. There are seventy many dioxins, out of which seven are considered most toxic to humans, aquatic and terrestrial organisms, causing congenital mental retardation (endocrine disrupting) and physical disorders. Many industrial processes are likely to generate a huge amount of industrial waste, which is openly burned without any safety measure, producing huge quantity of Polychlorinated dibenzo-p-dioxins (PCDD) and polychlorinated dibenzofurans (PCDF).

The polychlorinated dibenzo-p-dioxins (PCDDs) and dibenzofurans (PCDFs) are two groups of tricyclic, planar, aromatic, nonpolar, poorly water soluble, lipophilic and stable chemicals [8]. Out of 210 congener of these chemicals 17 are more toxic. These are toxic carcinogenic unintentional by-products which are found in lesser concentration in environment, reflecting drastic effect due to their extreme persistency. Their persistency is due to their long half-life of 10–20 years roughly [7].

1.1. Environmental concentration

Emission favors the formation, environmental release and distribution of these congeners of TCDDs and TCDFs signatures of the types of dioxin and furans associated with particular

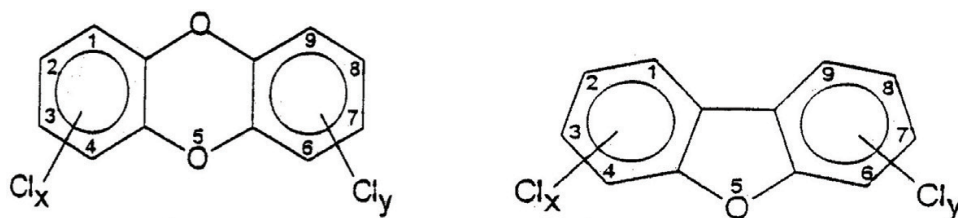


Figure 1. General structures of dioxin and PCDDs (left) and furan and PCDFs (right).

environmental sources of these compounds [9]. On the contrary, the PCDD/Fs in the ambient air samples, characterized by the abundance 76% in some parts of the world and it is dominated by 1,2,3,4,6,7,8-HpCDF (40%) followed by OCDD (10%), OCDF (6%), and 1,2,3,4,6,7,8-HpCDD (11%). Reported values not similar to any of the profiles reported by Cleverly; however, it reveals the simultaneous occurrence of two potential emission sources of TCDDs and TCDFs [9].

1.2. Dioxins

Dioxins chemically consist of two benzene rings, connected by a pair of oxygen atoms. Each of the eight carbon atoms on the rings that are not bonded to oxygen can bind with hydrogen atoms or atoms of other elements. The more toxic dioxin is one having chlorine atoms at the 2, 3, 7, and 8 positions. This isomer 2,3,7,8-TCDD is chemically most stable and toxic compound. Being Water insoluble dioxin is not diluted with rain water and remain in the soil thus penetrate and accumulate in fatty tissues. The molecular structure of dioxins can be determined by single crystal X-ray diffraction crystallography. The structure of TCDD in crystal which is also observed in molecular crystal of poly aromatic hydrocarbon (PAHs) [10].

1.3. Furans

Furans are also toxic organic compounds which are colorless, highly flammable and very low boiling point almost equal to room temperature. Furans are heterocyclic compound containing one oxygen atom, four carbon atoms and a 5-membered ring (**Figure 1**). The global environmental impact of volatile compounds depends on many factors such as air pressure, temperature and also the weather and terrain features which affect the deposition. It is interesting to know what derivatives are formed from reactions with furan, and also how fastly these potential products are removed from the environment. One of the 5 largest sinks of furan derivatives will probably be reaction with the hydroxyl radical (OH), which mostly initiates reactions with VOCs through hydrogen abstraction from a C-H bond.

2. History of dioxins and furans

The environmental distribution of dioxins and furans is a function of transport and source. The sources of dioxins and furans are combustion, soil deposition, volatilized and transported particulates which were sequestered and rereleased into the environment. It is evident that

