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Toxicity of polychlorinated biphenyls in aquatic environments – A review

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ABSTRACT

The assessment of polychlorinated biphenyls (PCBs) and their congeners resulting from the pollution of all environmental media is inherently related to its persistence and ubiquitous nature. In principle, determination of this class of contaminants are limited to the determination of their concentrations in the various environmental matrices. For solving many problems in this context, knowledge of the emission sources of PCBs, transport pathways, and sites of contamination and biomagnification is of great benefit to scientists and researchers, as well as many regulatory organizations. By far the largest amounts of PCBs, regardless of their discharged points, end up in the soil, sediment and finally in different aquatic environments. By reviewing relevant published materials, the source of origin of PCBs in the environment particularly from different pollution point sources, it is possible to obtain useful information on the nature of different materials that are sources of PCBs, or their concentrations and their toxicity or health effects and how they can be removed from contaminated media. This review focuses on the sources of PCBs in aquatic environments and critically reviews the toxicity of PCBs in aquatic animals and plants. The review also assesses the toxicity equivalency factors (TEFs) of PCBs providing valuable knowledge to other scientists and researchers that enables regulatory laws to be formulated based on selective determination of concentrations regarding their maximum permissible limits (MPLs) allowed. This review also supplies a pool of valuable information useful for designing decontamination technologies for PCBs in media like soil, sediment, and wastewaters.

1. Introduction

Polychlorinated biphenyls (PCBs), also called chlorinated diphenyls, chlorinated biphenyls, chlorinated hydrocarbons, chlorobiphenyls, or polychlorobiphenyls, are heterogeneous, synthetic, and widespread organochlorine compounds belonging to the Persistent Organic Pollutants (POPs); they are also known as semi-volatile organic pollutants (SOCs) that can be found in the vapour or particulate form (Helou *et al.*, 2019; Koss *et al.*, 1999). PCBs consist of carbon, hydrogen, and chlorine atoms (Hong *et al.*, 2012; Liu *et al.*, 2014; Neira *et al.*, 2018). These anthropogenic pollutants were first synthesized in 1876 by Oskar Gustav Doebner in Germany, but their manufacturing for commercial uses first took place in the United States in 1929 by *Monsanto Corporation* and consecutively throughout the world (Bursian, 2007). PCBs can be produced by direct catalytic chlorination of two benzene rings (biphenyl $C_{12}H_{10}$) with ferric chloride ($FeCl_3$), producing one out of 209 PCB congeners with the general formula $C_{12}H_{10\text{-m-n}}Cl_{m+n}$ with $m + n = 1$–10 (Fig. 1) (Bianco *et al.*, 2008; Crucello *et al.*, 2020; Gdaniec-Pietryka *et al.*, 2007; Hong *et al.*, 2005; Jia *et al.*, 2013; Vergani *et al.*, 2017). PCBs are among the most prevalent and notorious POPs that are found in environmental media (*e.g.* water, air, soil) resulting from several industrial activities and present a burden to the environment (Font *et al.*, 1996). Though their use and manufacturing were prohibited decades ago, they are still present in the environment because of their bioaccumulative and persistent nature as well as still existing releases (Dickerson *et al.*, 2019). However, PCBs are more abundant in marine environments/ecosystems *i.e.* water, sediments and aquatic organisms, as this is their final repository (Reddy *et al.*, 2019). About 30% of the historical worldwide production is still present in aquatic ecosystems and associated with biotic and abiotic organisms due to their high resistance to environmental processes, atmospheric deposition, industrial discharges, uncontrolled spillage and water treatment technologies (Wang *et al.*,

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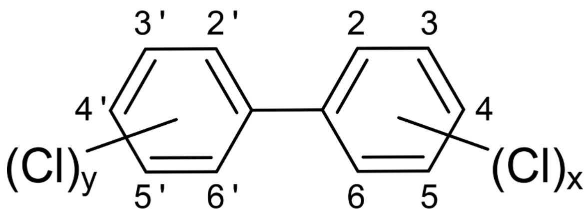

Fig. 1. Chemical configuration of PCBs. Chlorine atoms may substitute hydrogen atoms at positions 2, 2′,6 or 6′ (ortho), 3, 3′, 5, 5′ (meta) or 4, 4′ (para) (modified after Borja *et al.*, 2005).

2019b). Release of these compounds into aquatic environments is problematic for biotic organisms due to their toxicological effects through binding with the aryl hydrocarbon receptor (AhR), ubiquitous presence and slow biodegradability nature (Song *et al.*, 2018). Indeed, they are chemicals that can disrupt the body's hormone levels, thereby affecting hormonal, reproductive, immune, or endocrine systems (Buha Djordjevic *et al.*, 2020). These disruptions can cause cancer, congenital malformations, and other cognitive disabilities (Buha Djordjevic *et al.*, 2020). PCBs deteriorate water quality, pollute water, and represent a definite potential health hazard or risk when they are taken up by aquatic species. This is due to their accumulation in aquatic animal tissues or because of their lipophilicity, hydrophobicity and low biodegradability (Zani *et al.*, 2019). Bioaccumulation of PCBs in animal tissues poses adverse effects to their biology and threatens their survival which can result in a reduction in number of aquatic animals in water bodies (Zani *et al.*, 2019). Human health is threatened due to the consumption of biomagnified marine fatty food through aquatic food/trophic chains depending on the trophic level (Baldigo *et al.*, 2006). Humans are mostly exposed to PCBs during the consumption of seafood because PCB residues bioaccumulate at high concentrations in fish tissues due to their lipophilic nature (Miyashita *et al.*, 2015; Shin *et al.*, 2016). To protect humans and biota health, several maximum permissible limits (MPLs) of PCBs in food products have been established by various organizations in different countries (Font *et al.*, 1996).

2. Sources of PCBs in aquatic environments

The various applications of PCBs in appliances, past anthropogenic and industrial activities as well as spillages or thermal processes have resulted in their incessant release into biotic and abiotic environments through industrial or urban activities. As a result, they are found in every compartment (*e.g.* Air, soil, sediments, water, food products, and biota) of the global system at a large range of concentrations, mainly due to their persistence and high stability (Gao *et al.*, 2013; Reddy *et al.*, 2019; Safder *et al.*, 2018; Strek *et al.*, 1982; Wang *et al.*, 2019a; Wolska *et al.*, 2014).

Historical releases of PCB-contaminated discharges (sewage or wastewater effluents) in water bodies represent one of the primary causes of water pollution (Anh *et al.*, 2021). PCBs from badly maintained industrial, urban or mining wastes of manufacturing processes are sometimes discharged on hazardous waste dumps or leachate landfill sites, and overtime they enter the soil or groundwater (Chakraborty *et al.*, 2016). Subsequently, through wet deposition or precipitation, during washout in rain, storm and snow falls, surface runoff or erosion, PCB contaminants are washed away or evaporated from soils, move along with soil nutrients and enter nearby water bodies such as streams, rivers, lakes, and oceans (Estoppey *et al.*, 2015; Reddy *et al.*, 2019) (Fig. 2). Agricultural and domestic activities can also contribute to their presence in water bodies, for example from the use of PCB-containing pesticides (Chakraborty *et al.*, 2016; Kassegne *et al.*, 2020). PCBs can also access water bodies through atmospheric deposition, transport or fallout of PCB dust, which occurs, when highly chlorinated congeners are subjected to uncontrolled, open and low-temperature burning, municipal, industrial and hazardous solid waste incineration of PCB-containing materials such as plastics, open burning, combustion of coal or improper management of wastes as well as voluntary and involuntary fires or emissions (Batterman *et al.*, 2009; Dodoo *et al.*, 2012; Duinker, 1986; Hung *et al.*, 2006; Reddy *et al.*, 2019; Rusin *et al.*, 2019; Wolska *et al.*, 2014). These PCBs deposit in aquatic microorganisms, are swallowed by aquatic animals and result in the presence of PCBs in aquatic food chains (Hoogenboom, 2012; Xu *et al.*, 2019). Improper disposal of PCB-containing waste materials from construction