environmental dispersal and accumulation of these compounds is not necessarily depending upon a nearby source. Polychlorinated dibenzo-p-dioxins (PCDDs) and their cousins, the polychlorinated dibenzofurans (PCDFs), are notorious environmental contaminants. Depending on the position of chlorine atoms attached on ring, two hundred and ten chemically different toxic compounds of dioxins and furans are produced each of which is known as “congener” [11]. Collectively these congeners or compounds are often known as “dioxins and furans”. These compounds have received considerable public and scientific attention because of their acute toxicity. Out of all these congeners, 2,3,7,8-TCDD has lowest known LD₅₀ values. It takes only 0.6 µg/kg of body weight to kill male guinea pigs [12]. In 1957, a strange disease killed millions of young chickens in the eastern and mid-western U.S. and symptoms of this disease were excess of fluid in the heart sac and abdominal cavity, chased to the fatty acids that had been added to the chicken’s feed. Efforts of several years lead to the isolation of one of the identified toxic chemical [13] by X-ray crystallography; it was 1,2,3,7,8,9-hexachlorodibenzo-p-dioxin. In the 1960s and early 1970s, the Northeastern Pharmaceutical and Chemical Company (NEPACCO) established a plant in Veron for manufacturing of hexachlorophene from 2,4,5-trichlorophenol and formaldehyde. Unfortunately, 2,3,7,8-TCDD was an impurity in the 2,4,5-trichlorophenol starting material used in this process; thus, the hexachlorophene product needed to be purified before sale. Because of its neurotoxicity, the U.S. Food and Drug Administration restricted the use of hexachlorophene in 1971. In 1977 Olie et al. investigated dioxins which existed in fly ash from an industrial heating facility [14]. In 2000, Bumb et al. in a famous paper “Trace chemistries of fire: A source of chlorinated dioxins”, exposed that dioxins were exist in particles from the combustion of organic material, involving the combustion of municipal and chemical waste. This was marvelous discovery, suggested that “dioxins have been with us since the advent of fire” [15, 16]. In mid of 1970, a Swiss company established a chemical plant for manufacturing of 2,4,5-trichlorophenol by the reaction of 1,2,4,5-tetrachlorobenzene with NaOH. Unfortunately, accident occurred and chemicals from vessels were released and transported by wind. This caused a lot of disaster to plants, animals and human. Later on it was confirmed that the reason of this disaster was a notorious chemical 2,3,7,8-TCDD [17]. During war in Vietnam, US military showered Agent Orange from 1965 to 1971 as defoliant to kill food crops. Agent Orange was a mixture of n-butyl esters of 24-D and 245-T, the latter of which was formed from 2,4,5-trichlorophenol. Agent Orange was contaminated with small amounts of 2,3,7,8-TCDD [18]. Dioxins and furans including dioxin-like polychlorinated biphenyls collectively as known as DLCs. These organic compounds are highly toxic and accumulate, through the food chain, into the lipid component of animal foods. In another study it has been observed that levels of these compounds in the environment are declining since 1970s [19]. Highly exposed groups of dioxins and furans are found in breastfeeding infants, fishers and workers of cement industries. Although Dioxins and furans have been extensively studied as a contaminant, but still a great deal of research is needed regarding their potential toxicity.

3. Toxicity of dioxins and furans

The toxicity and exposure preferably depends on the composition and particle size of the mixtures containing toxic compounds. Biological activity of mixtures of dioxins and furans is desirable to express the common effect. Literature review reports the biological activities

CAS number	Hazardous substance	Mammalian TEF	Avian TEF
<i>Dioxins</i>			
1746-01-6	2,3,7,8-Tetrachloro dibenzo-p-dioxin	1	1
40321-76-4	1,2,3,7,8-Pentachloro dibenzo-p-dioxin	1	1
39227-28-6	1,2,3,4,7,8-Hexachloro dibenzo-p-dioxin	0.1	0.05
57653-85-7	1,2,3,6,7,8-Hexachloro dibenzo-p-dioxin	0.1	0.01
19408-74-3	1,2,3,7,8,9-Hexachloro dibenzo-p-dioxin	0.1	0.1
35822-46-9	1,2,3,4,6,7,8-Heptachloro dibenzo-p-dioxin	0.01	<0.001
3268-87-9	1,2,3,4,6,7,8,9-Octachloro dibenzo-p-dioxin	0.0003	0.0001
<i>Furans</i>			
51207-31-9	2,3,7,8-Tetra chloro dibenzofuran	0.1	1
57117-41-6	1,2,3,7,8-Pentachloro dibenzofuran	0.03	0.1
57117-31-4	2,3,4,7,8-Pentachloro dibenzofuran	0.3	0.1
70648-26-9	1,2,3,4,7,8-Hexachloro dibenzofuran	0.1	0.1
57117-44-9	1,2,3,7,8-Hexachloro dibenzofuran	0.1	0.1
72918-21-9	1,2,3,7,8,9-Hexachloro dibenzofuran	0.1	0.1
60851-34-5	1,2,3,4,6,7,8-Heptachloro dibenzofuran	0.01	0.01

Table 1. TEQ values for mammals and Avians (EPA, 2000).

of the various dioxin and furans congeners comparing with of 2,3,7,8-tetrachloro-dibenzo-p-dioxin (TCDD). Information has been gathered for this dioxin and furan congener, and it is found that TCDD is the most biologically toxic among the mixtures of dioxins and furans and highly potent. The toxicity of TCDD/TCDFs is therefore expressed in toxicity equivalents (TEQs) of highly potent component as TCDD (**Table 1**). As an example, exposure to a matter a potency of 2.0 ng TEQ/kg means that matter has the potency equal to 2.0 ng TCDD/kg. Toxicity equivalents values for various materials are calculated by multiplying the mass or concentration of each component by a TEF and adding all present components. The biological activities of TCDD/TCDFs vary depending on the human's exposure. Even so, public concern persists regarding food supply and adverse outcomes to TCDD/Fs exposure, especially in highly exposed populations. Sensitive population includes fetuses and new born infants. These populations may be at increased risk due to exposure through foods. However, many foods which are sources of dioxins and furans are also sources of important nutrients, such as calcium (Ca) and vitamins (A E C), protein, iron and fish.

4. Chemical formation and sources of dioxins and furans

PCDDs and PCDFs are not produced intentionally but as by product of various processes like chlorinated phenols, PCBs, phenoxy herbicides, chlorinated benzene, chlorinated aliphatic compounds, chlorinated catalysts and halogenated biphenyl ethers **Table 2** [20, 21]. There are

Equation no.	Reaction steps	Rate constants
R1	$P=P. +H$	$10^{15.5} \exp[-86,500/(RT)]$
R2	$P + OH = P. + H_2O$	$10^9 \exp[-86,500/(RT)]$
R3	$P. = Pr$	$10^{13.6} \exp[-57,654/(RT)]$
R4	$P + P. = PD + Cl$	$10^9 \exp[-26,000/(RT)]$
R5	$PD = D + HCl$	$10^{14} \exp[-45,000/(RT)]$
R6	$PD + OH = D + H_2O$	10^9
R7	$P. + R = P + R.$	$10^8 \exp[-26,000/(RT)]$
R8	$P. + OH = Pr$	10^9
R9	$D = Pr$	$10^{15.5} \exp[-80,000/(RT)]$
R10	$D + OH = Pr$	10^8
R11	$P. + O_2 = Pr$	10^8
R12	$R + OH = R. + H_2O$	10^9
R13	$R = Pr$	$10^{18} \exp[-90,000/(RT)]$

P = polychlorinated phenols, P. = polychlorinated phenoxy radicals, Pr = unspecified products, PD = polychlorinated 2-phenoxy phenols, D = PCDD, R = fuel molecules.

Table 2. Mechanism of PCDD/PCDFs formation according to Shaub and Tsang.

two mechanisms for the formation of PCDD/PCDFs from solid waste incinerator: formation from precursors and formation by de novo synthesis. PCDD/PCDFs followed a mechanism which uses macromolecule carbon and chlorine in fly ash to form dioxins at low temperature by involving the oxidative breakdown and conversion of macromolecular carbon structure to aromatic compound [22]. PCDD/PCDFs formed by this process have solid phase at one end and other part will desorb to gas phase and be carried by off-gas flow. Dioxin can be formed by variety of precursors like chlorobenzene and chlorophenol. Such precursors are produced by partial combustion or heterogeneous catalytic reaction on surface of fly ash [23, 24]. The examination of human tissue of earlier time showed little concentration of dioxins than today [25]. Analysis of sediment near industrial area showed that dioxin concentration is minor till 1920 [8]. However its concentration continued to increase from 1920 to 1970 [26].

4.1. Incineration sources

The most effective means of dealing with the problem is to reduce the amount of wastes generated by hospitals. Incineration is one of the key methods to reduce the amount of generated waste. But one serious drawbacks of this process is the emission of dioxins and furans in flue gas. Dioxins and furans are contaminants that are released into the environment from combustion processes. The combustion of plant material from forest, brush, and range fires contributed to preindustrial deposition of dioxins into soil, sediment, and clay. Postindustrial sources are varied and include industrial burning (e.g., steel, coke, ceramic,

and foundry), landfill fires, structural fires, utility pole and transformer storage yards, crematoriums, and backyard barrel burning of trash and woody and other plant material. Municipal solid waste incinerator produced massive amount of dioxins by following the precursors and de novo synthesis mechanism [27]. Past few decades demonstrated that formation of dioxin and dioxin like compound from municipal solid waste incinerator is about 50 ng I-TEQ/kg [28]. Hospital wastes are also treated by incineration without using high quality technologies. Thus incineration of chlorine containing product produced a lot of dioxins to atmosphere [28, 29]. Hazardous waste (explosive, oxidizing, highly inflammable, infectious, mutagenic) incinerations are responsible for the production of dioxin [30]. Solid residues produced from waste water treatment containing toxic organic pollutants are called sewage sludge. Limitation to Landfill disposal process it is also treated by incineration. Thus it also a big source of dioxins [31].

4.2. Industrial sources

Use of harmful material as fuel leads to major contribution toward dioxin and furans in clincker goes to air [32]. A number of studies of emission confirmed that wood burning is a major source of TCDD/PCDFs emission in air [33]. One of review that give appealing information is that dioxin emission from wood burning is 945 g I-TEQ per year [34]. Different researchers from Norway and Sweden have studied dioxin emission from vehicles engines combustion of fuels [35]. Waste products released from paper and pulp mills have large no of chlorinated and phenolic compounds as in wood pulps leads to emission of dioxin in water, land and paper product [36]. One report from china suggested that paper industry producing 300 pg/11-TEQ [37]. Metallurgical industries involving a no. of process like smelting operation and scrap metal recovery are major sources of dioxin and similar congeners [38]. Sintering of iron ore is also a source of these toxic congeners [39]. Annual production of dioxin in world is estimated to be 500–400 g I-TEQ.

4.3. Reservoir sources

PCDDs and PCDFs are persistent and water insoluble compounds having high tendency to accumulate in soil and sediment; vegetation, waste and in organic matter. These compounds have the ability to be recycled and redistributed in environment. Biological processes are also a source of dioxin and furans. When microorganisms catalyze chlorinated phenolic compounds then there occurs the emission of toxic compounds of dioxins and furans [40]. There are no miscellaneous sources e.g. power generation, thermal oxygen cutting metal at demolition sites, Kraft Liquor boiler, laboratory waste tire combustion, carbon activation services [41].