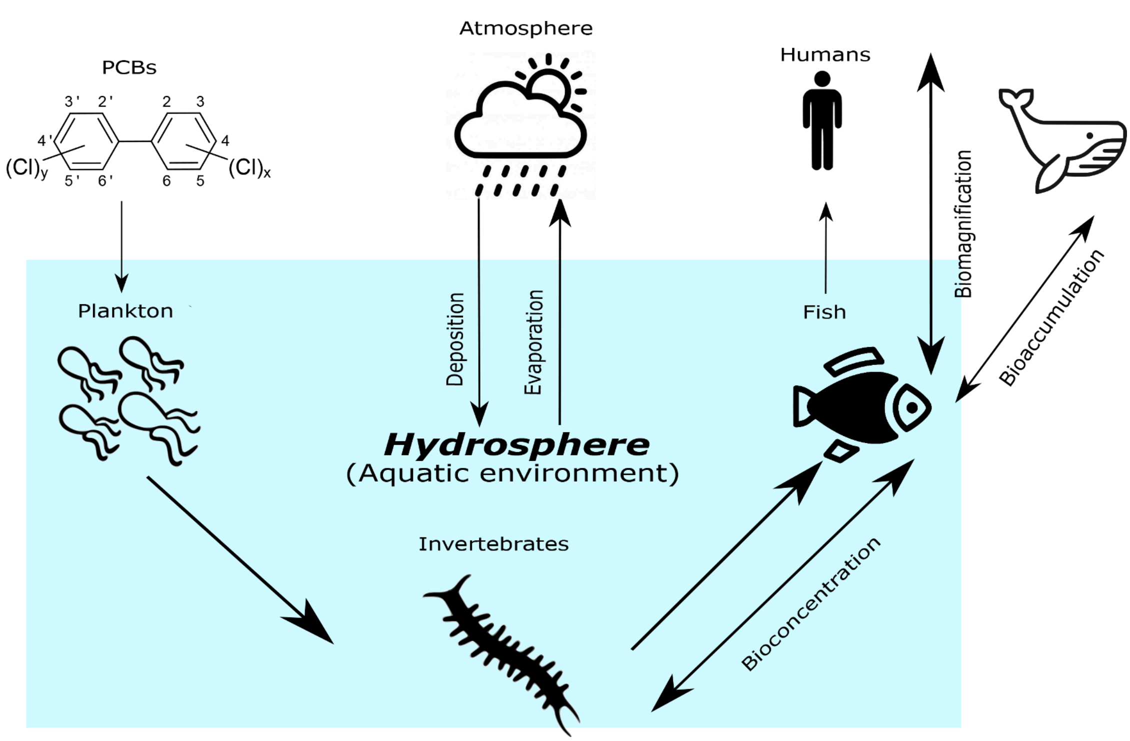

Fig. 2. Pathways of PCBs through the aquatic environment, from planktons to humans via the atmosphere (after Reddy *et al.*, 2019).

and building elements such as paints, plastics, glass, paper, rubber, calk, roofing, siding, sealants, electric appliances, or electrical facilities can also result in contamination of nearby water bodies (Chakraborty *et al.*, 2016; Jartun *et al.*, 2009) because they can easily move along with water during rain events (Chandra Yadav *et al.*, 2020; Jartun *et al.*, 2009). This is an important source, threatening the aquatic environment because some recently manufactured constructing equipment still might contain PCBs (Jartun *et al.*, 2009; Lambiase *et al.*, 2021). De-dusting of roads using PCB oils is also a source PCBs (Batterman *et al.*, 2009), as the PCBs attach to the dust particles and move along with them to nearby water courses (Anh *et al.*, 2019; Chandra Yadav *et al.*, 2020).

Lighter PCBs can evaporate because of their high volatility, low biodegradability, and high spreading, allowing them to be found in numerous environmental media due to their long-range atmospheric transport and their ability to accumulate in the atmosphere (Bursian, 2007; Gao *et al.*, 2013; Song *et al.*, 2018; Wolska *et al.*, 2014). PCBs half-lives in water vary from 0.165 to approximately 13.689 years (Reddy *et al.*, 2019). These congeners are rapidly and atmospherically transported from one place (mostly industrialized areas) to another due to their dispersion in the atmosphere from paints, coatings, and plastics (Arinaitwe *et al.*, 2018; Reddy *et al.*, 2019). They can also volatilize and condense during the transportation, synthesis, usage, and storage (Cui *et al.*, 2018; Greenfield *et al.*, 2013). Yet, the extent of volatilization depends on temperature because condensation can take place at low temperatures (Jartun *et al.*, 2009). Aerial transport is the main source for the global pollution of large water bodies through air-sea or air-soil exchange (Arinaitwe *et al.*, 2018; Wu *et al.*, 2020). Wastewater, mine water, sewage or industrial treatment facilities, and on-site sanitation systems can also lead to the presence of PCBs in the aquatic environment, which is considered as the secondary source of PCBs in marine ecosystems (Estoppey *et al.*, 2015).

Improper or illegal disposal of old electrical appliances, materials, wastewater, and waste fluids containing PCBs on inappropriate sites, non-engineered landfills or directly into water bodies can pollute aquatic environments (Bursian, 2007; Habibullah-Al-Mamun *et al.*, 2019; Yang *et al.*, 2009). This source is the main cause of PCB-contamination in Africa, particularly South Africa, where concentrations are still increasing despite their ban (Arinaitwe *et al.*, 2018; Batterman *et al.*, 2009). However, the main source of PCBs in mine or industrial water is the leakage, accidental or uncontrolled spillage of lubricating fluids on landfills or accidental spillage from old PCB-containing appliances such as transformers, capacitors, and automobiles during transport into sewers and streams (Bursian, 2007; Eqani *et al.*, 2013) or from one compartment to another and ending up in the aquatic environment (Borja *et al.*, 2005). Nearby human activities cause their concentrations to be higher inshore than offshore, with pollution sources influencing the congener types and concentrations (Borja *et al.*, 2005; Duan *et al.*, 2013).

When PCBs are in aquatic environments, they can remain there for decades due to their persistence and affinity for suspended solids (Kodavanti *et al.*, 2019; Storelli *et al.*, 2009) or microplastic. They also resist organic processes including oxidation and reduction as well as acids, bases and harsh temperature conditions due to their hydrophobic property, as well as high chemical and thermal stability (Reddy *et al.*, 2019). PCBs are even found in marine ecosystems in regions where they have never been used. Once again with concentrations higher near industrialized compared to urban areas (Batang *et al.*, 2016; Reddy *et al.*, 2019). This is because PCB-producers or users are mostly found in cities.

The EU maximum permissible limit (MPL) of all PCBs in the dissolved phase has been set at 1–2 μg L^{-1} for natural water and 0.1–0.2 μg L^{-1} for drinking waters (Font *et al.*, 1996). In humans or animals, the most targeted PCB congeners are the indicator PCBs PCB-28, 52, 101, 118, 138, 153 and 180 as well as non or mono-ortho highly chlorinated congeners (Storelli *et al.*, 2001). The *European Commission* has set the limit of these PCB indicators at 100 to 200 ng g^{-1} fresh weight in animal food products such as meat, egg, or poultry products (Antonijevic *et al.*, 2011; Davodi *et al.*, 2011; Li *et al.*, 2008). MPL of PCBs in Brazil has been established at 3000 ng g^{-1} lipid for animal products (Quinete *et al.*, 2011) and 2 μg g^{-1} and 100 ng g^{-1} by the FDA/EPA and the Italian government respectively (Quinete *et al.*, 2011).

3. Some superfund sites in the US

The Hudson River is a 507 km river that flows from the north of New York to the south of Manhattan while the New Bedford Harbor (NBH) is the tidal estuarine section of the Acushnet River, a river in the southeastern Massachusetts. These estuaries have been known to inhabit aquatic biota and are useful for several commercial and recreational activities. Unfortunately, in 1977, these rivers were found to exhibit high concentrations of PCBs as a result of discharges from nearby agricultural, commercial, industrial, and municipal activities, surface runoff and releases from the Troy Dam from 1940s (Ashley *et al.*, 2003; Chitsaz *et al.*, 2020; Nacci *et al.*, 2009; Yan *et al.*, 2008). These authors also reported that highest concentrations were recorded in the upper part of these rivers. From more than 70 years, industrial discharges from electronic component manufacturing plants, producing PCB-containing appliances, namely electrical capacitors, have been causing the most damages. Indeed, the two General Electric (GE) plants, located in Hudson Falls and Fort Edward in New York have been depositing their wastes in the Hudson River whilst Aerovox Corporation and Cornbell Dublier have been discharging their wastes in the New Bedford Harbor (Chitsaz *et al.*, 2020). As a result, in 1979, the Massachusetts Department of Public Health prohibited sections of the New Bedford Harbor for fishing as it was found to exceed Food and Drug Administration (FDA) standard limits. Although the manufacturing plants ceased their production and releases into the river from 1976, redistribution of previously discharged PCBs is still of a concern. Consequently, in 1982 and 1984, the New Bedford Harbor and the Hudson River, respectively, qualified as Superfund sites and included in the National Priorities List (NPL) by U.S. Environmental Protection Agency (USEPA) in 1984 (Deshpande *et al.*, 2013; Maceina *et al.*, 2015). From the 1940s to 1977, the total releases of PCBs into the Hudson river had been estimated to range between 94.8–603 t. PCBs sorbed onto sediments, deposited at the bottom of those rivers and accumulated in the tissues of aquatic animals such as fish (Pinkney *et al.*, 2017). Thus, from 2009 to 2015, dredging strategies have been established to remove PCB-sorbed sediments with concentrations between 10 mg L^{-1} and 50 mg L^{-1} from those rivers as to restore their sustainability (Deshpande *et al.*, 2013; Sandy *et al.*, 2013). Despite all remediation processes, concentrations are still elevated in these water bodies.