5. Effects of dioxins and furans

Comprehensive study has been carried out on toxicity of TCDD/PCDFs and its related compound [42]. Rodents when exposed to TCDD/Fs it lessened the reproductive capability of

female and disrupt the sperm production in male progeny. Many diseases like hypospadias, ectopic testes, vaginal pouches, agenesis of the ventral prostate, and nipple retention were noticed [43]. Exposure to TCDD leads to prevalence and complication endometriosis [44]. Dioxin and furans are well declared endocrine disruptors thus lessened the production of thyroid hormones [45–47]. Exposure of wildlife to dioxins cause many reproductive variations such as cryptorchidism in the Florida panther, small baculum in young male otters, small penises in alligators, sex reversal in fish, and altered social behavior in bird [45].

TCDD/PCDFs are recommended highly damaging to immune system and thus decreasing host resistance to infectious diseases and lowered immune responses. Dioxins also disturb production of inflammatory cytokines such as interleukin and necrosis factor [48]. TCDD/PCDFs significantly affect the neuron populations of vertebrate brain; however their damage to brain function is still not clear and need more research to reveal the truth. It is noticed that TCDD/PCDFs affect the gonadal and thyroid hormones and slow down the neural transmission network [49].

Chloracne is a skin damaging condition with both hyper keratotic and hyper proliferative responses of the epidermis is caused by the exposure of TCDD/Fs. Many animals such as cows, horses and rabbits also revealed this disease noticed [50]. In addition to these, loss of sebaceous gland and atrophy of hair follicle also noticed due the severe exposure of TCDD/PCDFs [51]. With the exposure to TCDD/PCDFs excessive keratinization may also occur in dermal epithelium [52].

Some biochemical changes also seen with exposure to congeners of TCDD/Fs [53]. It was noticed that insulin level goes down after TCDD/PCDFs massive accumulation. It is also observed TCDD/PCDFs exposure effect the body growth, deplete the energy stores and thus organism has to lower insulin level to sustain blood glucose levels. On the other side tryptophan concentrations of brain increase, due to increase free fatty acids in blood circulation. These changes allow tryptophan to compete with binding site of albumin and help its transport to central nervous system [54]. Similarly, oxidative processes have been considered necessary for metabolic (e.g., porphyria) and morphological damage of the liver [55].

Another study reports that TCDD/PCDFs have effects on female Rhesus monkeys when exposed to 0, 5, or 25 ng/L in their diet for 4 years and then protected for 10 additional years. It was noticed that these monkeys caught with severity of endometriosis [56]. On the other hand, endometriosis cyst growth in both rats and mice has also been enhanced by exposure to TCDD/PCDFs at very low doses where no ovarian toxicity occurred [57]. However, a dose of 10 mg TCDD/kg for a 16 week period resulted in ovarian atrophy in mice [58].

6. Mitigation and control of dioxins and furans

Various techniques are available for the controlled emission of dioxins and furans depending upon the type of feed stocks. Some popular and effective methods are: using sulfur compounds namely $(\text{NH}_4)_2\text{SO}_4$, pyrite (FeS_2), changing the operating conditions of incinerations.

Present chapter discuss methods to minimize effect of dioxins and benzofurans and their formation in different types of incineration systems. Municipal solid waste incineration system, hazardous solid waste incineration system and bio medical waste incineration system. Formation mechanism and the various sources including the precursors of PCDD/Fs formation need to be controlled during the combustion. It is also evident through literature that risk management strategies to reduce polychlorinated dibenzo-dioxins (PCDDs) and dibenzofurans (PCDFs) exposure, consideration may be given to the potential impact of changes to food and nutrition policies, particularly those related to public health and food.

There are number of technologies practiced around the globe to control combustion practice coupled with end of pipe treatment. Most preferably these are selective catalytic reduction, addition of suitable inhibitors for dioxins and furans eradication in the flue gas of waste incinerators. A brief background with pictorial images of the present technologies has been presented in this chapter (**Figure 2**) three different technologies used for the control of dioxins and furans have been discussed. Selective catalytic oxidation or reduction (SCR) using NH_3 -SCR catalysts (commercial $\text{V}_2\text{O}_5\text{-WO}_3/\text{TiO}_2$) could effectively promote the decomposition of PCDD/PCDFs without hampering the normal operating conditions of the incineration studied by [60]. Vermeulen et al. added urea for the purpose of decomposition of dioxins reducing it upto 90% under the same operating conditions as of ammonia [59]. In the process of adding compounds of sulfur to control the formation different congeners of PCDD/PCDFs (**Figure 3**) the feedstock is firstly prepared, homogenized with crushing and adding into the rotary kiln. The emission of dioxin compounds were controlled by a series of operations that includes quenching tower, acid neutralizing tower, wet scrubber, bag filter and activated

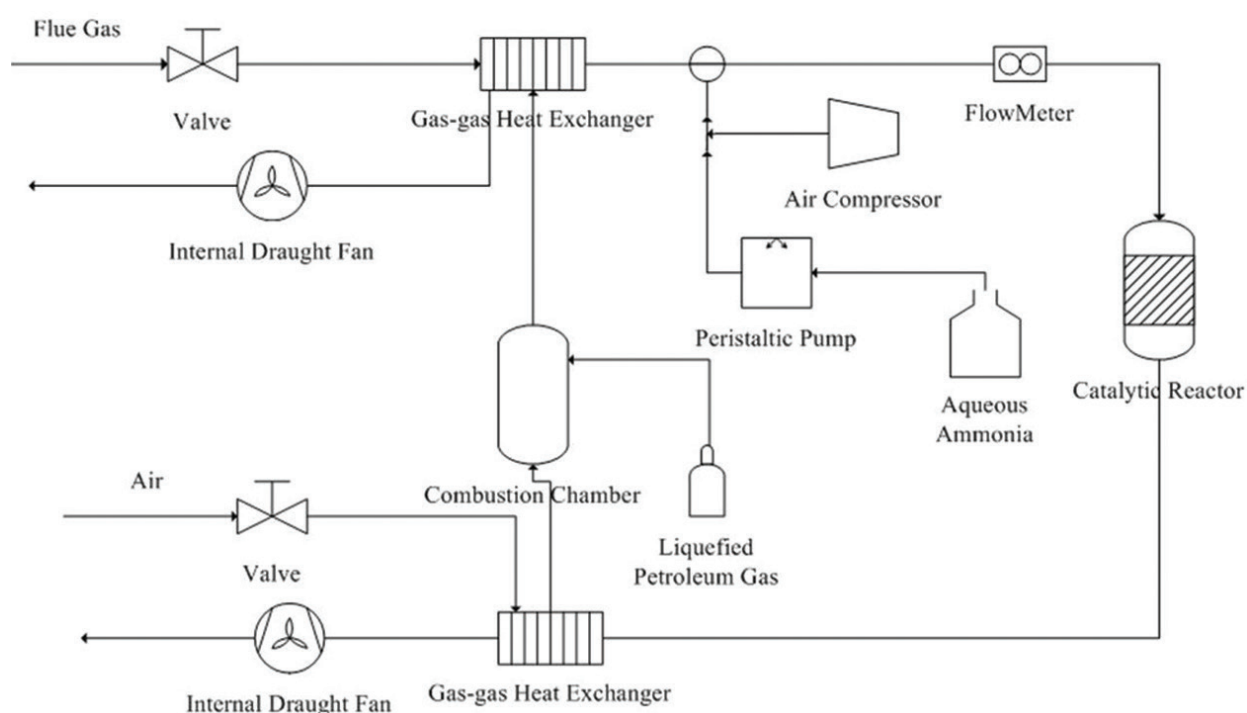


Figure 2. Inhibition of PCDD/PCDFs by the aid of $\text{V}_2\text{O}_5\text{-WO}_3/\text{TiO}_2$ catalysts [59].

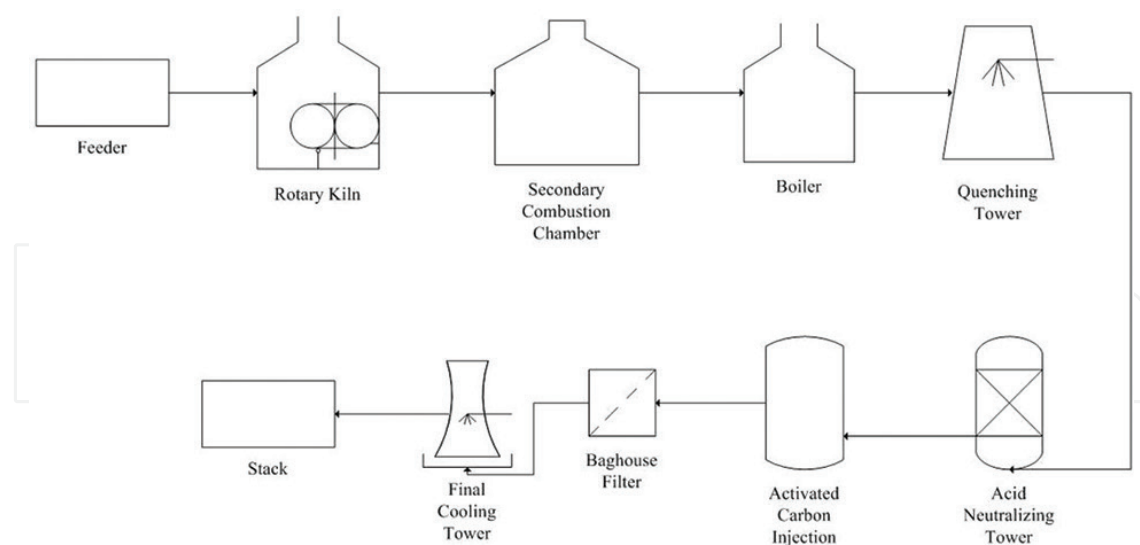


Figure 3. Flow diagram of sulfur dioxide circulation system [61].