All great lakes were also found to exhibit high concentrations of PCBs (Martinez *et al.*, 2015). However, Lake Michigan was declared to be more polluted compared to other lakes. As a result, lake Michigan was also classified as a superfund site from high pollutant loads from tributaries (*e.g.*: Sheboygan River) and industrial watersheds located in east Chicago (*e.g.*: Indiana Harbor and Ship Canal (IHSC)) (Martinez *et al.*, 2015). Gas absorption, atmospheric deposition, and precipitation in nearshore southern Lake Michigan, Chicago are also substantial (Jahnke *et al.*, 2022; Offenberg *et al.*, 2000). Yet, industrial operations and emissions as well as urbanisation in Chicago metropolitan area have heavily enhanced PCB loads in lake Michigan (Martinez *et al.*, 2010). For instance, Aroclor 1248 used as additives and insulating oils in several industrial appliances has been an important source of PCBs in the lake. When PCBs enter the lake, they sorb onto sediments at the bottom of the lake, thus, the lake becomes a source repository for PCBs and a source of PCBs to Chicago (Rasmussen *et al.*, 2014). As fishes have a high lipid and organic content, they easily bioaccumulate lipophilic compounds such as PCBs at levels exceeding tolerance levels set by the U.S. Food and Drug Administration (FDA). In 1987, this seriously endangered fishing livestock in its tributaries leading a more stringent restriction of fishing activities imposed by the Wisconsin Department of Natural Resources (WDNR), resulting in economic downfalls (Rasmussen *et al.*, 2014).

Remediation measures were implemented, and concentrations were found to decrease between 1985 and 1994 (Madenjian *et al.*, 1999; Rasmussen *et al.*, 2014).

4. PCBs in aquatic environments

In aquatic media, PCBs are present in two phases: the suspended or the dissolved phase. In particular, PCBs attach to suspended solids or sediments via sedimentation due to their hydrophobic nature. As a result, their concentration in sediments is higher than the concentration in the bulk water (Hong *et al.*, 2012; Kruse *et al.*, 2014; Wang *et al.*, 2019a). Indeed, their properties allow them to sorb onto the organic matter present at the surface of these solid particles (Birgül *et al.*, 2017; Čonka *et al.*, 2014; Endo *et al.*, 2017; Falandysz *et al.*, 2001; Reddy *et al.*, 2019; Yang *et al.*, 2009). This process is called sedimentation. Sediments are considered as secondary reservoirs of PCBs in water media and can be used to assess their concentrations and fate in the aquatic environment (Irerhievwie *et al.*, 2020; Ranjbar Jafarabadi *et al.*, 2019; Wang *et al.*, 2016b). Sorption of PCBs onto sediments relies on congener properties and sediment properties. Highly chlorinated congeners have a higher sorption capacity to sediments than less chlorinated congeners (Ranjbar Jafarabadi *et al.*, 2019). But, less chlorinated congeners can sometimes be found at elevated concentrations in sediments due to their ability to be transported away from their emission source (Gao *et al.*, 2013). Sediment properties, matrix constituents, as well as its type, structure (surface area, pore-volume, pore size), morphology (grain size and surface structure), crystal structure (amorphous and crystal), and surface chemistry influence sorption (Choi *et al.*, 2009). For instance, high organic carbon content and small-size sediments adsorb more PCBs than large-size sediments (Cho *et al.*, 2012). Sediment quality guidelines (SQGs) define maximum levels for PCBs in sediments considered safe for aquatic life. In other words, it helps to evaluate the adverse chemical and biological effects of PCBs in sediments on aquatic organisms (Merhaby *et al.*, 2020). SQGs of PCBs established by the National Oceanic and Atmospheric Administration (NOAA) of the US were set at 22.7, 180, 34.1 and 277 ng g^{-1} for effect range low (ERL), range of effect median (ERM), threshold effect level (TEL) and probable effect level (PEL), respectively (Long *et al.*, 1995; Merhaby *et al.*, 2015; Merhaby *et al.*, 2020).

5. Sampling and analysis of PCBs in aquatic environments

To sample PCBs from aquatic media, grab sampling is usually used to represent the entire medium. When sampling is carried out in shallow water sites, extra sampling devices are not needed; yet, when sampling is carried out in deep water sites such as lakes and rivers, dippers, and thief samplers are required (Thuan *et al.*, 2011). Samplers that are usually used for the sampling of water for the analysis of chlorinated compounds such as PCBs are Kemmerer, Van Dorn and double check valve bailer devices (Kishida, 2013). Sampling of underground water is carried out using portable peristaltic pumps (Martínez-Guijarro *et al.*, 2017). Particulate water can be sampled using passive sampling devices, vacuum filtration devices, or simply centrifugation (Nguyen *et al.*, 2017).

For the analysis of PCBs, sample enrichment is essential because they are usually present at low concentrations in environmental media (Wang *et al.*, 2016a). Several extraction techniques are used for extracting PCBs from aquatic media: pressurized liquid extraction (PLE), ultrasound-assisted extraction (UAE), solid phase micro-extraction (SPME), solid phase extraction (SPE), stir-bar sorptive extraction (SBSE), hollow fibre liquid phase microextraction (HF-LPME), hollow fibre liquid-liquid microextraction (HF-LLME), ultrasonic extraction (USE), liquid-phase microextraction (LPME), magnetic solid phase extraction (MSPE), dispersive solid phase extraction (DSPE), and selective pressurized liquid extraction (SPLE) (Olanca *et al.*, 2014; Reddy *et al.*, 2019; Wang *et al.*, 2016a).

Methods used to clean-up PCB extracts can be non-destructive or destructive and may be performed together or in an acidic environment to increase congeners' stability. Non-destructive lipid removal methods consist of gel permeation chromatography (GPC) and column chromatography. Destructive lipid removal methods are sulfuric acid treatment and saponification that both remove sulfur (Reddy *et al.*, 2019). GPC is like size exclusion where low molecular weight compounds such as PCBs are preserved while high molecular weight compounds such as lipids are retrenched. Column chromatography uses cartridges or columns composed of silica, alumina, florisil, and carbon adsorbents to clean the samples (Megson, 2019). On the opposite, sulfuric acid treatment helps to eliminate undesired organic compounds from the matrix using sulfuric acid. Saponification relies on the heating of extracts to evaporate interferences (Olanca *et al.*, 2014).

The analytical instrument used to analyse PCBs in environmental matrices is gas chromatography. Gas chromatography can be coupled to several detectors such as electron capture detector (ECD), electron ionization mass spectrometry (EI-MS), and high-resolution mass spectrometer (HRMS) depending on the analyte of interest and intended concentration in sample matrix (Castro-Jiménez *et al.*, 2011). Mass spectrometers can be magnetic sector, orbitrap, quadrupole (QMS), ion traps (IT-MS), time-of-flight (TOF-MS), and triple quadrupole (QqQ-MS (Peng *et al.*, 2015). Due to its high sensitivity, high selectivity towards halogenated compounds and high precision, ECD is more appropriate for the analysis of PCBs (Derouiche *et al.*, 2007; Hong *et al.*, 2005; Li *et al.*, 2012; Verenitch *et al.*, 2007). Yet, this analytical technique is sometimes costly and time-consuming, and co-elution is often frequent (Castro-Jiménez *et al.*, 2011). On the other hand, mass spectrometry detectors coupled to GC are also efficient in determining PCBs in environmental samples since they are semi-volatile and thermally stable compounds (Derouiche *et al.*, 2007). They improve the reliability and selectivity of the method (Verenitch et al., 2007). In some cases, the selected ion monitoring mode (SIM) improves the sensitivity of the method, that is because only a specific mass-to-charge ratio (ion) is detected with high sensitivity. Splitless or on-column injection methods should be used for PCBs and temperature programming is more suitable (Reddy *et al.*, 2019). The most commonly used column capable of resolving the 209 congeners is the (5%-Phenyl)-methylpolysiloxane column referred to as DB-5, Rtx-5, SPB-5, or BP-5, depending on the manufacturer (Megson, 2019).