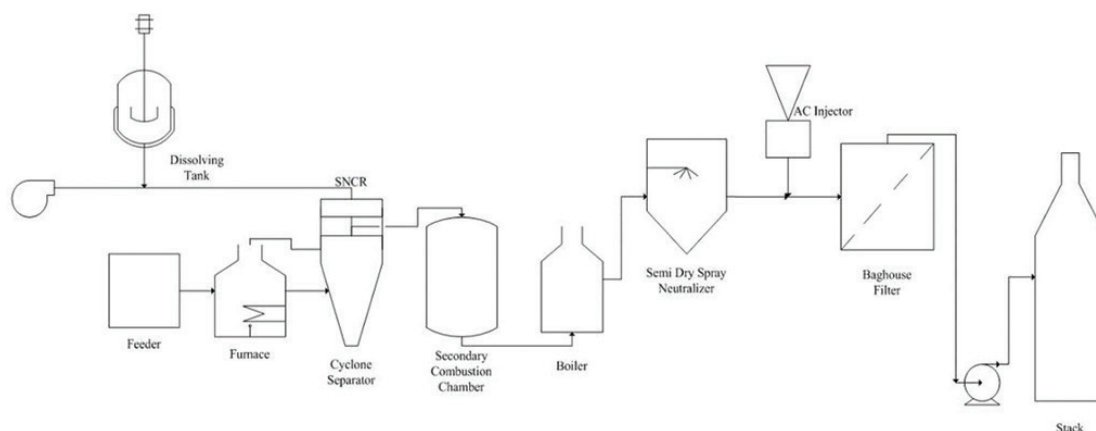


Figure 4. Flow diagram of the waste incinerator that uses thiourea (1: feeder; 2: furnace; 3: cyclone separator; 4: SNCR; 5: secondary combustion chamber; 6: boiler; 7: semi-dry spray neutralizer; 8: AC injector; 9: bag house filter; 10: stack; 11: dissolving tank) [60].

carbon dosing [61]. One important criterion to optimize the minimization of PCDD/PCDFs is the proper selection of sulfur compounds. In third technology, compounds of nitrogen such as ethanolamine, mono-ethanolamine (MEA), urea, ammonia, tri-ethanolamine, di-methylamine were added to control the formation of dioxins and furans. It was studied that thiourea is a suitable dioxin inhibitor with high S and N-content **Figure 4** [61].

7. Conclusions

The concentration of PCDD/PCDFs in environment has been increased up to the range of toxicity so these compounds may affect the biological systems. There are various means of production of PCDD/PCDFs which has significant impact on rise of levels of these compounds in

ambient air. The concentrations of PCDD/PCDFs in the environment are not well controlled due to many reasons like temperature variations and seasonal pattern. Although a large no of research studies have been conducted to investigate and control PCDD/PCDFs but even then information is limited to get about these compounds therefore it is difficult to get rid of them. Further investigations should be conducted to establish a comprehensive approach to investigate the recent profiles of PCDD/PCDFs found in various parts of the world especially in developing countries. This chapter will help the stake holders in decision making process to establish a sustainable waste management system in future. New and better innovative research plans are necessary for addressing the problems related to pollution.

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References

- [1] Colbeck I, Nasir ZA, Ali Z. The state of ambient air quality in Pakistan—A review. *Environmental Science and Pollution Research*. 2010;**17**(1):49-63
- [2] Zhang Q, Jiang X, Tong D, Davis SJ, Zhao H, Geng G, et al. Transboundary health impacts of transported global air pollution and international trade. *Nature*. 2017;**543**(7647):705
- [3] Kumar JM, Deepika D, Srinithya B, Kalaichelvan P. Polychlorinated dibenzo P dioxins and furans—A review. *International Journal of Current Research and Review*. 2013;**5**(3):14
- [4] Boffetta P, Gouas DA, da Costa AN, Abedi-Ardekani B, Hainaut P. Cancers of the Intestine, Liver, and Biliary Tract. *Occupational Cancers*. Springer; 2014. pp. 127-137
- [5] Safe S, Hutzinger O, Hill TA. Polychlorinated Dibenzo-p-Dioxins and-Furans (PCDDs/PCDFs): Sources and Environmental Impact, Epidemiology, Mechanisms of Action, Health Risks. Springer Science & Business Media; 2012
- [6] Hoogenboom RL, ten Dam G, van Bruggen M, Jeurissen SM, van Leeuwen SP, Theelen RM, et al. Polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) and biphenyls (PCBs) in home-produced eggs. *Chemosphere*. 2016;**150**:311-319
- [7] Urban JD, Wikoff DS, Bunch AT, Harris MA, Haws LC. A review of background dioxin concentrations in urban/suburban and rural soils across the United States: Implications for site assessments and the establishment of soil cleanup levels. *Science of the Total Environment*. 2014;**466**:586-597