6. Toxicity equivalency factors (TEFs)

Toxicity equivalency factors (TEFs) are established by the *World Health Organization* (WHO), whose aim is to evaluate the toxicity and hazardous effects of environmental pollutants such as PCBs (Corsolini *et al.*, 1995; Wu *et al.*, 2018). Their toxic character is based on their ability to inhibit functioning of body systems, instigate malformations and develop cancers (Xia *et al.*, 2012). PCB congener' TEFs have been established in relation to 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD), whose TEF is 1, based on their relationship with the AhR receptor (Corsolini *et al.*, 1995; Dodoo *et al.*, 2013). Toxic equivalent concentrations are calculated by multiplying the congener concentration with its corresponding TEF value (Te *et al.*, 2020), which is directly proportional to the trophic level of the organism because high trophic level organisms accumulate higher concentrations of PCBs (Te *et al.*, 2020). TEF values of non-ortho substituted PCBs such as PCB-77 and PCB-81 are 1×10^{-4} and 3×10^{-4} respectively, and that of mono-ortho substituted PCBs such as PCB-105 and 114 are 3×10^{-5} respectively (WHO, 2005).

7. Toxicity of PCBs

7.1. Animals

7.1.1. Bioaccumulation of PCBs in aquatic organism

High concentrations of low or high-chlorinated PCBs (dioxin and non-dioxin-like congeners) have been recorded in water bodies resulting in global pollution of marine environments from nearby industrial activities since the 1960s. This pollution presents a risk for aquatic animals' health both pelagic and benthic as well as invertebrates (plankton, salmons, fishes, shellfishes, farmed fishes, swordfishes, sea birds, sea turtles, sea eagles frogs, dolphins, sharks, whales, seals, and other marine mammals) (de Azevedo e Silva *et al.*, 2007; Di Bella *et al.*, 2006; Estoppey *et al.*, 2015; Greenfield *et al.*, 2013; Lambiase *et al.*, 2021; Nordlöf *et al.*, 2012). Marine mammals are at risk due to a higher bioaccumulation of PCB in their tissues (Storelli *et al.*, 2001) resulting from the consumption of small aquatic animals such as fish (Hope *et al.*, 1997; Tanabe *et al.*, 1997). Even though concentrations of chlorinated congeners found in aquatic animal tissues are sometimes below the detection limits, they can still be toxic and fatal (Di Bella *et al.*, 2006; Dodoo *et al.*, 2013). Higher chlorinated congeners (PCB-138, 153 180 and 187) are more abundant and harmful than lower chlorinated ones due to a difficulty to penetrate the cytoplasmic membrane, which separates the cells from the outside environment. In addition, they are also more stable, lipophilic, widespread, and retained for longer periods in fatty tissues of aquatic animals (Dodoo *et al.*, 2013; Li *et al.*, 2008). Lower chlorinated congeners can also be abundant due to their high resistance to environmental transformations, facilitating their migration from polluted sites to aquatic ecosystems (Iwegbue *et al.*, 2020). The difference of high or low chlorinated congeners' concentrations in the marine environment depends on nearby industrial uses. Some countries may have preferred to use higher chlorinated congeners for manufacturing while others may choose to use lower chlorinated congeners for manufacturing the same product (Iwegbue *et al.*, 2020). Nevertheless, analysis of high and low chlorinated congeners may be challenging due to their high hydrophobicity, low long-range transport and low chemical degradation or high volatility and high biotransformation respectively (Iwegbue *et al.*, 2020).

Chronic exposure to PCBs occurs through the inhalation *via* the respiratory tract (gills), skin adsorption *via* flesh, or diet *via* ingestion (Borja *et al.*, 2005; Visha *et al.*, 2018). Lower trophic level organisms such as planktons are usually contaminated *via* inhalation or dermal contact, whereas upper trophic level organisms are commonly contaminated *via* diet, accumulating high concentrations of PCBs due to their slow body metabolism, high fat tissues and high lipophilicity (Di Bella *et al.*, 2006; Visha *et al.*, 2018). Ingestion of contaminated lower trophic level organisms, bottom-dwelling micro-organisms, benthic organisms, PCB-containing plastics or detritus or PCB sorbed on suspended solids is the major source of aquatic animals' exposure to PCBs as is the exposure through dermal contact within the aquatic environment (Çelik Çakıroğulları *et al.*, 2010; Lambiase *et al.*, 2021; Ming-ch'eng Adams *et al.*, 2016; Moraleda-Cibrián *et al.*, 2015; Olanca *et al.*, 2014; Warenik-Bany *et al.*, 2019). Higher trophic level organisms including humans mainly are exposed to PCBs *via* ingestion of lower trophic level organisms (Juei Shen *et al.*, 1998; Nordlöf *et al.*, 2012). Accumulated PCB concentrations are directly proportional to the trophic level, but biomagnification through the food chain increases these concentrations (Te *et al.*, 2020). However, lower-trophic level organisms can bioaccumulate organic contaminants at high concentrations, when they constantly ingest contaminated feed (*e.g.* microorganism, suspended matter) present in their nearby habitats (Wan *et al.*, 2010). In most cases, the xenobiotic accumulates in animal tissues and biomagnify in the aquatic food chain (Dodoo *et al.*, 2013; Huang *et al.*, 2018). As not all ingested PCBs are excreted from the body, some amass in body fatty tissues because of their lipophilic and poor metabolizing nature and long half-lives (Huang *et al.*, 2018; Péan *et al.*, 2013; Ross, 2004).

PCBs bioaccumulate at high or low concentrations in muscle, liver and branchiate of fish with higher concentrations in branchiate or liver due to the permanent contact with contaminated water and high lipid levels (Klinčić *et al.*, 2020). PCBs accumulate in the following order: liver > kidney > lung > eggs > muscle tissues (Storelli *et al.*, 2007; Storelli *et al.*, 2001). The effects of PCB bioaccumulation depend on the aquatic animals' system characteristics such as class or species, gender, age, weight, biomass, metabolism, morphology, physiology, longevity, development stage, mobility, lipid-rich or tissues type and level, food chain position, feeding habits, living habits, body systems condition (reproductive system), and their health status. PCB characteristics such as physico-chemical properties, congener type, source and affinity for lipids as well as environmental characteristics such as habitat conditions, climate, water quality, source pollution concentration, and temporal weather conditions also play a role in the bioaccumulation (Bazzanti *et al.*, 1997; Çelik Çakıroğulları *et al.*, 2010; Delistraty, 2013; Jaffal *et al.*, 2011; Lambiase *et al.*, 2021; Olanca *et al.*, 2014; Storelli *et al.*, 2009; Yaghmour *et al.*, 2020). Level and duration of exposure, position in the food chain and lipid content are the most crucial characteristics (Juei Shen *et al.*, 1998; Storelli *et al.*, 2009; Storelli *et al.*, 2001). Animals living in waters near industrial facilities using POPs are susceptible to bioaccumulate higher concentrations of congeners (Cui *et al.*, 2018).

7.1.2. General PCB Toxicity in Animals

PCBs have disastrous influences on the health of aquatic animals resulting in a delay of their normal growth or development, engender physiological modifications, threaten their survival, or lead to death of tissues and cells (necrosis) (Baldigo *et al.*, 2006; Delistraty, 2013; Hall *et al.*, 2018; Harmon, 2015; Ming-ch'eng Adams *et al.*, 2016; Salice *et al.*, 2014). The endocrine system is made up of tissues that provide necessary enzymes and hormones for the functioning of the entire body system (Baldigo *et al.*, 2006). PCBs are endocrine disruptors, they disturb the functioning of the hormonal system or alter the level of hormones namely androgen, oestrogen, testosterone, corticosterone or thyroid during the interaction with the plasma protein of these hormones; this occurs because their chemical structure is similar to that of hormones (Baldigo *et al.*, 2006; Gonzalez *et al.*, 2016).

PCBs also affect the immunological, developmental, behavioural, neurobehavioral, neurological, mental, cognitive, sensory, motor, ocular or visual, brain, heart, intellectual, cardiac, central nervous, respiratory, dermal, physiological, morphological, peripheral, eye, skeletal, haematological, hepatic, congenital, reproductive, genetic, and sexual systems of both aquatic and terrestrial animals, and they can develop cancers, hearing, birth and sex malformations as well as tumours, mucosa, ulcers, pathologies, infections and cardiovascular diseases (viral, bacterial, fungal and parasites) and lesions (Baldigo *et al.*, 2006; Blanc *et al.*, 2021; Brouwer *et al.*, 1989; Buha Djordjevic *et al.*, 2020; Grilo *et al.*, 2014; Guruge *et al.*, 1997; Luzardo *et al.*, 2014; Péan *et al.*, 2013; Sonne *et al.*, 2020; von Stackelberg, 2019). The visual system is affected with malformations of the retinas whilst the peripheral system is damaged from hair loss in the ear (Harmon, 2015; Hayashi *et al.*, 2015). Suppression of the immune system or immune response by endocrine disruptors renders the organism unable to resist host bodies hence reducing chances of survival (Hall *et al.*, 2018; Roy *et al.*, 2019; Sonne *et al.*, 2020). PCBs can also reduce memory, swimming and learning abilities of aquatic animals (Blanc *et al.*, 2021; Harmon, 2015). Exposure of amphibian animals such as frog, salamander, toad, caecilian, hyla, newt, proteus to PCBs can lead to various health issues such as hypergenesis which is an organ expansion due to tissues growth, ocular anomalies, forelimb and stomach deformities, demasculinization, defeminisation, and weakening of the male voice box (Buha Djordjevic *et al.*, 2020; Harmon, 2015). PCBs can also initiate tension, gene alteration, and DNA mutation (Cocci *et al.*, 2018; Harmon, 2015), disturb DNA methylation resulting in a non-expression of genes such as *CPY1A* in both aquatic and terrestrial animals (Cocci *et al.*, 2018; Hung *et al.*, 2012). This enhances the risk of getting infections and exhibiting symptoms such as diarrhoea, fever, fatigue, weight loss, and headache (Hall *et al.*, 2018; Monnolo *et al.*, 2020; von Stackelberg, 2019). Fat metabolism and calories equilibrium of aquatic animals can be altered, resulting in overweight, anorexia, growth delay, stress, anaemia, and even death (Cocci *et al.*, 2018; Li *et al.*, 2019). Production of enzymes such as glutathione peroxidase (GPx) and glutathione (GSH) can also be inhibited, while activity of biomarkers such as ethoxyresorufin-O-deethylase (EROD) and pentoxyresorufin-O-deethylase (PROD) can be increased (Kodavanti *et al.*, 2010; Kodavanti *et al.*, 1998; Kodavanti *et al.*, 2017). The hepatic effect of PCBs is displayed as an expansion of the liver resulting from an increase of its cells size, a modification of tissues characteristic of a disease, an inability of enzymes to degrade drugs, an alteration of retinoid level in liver and a development of tumours (Roos *et al.*, 2011). Anti-estrogenic effects of non-ortho PCB congeners hinder the production of calcium and reduce vitamin A levels during offspring development, resulting in fragile eggshells (Borja *et al.*, 2005; Brouwer *et al.*, 1989). It also prevents the attachment of oestrogen to the receptors by bonding to the receptor, thus preventing the expected oestrogen response (Johnson *et al.*, 2009). Modulation of oestrogen levels in the body influences foetal bone strength, bone tissue and skeletal development, leading to weak bones or osteoporosis because oestrogen is necessary for the metabolism and equilibrium of bone tissue (Johnson *et al.*, 2009; Lundberg *et al.*, 2006). This is materialized by alteration of bone dimensions, delayed bone development, shortening, dissolution and loss of bones which all result in lowering of bone mineral density (BMD) and bone bending strength (Johnson *et al.*, 2009; Lundberg *et al.*, 2006; Salice *et al.*, 2014; Sonne *et al.*, 2020). A decrease of BMD can also be due to *in utero* and breastfeeding, or elevation of vitamin A (retinol), vitamin C (ascorbic acid) and thyroid hormone levels concurrently with a suppression of vitamin D (calciferol), cortisol and sex steroids hormone levels from PCB-exposure, with humerus and femur being the most affected bones (Johnson *et al.*, 2009; Lundberg *et al.*, 2006; Sonne *et al.*, 2020). Break or weakening of spine, skull, and face bones can also occur in fish from aquatic exposure to PCBs leading up to several body malformations, inability to swim normally, reproductive difficulties and even death (Hung *et al.*, 2012). PCBs can also initiate pericardial effusion and instigate yolk sac oedema in all animals (Harmon, 2015). Bioaccumulation of PCBs in aquatic animal tissues can slow down energy production by either impeding on mitochondrion functioning or disrupting production of lipids such as triglycerides, phospholipids, and sterols affecting reproductive and brain systems commonly seen in male rather than female aquatic animals due to distinct diet habits and body metabolism (Blanc *et al.*, 2021; Jaffal *et al.*, 2011).

7.1.3. Male reproductive system in animals

Estrogenic and anti-estrogenic properties of PCBs are responsible for reproductive system impairment in aquatic animals (Lundberg *et al.*, 2006), the extent depending on the type of congener, route of exposure, sexual maturation and animal age (Kodavanti *et al.*, 2010; Kodavanti *et al.*, 2017). This impairment is usually manifested *via* genital modification and mutilation, whereby sex organs and reproductive processes are modified or altered (Kodavanti *et al.*, 2017; Lundberg *et al.*, 2006).

Fish: Aroclors (1242, 1254 and 1260) can modify the testicle morphology (smaller sizes) and slow down spermatozoids production along with gene modifications, reducing the chances of fertility of aquatic animals such as fish (Kodavanti *et al.*, 2017; Lundberg *et al.*, 2006). Moreover, even when spermatozoids are produced, they might be unable to fecund eggs (Guo *et al.*, 2000; Kodavanti *et al.*, 2017).

Birds: Alteration of the luteinizing hormone (LH) and the follicle stimulating hormone (FSH) concentrations from PCB exposure can prevent the production of testosterone and sperms, delay puberty and development of genital parts of aquatic animals such as seabirds (Lyche *et al.*, 2004).

Mammals: Male aquatic mammals animals are sometimes more susceptible to severe modifications or malformations than females mammals (Kodavanti, 2017; Li *et al.*, 2019; Reddy *et al.*, 2019). The growth of male mammals can be delayed by PCBs while at the same concentration mild effects on female mammals will be induced; yet, effects are generally directly proportional to the concentration and duration of exposure (Li *et al.*, 2019). Milder effects in aquatic oviparous mammals are due to the fact that females can get rid of organic pollutants through production of eggs while male animals cannot (Olanca *et al.*, 2014). Female viviparous mammals can transfer some of the PCB residues to their young ones *via* the umbilical cord, placenta (prenatal transfer), or breastfeeding (postnatal transfer), thus reducing the concentration of PCBs in the mother's organism, whereby usually low chlorinated congeners are transferred (Imaeda *et al.*, 2014; Tanabe *et al.*, 1997).

7.1.4. Female reproductive systems in animals

Fish: Some female aquatic animals such as fish may experience an inability of their sex organs to produce hormones responsible for fertilizable eggs production (Luzardo *et al.*, 2014). An alteration of oestrogen, luteinizing and thyroid hormone concentrations by endocrine-disrupting PCBs may exhibit numerous effects such as early or late maturation of sexual parts, especially during offspring development (Hayashi *et al.*, 2015; Li *et al.*, 2019; Lyche *et al.*, 2004). Hormone concentration variations can delay puberty or the juvenile-life stage of female aquatic animals (Lyche *et al.*, 2004).

Bird: PCBs suppress the reproductive system and the abilities of aquatic female animals, such as seabirds or sea eagles, by preventing them to bread successfully, by reducing their fecundity or fertility, by modulating pregnancy symptoms and by altering the menstruation (Bohannon *et al.*, 2018; Hall *et al.*, 2018; Harmon, 2015; Luzardo *et al.*, 2014; Reindl *et al.*, 2019). A principal cause of infertility associated with PCBs is the imbalance of hormones which prevents ovulation to take place (Buck *et al.*, 2000; Kodavanti *et al.*, 2017).