- [8] Alcock RE, Jones KC. Dioxins in the environment: A review of trend data. *Environmental Science & Technology*. 1996;**30**(11):3133-3143
- [9] Cleverly D. The congener profiles of anthropogenic sources of chlorinated dibenzo-p-dioxins and chlorinated dibenzofurans in the United States. *Organohalogen Compounds*. 1997;**32**:430-435
- [10] Boer F, Neuman M, Van Remoortere F, North P, Rinn H. X-Ray Diffraction Studies of Chlorinated Dibenzo-p-Dioxins. ACS Publications; 1973
- [11] Hites RA. Dioxins: An Overview and History. ACS Publications; 2010
- [12] Schwetz B, Norris J, Sparschu G, Rowe V, Gehring P, Emerson J, et al. Toxicology of Chlorinated Dibenzo-p-Dioxins. ACS Publications; 1973
- [13] Van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, et al. The 2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. *Toxicological Sciences*. 2006;**93**(2):223-241
- [14] Kulkarni PS, Crespo JG, Afonso CA. Dioxins sources and current remediation technologies—A review. *Environment International*. 2008;**34**(1):139-153
- [15] Bumb R, Crummett W, Cutie S, Gledhill J, Hummel R, Kagel R, et al. Trace chemistries of fire: A source of chlorinated dioxins. *Science*. 1980:385-390
- [16] Rawls RL, Swanson D, Meselson M. Dow finds support, doubt for dioxin ideals. *Chemical and Engineering News (United States)*. 1979;**57**(7)
- [17] Mocarelli P. Seveso: A teaching story. *Chemosphere*. 2001;**43**(4-7):391-402
- [18] Stone R. Agent Orange's Bitter Harvest. American Association for the Advancement of Science; 2007
- [19] Päpke O. PCDD/PCDF: Human background data for Germany, a 10-year experience. *Environmental Health Perspectives*. 1998;**106**(Suppl 2):723
- [20] Huang H, Buskens A. Comparison of dioxin formation levels in laboratory gas-phase flow reactors with those calculated using the Shaub-Tsang mechanism. *Chemosphere*. 1999;**38**(7):1595-1602
- [21] Sidhu S, Edwards P. Role of phenoxy radicals in PCDD/F formation. *International Journal of Chemical Kinetics*. 2002;**34**(9):531-541
- [22] Addink R, Olie K. Mechanisms of formation and destruction of polychlorinated dibenzo-p-dioxins and dibenzofurans in heterogeneous systems. *Environmental Science & Technology*. 1995;**29**(6):1425-1435
- [23] Delvaux T, Buekens P, Godin I, Boutsen M. Barriers to prenatal care in Europe. *American Journal of Preventive Medicine*. 2001;**21**(1):52-59

- [24] Tuppurainen K, Halonen I, Ruokojärvi P, Tarhanen J, Ruuskanen J. Formation of PCDDs and PCDFs in municipal waste incineration and its inhibition mechanisms: A review. *Chemosphere*. 1998;**36**(7):1493-1511
- [25] Ligon W, Dorn SB, May RJ, Allison MJ. Chlorodibenzofuran and chlorodibenzo-p-dioxin levels in Chilean mummies dated to about 2800 years before the present. *Environmental Science & Technology*. 1989;**23**(10):1286-1290
- [26] Czuczwa JM, Hites RA. Environmental fate of combustion-generated polychlorinated dioxins and furans. *Environmental Science & Technology*. 1984;**18**(6):444-450
- [27] Altwicker E. Relative rates of formation of polychlorinated dioxins and furans from precursor and de novo reactions. *Chemosphere*. 1996;**33**(10):1897-1904
- [28] Abad E, Adrados M, Caixach J, Rivera J. Dioxin abatement strategies and mass balance at a municipal waste management plant. *Environmental Science & Technology*. 2002; **36**(1):92-99
- [29] Stanmore B, Clunies-Ross C. An empirical model for the de novo formation of PCDD/F in medical waste incinerators. *Environmental Science & Technology*. 2000;**34**(21):4538-4544
- [30] Karademir A, Bakoglu M, Ayberk S. PCDD/F removal efficiencies of electrostatic precipitator and wet scrubbers in izaydas hazardous waste incinerator. *Fresenius Environmental Bulletin*. 2003;**12**(10):1228-1232
- [31] Fullana A, Conesa JA, Font R, Sidhu S. Formation and destruction of chlorinated pollutants during sewage sludge incineration. *Environmental Science & Technology*. 2004; **38**(10):2953-2958
- [32] Eduljee G. Waste disposal in cement kilns: A review of dioxin formation and control. *Environmental & Waste Management*. 1999;**2**(1):45-54
- [33] Quaß U, Fermann MW, Bröker G. Steps towards a European dioxin emission inventory. *Chemosphere*. 2000;**40**(9-11):1125-1129
- [34] Lavric ED, Konnov AA, De Ruyck J. Dioxin levels in wood combustion—A review. *Biomass and Bioenergy*. 2004;**26**(2):115-145
- [35] Marklund S, Andersson R, Tysklind M, Rappe C, Egeback K-E, Björkman E, et al. Emissions of PCDDs and PCDFs in gasoline and diesel fueled cars. *Chemosphere*. 1990; **20**(5):553-561
- [36] Rappe C, Andersson R, Bergqvist P-A, Brohede C, Hansson M, Kjeller L-O, et al. Overview on environmental fate of chlorinated dioxins and dibenzofurans. Sources, levels and isomeric pattern in various matrices. *Chemosphere*. 1987;**16**(8-9):1603-1618
- [37] Wang X, Ni Y, Zhang H, Zhang X, Chen J. Formation and emission of PCDD/Fs in Chinese non-wood pulp and paper mills. *Environmental Science & Technology*. 2012; **46**(21):12234-12240