Mammals: Consumption of food products containing PCB mixtures such as Aroclor 1260 by aquatic mammals such as whales female animals can delay the blastocyst stages preventing the formation or development of embryos or eggs (Kodavanti *et al.*, 2017; Seiler *et al.*, 1994). Effects on viviparous animal offspring are due to the transfer of PCBs *via* the placenta or mother's milk to the offspring, sometimes without DNA alteration (Blanc *et al.*, 2021; Dickerson *et al.*, 2019). This may result in pre-term death or pre-term deliveries of underweight offspring with a reduced growth rate due to affected embryonic development and sometimes dead eggs (Blanc *et al.*, 2021; Harmon, 2015; Nordlöf *et al.*, 2012; Salice *et al.*, 2014). The offspring may also show developmental toxicities, functional disorders, malformations (muscles), organism malfunctioning as well as behavioural, sexual (sex ratio), genital and physiological differences at an early development stage (Dickerson *et al.*, 2019; Harmon, 2015; Péan *et al.*, 2013). Developmental exposure to PCBs can also reduce normal abilities and cause later reproductive impairment (Hayashi *et al.*, 2015). A rare effect is a disorder sex development (DSD), whereby the genetic sex of the foetus is different from their phenotypic sex (Li *et al.*, 2019). Compartmental disorders, morphological abnormalities with several developmental delays are also common (Kodavanti *et al.*, 2019; Ming-ch'eng Adams *et al.*, 2016). Consequently, the gestation period during which the mother encountered PCBs is crucial (Kodavanti *et al.*, 2010; Kodavanti *et al.*, 2017)

7.1.5. Neurobehavioral and Neurochemical Effects in animals

Fish: Nervous, behavioural, and intellectual systems of aquatic animals' offspring can be affected from consumption of PCB-containing food during gestation or lactation by the mother (Chen et al., 1994; Gonzalez et al., 2016; Kodavanti, 2017; Schantz et al., 1995). Prenatal or neonatal exposure of PCBs can engender behavioural deficits particularly Attention Deficit Hyperactivity Disorder (ADHD) at an advanced age (Berger et al., 2001; Branchi et al., 2005; Eriksson et al., 1996; Gonzalez et al., 2016; Kodavanti, 2017).

Bird: PCBs are also able to modify the functioning of the cholinergic system by misleading transduction of action potentials (Eriksson, 1997; Eriksson *et al.*, 1986; Kodavanti *et al.*, 2017). Other systems that are modified are the glutamatergic, GABAergic, and dopaminergic systems (Kodavanti *et al.*, 2017; Myhrer, 2003), the homeostatic control system causing a malfunctioning of cells in the animal body (Kodavanti *et al.*, 2017), and PCB can modulate the hypothalamus-pituitary-adrenal (HPA), which will alter the central stress response system, leading to an inability to raise cortisol and glucose rates in blood during times of stress (Dickerson *et al.*, 2019). Similar effects of PCBs can be manifested in animals at higher trophic levels including terrestrial animals, consumers of small animals at lower trophic levels such as fish or insects living in a polluted aquatic ecosystem caused by PCB biomagnification in the food chain (Dodds *et al.*, 2020; Guruge *et al.*, 1997; She *et al.*, 2008).

Mammals: Consumption of PCB-contaminated food by aquatic mammals or others can seriously damage the neuro-transporter system, namely the vesicular monoamine transporter (VMAT) in synaptic vesicles of presynaptic neurons and plasma membrane dopamine (DA) transporter (DAT) in synaptosomes, causing a disproportion and fluctuations in the DA level and can lead to DA-induced neurotoxicity (Kodavanti *et al.*, 2017; Mariussen *et al.*, 2001a; Mariussen *et al.*, 2001b; Miller *et al.*, 1999). This modifies the production, transporters, and receptors of the dopamine system (Dickerson *et al.*, 2019), and may result in signal transmission distortion between neurons and brain, triggering of the brain's pleasure centre, neurotransmission signal distortion from the body to the brain, and lowering of the hypothalamic hormone concentration (Dickerson *et al.*, 2019; Giesy *et al.*, 1998; Kodavanti *et al.*, 2017; Mariussen *et al.*, 2006; Seegal, 1996). All these effects are susceptible to weaken the physical and mental wellbeing and modify neural circuitries resulting in behavioural and psychological modifications characteristics (Dickerson *et al.*, 2019; Kodavanti *et al.*, 2017). However, effects on the brain depend on the DA concentrations, congener type, route, and duration of exposure (Dickerson *et al.*, 2019; Kodavanti *et al.*, 2017). Dopamine concentration of adults usually decreases when that of offspring increases (Kodavanti *et al.*, 2017; Seegal, 1996; Seegal *et al.*, 2002). Prenatal exposure to PCBs can also result in a decrease of long-term potentiation (LTP) with a reduced number of *N*-methyl-D-aspartate (NMDA) receptor binding sites and is more common in females than in males (Dickerson *et al.*, 2019), which happens also to other biogenic amines namely norepinephrine (noradrenaline) and serotonin (Kodavanti *et al.*, 2017; Lee *et al.*, 2004; Messeri *et al.*, 1997).

7.2. Humans

7.2.1. General aspects of PCB Toxicity in Humans

PCBs present in aquatic environments can be transferred to the human body through several pathways: dermal contact, inhalation, direct consumption of polluted water, or biomagnification in the food chain (Gwenzi *et al.*, 2018; Visha *et al.*, 2018). Dermal exposure of humans to PCBs takes place when PCBs are absorbed by a particular body skin surface (Cao *et al.*, 2019; Heiger-Bernays *et al.*, 2020), which can also take place at contact with contaminated water (Heiger-Bernays *et al.*, 2020). This extent of dermal exposure can be evaluated through the wipe method which is carried out by analysing a wipe passed over a body surface. Females are more susceptible to be affected than males because they have a more fragile skin, and using makeup alters the state of the skin facilitating the absorption of environmental pollutants. Additionally, ingredients of cosmetics such as emulsifiers and preservatives modulate the lipophilic property of the skin (Cao *et al.*, 2019). The presence of PCBs, namely lower chlorinated congeners in the atmosphere from industrial emissions is a pertinent issue as they promote widespread contamination. Indeed, PCBs as part of semi-volatile organic compounds, can evaporate from a primary source to the air (Andersen *et al.*, 2021). Inhalation of PCB-contaminated air is more problematic than dietary intake especially near urbanised and industrialised areas, as it is about a third of the total exposure (Ampleman *et al.*, 2015). That is because PCBs in air can move to food products (WHO, 2005). Moreover, human exposure to atmospheric PCBs is incessant because people must breathe continuously, thus, they are constantly being inhaled and absorbed into the body. As such, inhalation of PCBs by humans is a relevant pathway of exposure (Casey *et al.*, 2022). Air exposure to PCBs causes specific organs failure, initiates diabetes and hypertension and shortens life expectancy (Dziubanek *et al.*, 2017; Wang *et al.*, 2020). Releasing of improperly treated or contaminated water for domestic use, namely drinking water, consumption of fruits and vegetables watered, grown or fertilized with PCB-contaminated water is a toxic pathway (Gwenzi *et al.*, 2018). Therefore, the maximum contaminant level (MCL) of PCBs in drinking water has been established at 500 ng L^{-1} by the United States Environmental Protection Agency (US EPA), as above this concentration, development of cancers is accentuated (Kassegne *et al.*, 2020; Yang *et al.*, 2015b). Consumption of aquatic animals such as fish or other seafood is considered to have several health benefits by most of the research (Antonijevic *et al.*, 2011; Jacobs *et al.*, 2002; Visha *et al.*, 2018). However, when these food sources originate from a polluted aquatic environment, endocrine disruptors such as PCBs (mostly dioxin-like congeners) are taken up and accumulate in the tissues depending on the pollutants concentrations (it is indirectly related to the extent of the health risks). PCBs are then biomagnified through the food chain and transferred to humans where they might initiate cancerous and non-cancerous effects depending on their concentrations and properties (Ameur *et al.*, 2013; Delistraty, 2013; Moraleda-Cibrián *et al.*, 2015; Schnitzler *et al.*, 2011).

Approximately 95% of human exposure to PCBs is through dietary intake of lipid-rich animal food products such as fish (Birgül *et al.*, 2017; Rusin *et al.*, 2019; Zuccato *et al.*, 1999). Consequently, the MPL (maximum permissible limit) of indicator-PCBs (PCB-28, 52, 101, 118, 138, 153, 180) in seafood or aquatic animal food products has been established at 3000 ng g^{-1} w/w in Japan, 2000 ng g^{-1} w/w in the US, 500 ng g^{-1} w/w in China, Australia and New Zealand and 75 ng g^{-1} fish in the *European Union* (Batang *et al.*, 2016; Shang *et al.*, 2016). Acceptable daily intake (ADI) and minimum risk level (MRL) of PCBs accumulated in fish species have been established at 7.4 $\pm$ 8.6 ng g^{-1} per person to prevent PCB-induced effects (Li *et al.*, 2008). MPLs of total PCBs congeners both non-dioxin-like (ndl) and dioxin-like and ndl PCBs congeners have been established at 2000 ng g^{-1} and 3000 ng g^{-1} fresh weight in edible fish by the *US Food and Drug Administration* (FDA) and *Serbian Rulebook* respectively in 1992 (Antonijevic *et al.*, 2011; Davodi *et al.*, 2011).

When PCBs enter humans, they go through the gastrointestinal track and are directed to tissues, fat rich organs or body samples such as blood, breast milk, lymph, glands, and serum; from there, deleterious modifications take place (Heiger-Bernays *et al.*, 2020; Kodavanti *et al.*, 2019; Ottonello *et al.*, 2014; Ssebugere *et al.*, 2019). Different PCB effects are from distinct interactions with body tissues or fluids (Ming-ch'eng Adams *et al.*, 2016). Consequently, inspection of food and risk assessment of organic pollutants is important to avoid harming humans (Bursian, 2007; Li *et al.*, 2017; Reddy *et al.*, 2019).

Toxicological effects of PCBs on humans depends on the congener properties, environment, route, dose and duration of exposure, sex, age, dietary habits, body metabolism and body systems (Bernard *et al.*, 2002; Kodavanti *et al.*, 2017; Müller *et al.*, 2001; Van den Berg *et al.*, 1994; von Stackelberg, 2019). Dioxin-like congeners (endocrine disruptors) effect the operation of the aryl hydrocarbon receptor (AhR), while this is not the case of non-dioxin-like congeners (neurotoxic). However, non-dioxin like congeners are the most abundant in human and environmental samples, but aquatic exposure mostly subjects humans and marine animals to dioxin-like PCBs (Buha Djordjevic *et al.*, 2020; Esposito *et al.*, 2020; Greenfield *et al.*, 2013; Olanca *et al.*, 2014; Roos *et al.*, 2011; Xu *et al.*, 2019). While ortho congeners (PCB-18, 31, 44, 48, 52, 70, 99, 101, 126,136,153 and 188) are known to be estrogenic, non-ortho congeners (PCB-105, 114, 126 and 169) are anti-estrogenic (Yao *et al.*, 2017). Estrogenic congeners can modulate the menstrual cycle, replace the oestrogen hormone, and impair the reproductive and sexual systems (Kodavanti *et al.*, 1998; Pflieger-Bruss *et al.*, 2004). They also affect motor, intellectual and neurological systems of new born from pre-natal exposure *via* the mother (Reddy *et al.*, 2019). The individuals' gender influences physiological manifestations of chronic and cardio-vascular diseases caused by PCBs, their extent and development; hence, they are called sex-specific chemicals. This is generally due to differences in body reactions taking place after the association of PCBs with body substances and sex hormones of either males or females. Men can easily have hypertension while women can easily contract diabetes (Wahlang *et al.*, 2019).

The bioaccumulated PCBs damage neurodevelopmental, neurobehavioral, neurological, neurobiological, neuropsychological, psychological, developmental, mental, intellectual, central nervous, immunological, sexual differentiation, skeletal, lymphoid, haematological, reproductive, embryonic, hormonal, dermal, endocrine, peripheral, oral, ocular, sexual, and entire body systems (Burns *et al.*, 2020; Butterworth *et al.*, 1995; Cui *et al.*, 2018; Dickerson *et al.*, 2019; Fimm *et al.*, 2017; Hayashi *et al.*, 2015; Helou *et al.*, 2019; Li *et al.*, 2012; Luo *et al.*, 2007; Qiu *et al.*, 2019; Xu *et al.*, 2019; Zhong *et al.*, 2020; Zuccato *et al.*, 1999). Neurodevelopmental dysfunctions can lead to individuals becoming indelible alcoholic or drug users (Dickerson *et al.*, 2019). Disruption of the central nervous system may result in paralysis, headache, weakness, dizziness, vomiting, speech impediment, depression, nervousness, and tiredness (Borja *et al.*, 2005; Kodavanti, 2017; Yang *et al.*, 2015a). Modulation of oestradiol hormone levels by PCB congeners such as PCB-126 can generate endometriosis (Bartalini *et al.*, 2019; Yao *et al.*, 2017). Interference with the thyroid hormone level causes thyroid, liver, lung, oral cavity and skin tumours, intellectual, and physical disabilities (Buha Djordjevic *et al.*, 2020; Hoogenboom, 2012; Lerro *et al.*, 2018; Rusin *et al.*, 2019; Zani *et al.*, 2019). PCBs can also cause anorexia, bloating, body pains, and megalohepatia (Te *et al.*, 2020).

PCBs can act in several ways either by producing sensitive oxygen species (carcinogenic), by developing tumours (oncogenic), by altering genes (genotoxic), by modifying DNA or genes (mutagenic), by altering the oestrogen hormone level (estrogenic), by affecting brain or nerves (neurotoxic), by suppressing the immune system (immunotoxic), by causing physiologic abnormalities (teratogenicity), by damaging cell or tissues or by binding to receptors (cytotoxicity or apoptotic), by interfering with normal reproduction (reprotoxic), by initiating embryo deformities or death (embryotoxic), by acting *via* the synapses (synaptic), by injuring the liver (hepatotoxic), or by damaging organs (toxic) (Butterworth *et al.*, 1995; Dodoo *et al.*, 2012; Fernlöf *et al.*, 1997; Font *et al.*, 1996; Gwenzi *et al.*, 2018; Iwegbue *et al.*, 2020; Li *et al.*, 2012; Park *et al.*, 2020; Reddy *et al.*, 2019; Rusin *et al.*, 2019; Wang *et al.*, 2019b; Yao *et al.*, 2013; Zacs *et al.*, 2015). Individual congeners, especially highly chlorinated congeners, are known to actuate and stimulate growth of lifetime cancers, namely melanoma skin cancer, gall bladder cancer, brain cancer, prostate cancer, pancreas cancer, liver cancer including clinical hepatitis, biliary tract cancer, gastrointestinal tract cancer, stomach cancer, intestines cancer, ovarian cancer, and breast cancer (Magoni *et al.*, 2019; Niehoff *et al.*, 2020; Park *et al.*, 2020; Reddy *et al.*, 2019; von Stackelberg, 2019; Wahlang *et al.*, 2019). Of all these, liver cancer is the most common cancer caused by exposure to PCBs, because they tend to mostly accumulate in the liver; additionally, the liver is responsible for POPs' management and excretion (Di Bella *et al.*, 2006). Women readily develop uterine, thyroid and cervical cancers, while men develop testicular, lung, lymphatic, and rectum cancers (Helmfrid *et al.*, 2012; Lerro *et al.*, 2018; Li *et al.*, 2012), with men succumbing to death more often than women (Koss *et al.*, 1999). Cancer development is initiated when the PCBs produce reactive elements that obstruct the body's normal functioning by attacking cells or organs (Niehoff *et al.*, 2020). This can occur *via* the attachment of PCBs on particular sites, the interaction of PCBs with body substances or the biological accumulation of PCBs in adipose tissues (von Stackelberg, 2019). For instance, instigation and growth of breast cancer results from a long time or early exposure to anti-estrogenic PCBs that damages breast tissues and the mammary gland (Guo *et al.*, 2020; Parada *et al.*, 2020; Parada *et al.*, 2016). PCBs can also cause various chronic, cardiovascular, metabolic and endocrine diseases such as: stroke, hypertension, dyslipidaemia, mononeuritis, Alzheimer, blood deficiency, paraesthesia, adenoma, hepatitis, diabetes type II, gastrointestinal malfunctioning, thyroid defects as well as regenerative and formative illness (Kodavanti *et al.*, 2017; Park *et al.*, 2020; Peinado *et al.*, 2020; Peters *et al.*, 2014; Raffetti *et al.*, 2020; Wahlang *et al.*, 2019; Yadav *et al.*, 2017; Yang *et al.*, 2015a). Exposure to PCB-containing oils can cause skin alterations, irritations or colorations, acne, severe chloracne, inflammations with inconsistency, malformed nails, dermal lesions, struma, hyperpigmentation, rashes, infections, allergies, itching eyes, hair loss, and pigmentation (Hayashi *et al.*, 2015; Magoni *et al.*, 2019; Müller *et al.*, 2017; Parada *et al.*, 2020; Peinado *et al.*, 2020; Schettgen *et al.*, 2018; Yang *et al.*, 2015a). Nevertheless, many toxicological effects of PCBs are still under investigation, namely their diabetic effect and the Non-Hodgkin-lymphoma effect caused by polycythaemia (Paliwoda *et al.*, 2016; Schettgen *et al.*, 2018). Yet, it is known that this lymphoma results from modifications of the immune system and PCBs are known to exhibit immunotoxic effects weakening the immune system(Maifredi *et al.*, 2011).