- [38] Anderson DR, Fisher R. Sources of dioxins in the United Kingdom: The steel industry and other sources. *Chemosphere*. 2002;**46**(3):371-381
- [39] Cieplik MK, Carbonell JP, Muñoz C, Baker S, Krüger S, Liljelind P, et al. On dioxin formation in iron ore sintering. *Environmental Science & Technology*. 2003;**37**(15):3323-3331
- [40] Shiomitsu T, Hirayama A, Iwasaki T, Akashi T, Fujisawa Y. Volatilization and decomposition of dioxin from fly ash. In: NKK Technical Report-Japanese Edition. 2001. pp. 8-11
- [41] Anthony E, Jia L, Granatstein D. Dioxin and furan formation in FBC boilers. *Environmental Science & Technology*. 2001;**35**(14):3002-3007
- [42] Petroff BK, Roby KF, Gao X, Son D-S, Williams S, Johnson D, et al. A review of mechanisms controlling ovulation with implications for the anovulatory effects of polychlorinated dibenzo-p-dioxins in rodents. *Toxicology*. 2001;**158**(3):91-107
- [43] Gray Jr LE, Kelce WR. Latent effects of pesticides and toxic substances on sexual differentiation of rodents. *Toxicology and Industrial Health*. 1996;**12**(3-4):515-531
- [44] Rier S, Foster WG. Environmental dioxins and endometriosis. In: *Seminars in Reproductive Medicine*. New York, NY, USA: Thieme Medical Publishers, Inc.; 2003
- [45] Gray Jr LE. Xenoendocrine disrupters: Laboratory studies on male reproductive effects. *Toxicology Letters*. 1998;**102**:331-335
- [46] Pop VJ, Kuijpers JL, van Baar AL, Verkerk G, van Son MM, de Vijlder JJ, et al. Low maternal free thyroxine concentrations during early pregnancy are associated with impaired psychomotor development in infancy. *Clinical Endocrinology*. 1999;**50**(2):149-155
- [47] Vulsma T. Impact of exposure to maternal PCBs and dioxins on the neonate's thyroid hormone status. *Epidemiology*. 2000;**11**(3):239
- [48] Kerkvliet NI. Immunological effects of chlorinated dibenzo-p-dioxins. *Environmental Health Perspectives*. 1995;**103**(Suppl 9):47
- [49] Takeyama M, Tohyama C. Developmental neurotoxicity of dioxin and its related compounds. *Industrial Health*. 2003;**41**(3):215-230
- [50] Stohs SJ. Oxidative stress induced by 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD). *Free Radical Biology and Medicine*. 1990;**9**(1):79-90
- [51] Hebert C, Harris M, Elwell M, Birnbaum L. Relative toxicity and tumor-promoting ability of 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD), 2, 3, 4, 7, 8-pentachlorodibenzofuran (PCDF), and 1, 2, 3, 4, 7, 8-hexachlorodibenzofuran (HCDF) in hairless mice. *Toxicology and Applied Pharmacology*. 1990;**102**(2):362-377
- [52] Panteleyev AA, Thiel R, Wanner R, Zhang J, Roumak VS, Paus R, et al. 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD) affects keratin 1 and keratin 17 gene expression and differentially induces keratinization in hairless mouse skin. *Journal of Investigative Dermatology*. 1997;**108**(3):330-335

- [53] Pohjanvirta R, Tuomisto J. Short-term toxicity of 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin in laboratory animals: Effects, mechanisms, and animal models. *Pharmacological Reviews*. 1994;**46**(4):483-549
- [54] Unkila M, Pohjanvirta R, Tuomisto J. Biochemical effects of 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin (TCDD) and related compounds on the central nervous system. *The International Journal of Biochemistry & Cell Biology*. 1995;**27**(5):443-455
- [55] Smith AG, Clothier B, Robinson S, Scullion MJ, Carthew P, Edwards R, et al. Interaction between iron metabolism and 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin in mice with variants of the Ahr gene: A hepatic oxidative mechanism. *Molecular Pharmacology*. 1998; **53**(1):52-61
- [56] Rier SE, Martin DC, Bowman RE, Dmowski WP, Becker JL. Endometriosis in rhesus monkeys (*Macaca mulatta*) following chronic exposure to 2, 3, 7, 8-tetrachlorodibenzo-p-dioxin. *Toxicological Sciences*. 1993;**21**(4):433-441
- [57] Heilier J-F, Donnez J, Lison D. Organochlorines and endometriosis: A mini-review. *Chemosphere*. 2008;**71**(2):203-210
- [58] Johnson KL, Cummings AM, Birnbaum LS. Promotion of endometriosis in mice by polychlorinated dibenzo-p-dioxins, dibenzofurans, and biphenyls. *Environmental Health Perspectives*. 1997;**105**(7):750
- [59] Vermeulen I, Van Caneghem J, Vandecasteele C. Indication of PCDD/F formation through precursor condensation in a full-scale hazardous waste incinerator. *Journal of Material Cycles and Waste Management*. 2014;**16**(1):167-171
- [60] Lin X, Yan M, Dai A, Zhan M, Fu J, Li X, et al. Simultaneous suppression of PCDD/F and NO_x during municipal solid waste incineration. *Chemosphere*. 2015;**126**:60-66
- [61] Liu X, Wang J, Wang X, Zhu T. Simultaneous removal of PCDD/Fs and NO_x from the flue gas of a municipal solid waste incinerator with a pilot plant. *Chemosphere*. 2015; **133**:90-96