7.2.2. *Human male reproductive system*

PCBs in males can retard the production of male proteins and hormones (testosterone) leading to abnormal sexual and genetics development (Buha Djordjevic *et al.*, 2020; Kodavanti *et al.*, 2017; Pflieger-Bruss *et al.*, 2004). Male can also experience low sperm count and altered spermatozoid abilities and physical morphology (Buha Djordjevic *et al.*, 2020; Kodavanti *et al.*, 2017; Pflieger-Bruss *et al.*, 2000). Sometimes, the development of the genetics is modified and their entire body development accelerated (Reddy *et al.*, 2019). This results in young males exhibiting adult males sexual characteristics (Reddy *et al.*, 2019). In some cases, the expression of genes is weak, instigating demasculinization (Buha Djordjevic *et al.*, 2020; Ssebugere *et al.*, 2019).

7.2.3. *Human female reproductive system*

PCBs tend to affect the genitourinary or urogenital system of females by reducing their chance of conception or by modulating their pregnancy period when they have been exposed to a PCB-contaminated environment before the age of five (Bursian *et al.*, 2011; Helmfrid *et al.*, 2012; Peinado *et al.*, 2020). This disruption of the female reproductive system may be materialized by an irregular menstrual cycle or menstruation period, irregular bleeding, incapacity to carry a pregnancy to term, low body weight gain during pregnancy as well as pre-term or post-term deliveries (Bursian *et al.*, 2011; Müller *et al.*, 2017). PCBs can disrupt the mother's thyroid hormone level affecting normal progress, maturation and development of foetus systems and initiate birth and growth malformations (Buha Djordjevic *et al.*, 2020; Guo *et al.*, 2020). When a pregnancy succeeds to come to term, the following effects have been observed: stillbirths or new-borns with impeded foetal development babies with birth defects and reduced metabolism, affected growth and motor development *i.e.* abnormal weight (obesity or anorexia), height, skull and chest circumference (Lynch *et al.*, 2012; Miyashita *et al.*, 2015). New-borns can also have several serious disabilities or delay behavioural, verbal, and mental, memory, neuro-developmental, developmental, neurobehavioral, neurological, nervous physiological, psychomotor, hormonal, or intellectual as well as functional alterations. These effects can be severe when the mother accumulated large amounts of organic pollutants in her tissues and transferred them to the foetus at developmental stages (Antonijevic *et al.*, 2011; Batterman *et al.*, 2009; Koss *et al.*, 1999; Li *et al.*, 2012; Lynch *et al.*, 2012; Miyashita *et al.*, 2015).

The reason why PCBs are more dangerous to children is because their organism is not mature enough to fight potential diseases and their diet is restrained (Jacobs *et al.*, 2002). PCBs reach the foetus when contaminated food goes through the umbilical cord and encounters the embryo *via* the placenta during blood exchange (Dickerson *et al.*, 2019; Koss *et al.*, 1999; Reddy *et al.*, 2019). This damages the fragile foetal organs and tissues such as pancreatic islets even at low PCB concentrations. They can also alter the body metabolism that limits the development of chronic diseases such as diabetes I and II (Müller *et al.*, 2017; Timme-Laragy *et al.*, 2015). PCBs are capable of varying sex-hormones and the calcium concentrations of the embryo and they can also reach the infant through breastfeeding, which is the main pathway of children exposure to organic pollutants (Ssebugere *et al.*, 2019). Utero, prenatal or trans-placental exposure is generally more devastating than lactational, neonatal or early-life exposure because benefits of breastfeeding can outweigh the detriments of PCBs (Gonzalez *et al.*, 2016; Müller *et al.*, 2017). Deficits in the mechanical, physiological, cognitive, neurodevelopmental, and behavioural system may be noted from embryonic exposure (Gonzalez *et al.*, 2016; Lynch *et al.*, 2012). Those deficits can move from childhood to adulthood, resulting in several physical malformations or disease development such as childhood leukaemia (Antonijevic *et al.*, 2011; Burns *et al.*, 2020; Gonzalez *et al.*, 2016; Shen *et al.*, 2019). Consumption of PCB contaminated food in addition to a consumption of alcohol by the mother can even enhance the foetal alcohol syndrome (Buha Djordjevic *et al.*, 2020; Dickerson *et al.*, 2019). Contact with organic pollutants at even low concentrations can accelerate the breast maturation of young females and disturb their sexual system (Croes *et al.*, 2014; Den Hond *et al.*, 2002; Kodavanti *et al.*, 2017).

7.2.4. Dental system

Ingestion or exposure to PCB-containing materials can initiate several dental and enamel defects (Sonne *et al.*, 2020). Consumption of PCB-containing food such as rice oil can lead to periodontitis, whereby the teeth are infected leading to shorter gums or teeth lost (Kodavanti *et al.*, 2017; Shimizu *et al.*, 1992; Sonne *et al.*, 2020). Children can also get caries, and adults can develop de-colouring and inflamed gums (Guo *et al.*, 1999; Kodavanti *et al.*, 2017).

7.2.5. Neurobehavioral and Neurochemical Effects

PCB congeners can delay the development, comportment, neurodevelopment, neurological, psychomotor, memory and intellectual abilities of an individual at contact during adulthood or childhood (Ennaceur *et al.*, 2008). This can lead to a reduced ability motor and brain function of hands or brain during adolescence and adulthood (Kodavanti *et al.*, 2017; Newman *et al.*, 2006; Rogan *et al.*, 1988; Schantz *et al.*, 2001). Adolescents that have been previously exposed to PCBs such as PCB-66 in their childhood showed a higher likelihood to become lifetime alcohol and drug users compared to a control group, and they may also be incapable to make decisions on their own or modulate their pleasures (Dickerson *et al.*, 2019). Pins-and-needles sensation, numbness and weakness show an affected peripheral nervous system, whilst reduced ability to move, memory loss and loss of sensation show a damaged central nervous system from consumption of PCB-contaminated food (Fonnum *et al.*, 2009; Kodavanti *et al.*, 2017; Newman *et al.*, 2006). Some people can develop hyperactivity or ADHD-like (Attention Deficit Hyperactivity Disorder) symptoms (Bowman *et al.*, 1981; Gonzalez *et al.*, 2016; Hardell *et al.*, 2002; Heiger-Bernays *et al.*, 2020; Kodavanti *et al.*, 2017), and they are sometimes unable to concentrate and control their emotions (Dickerson *et al.*, 2019).

7.3. Plants

Historical anthropogenic activities using organic pollutants such as PCBs have engendered the destruction of several environmental matrices due to their eco-toxicological effects (Li *et al.*, 2017). The presence of PCB-91, 95, 136, 149, 176 and 183 in water sediments can result in plant cell poisoning from the adsorption of PCBs by aquatic plants such as eelgrass, water lettuce, blue iris, coontail, and musk grass (Dai *et al.*, 2014). Even though the concentration of accumulated PCBs by plants from sediments is usually very low, it becomes stable after some days depending on the type of plants (Dai *et al.*, 2014). Poisoning of aquatic plants can take place through up-taking of PCB-sorbed soil organic matter (SOM), hereby limiting the amount of non-contaminated soil available to plants and microorganisms for uptake (Reddy *et al.*, 2019). Poisoning of plants first takes place on the root surface followed by the root system (stems and leaves) and finally the plant cells. In addition, PCBs can be adsorbed by leaves and stems and get distributed to the plant tissues (Dai *et al.*, 2014; Reddy *et al.*, 2019). Therefore, the concentration of PCBs is higher in the roots because that is the first point of contact of PCBs with plants (Dai *et al.*, 2014). The concentration of PCBs in aquatic plant parts usually increases with time (Dai *et al.*, 2014). Adsorption of PCBs on the plant roots has negative effects on organisms (*e.g.* animals, fungi and bacteria) because they use plant materials as feed (Reddy *et al.*, 2019). PCBs in plant tissues alter the plants' processes namely its productivity, photosynthesis (by lowering concentration of chlorophyll), photomorphogenesis, photoperiodism, biosynthesis, genotype, composition, and DNA as well as several biological processes in the plants (Borja *et al.*, 2005; Reddy *et al.*, 2019). The composition and genetic identity of leaves can also be modified (Zhang *et al.*, 2017). All these transformations will render plants unable to incorporate nutrients and energy, generate their own food using available environmental resources, to grow normally due to physical changes (inhibited growth) and to metabolize organic compounds (Reddy *et al.*, 2019). Non-aquatic plants such as tobacco plants that were in contact with PCBs in soil had a delay in blooming and their leaves were found to be deformed, decoloured and malformed (Dai *et al.*, 2014; Reddy *et al.*, 2019). Additionally, processes such as root and stem growth, seed germination, leaf growth and bud opening can be delayed (Reddy *et al.*, 2019). Generally, the extent of effects caused by PCBs depends on the congener's physico-chemical properties, the plant's biological conditions as well as environmental conditions. Congener properties such as molecular weight and lipophilicity are crucial, as highly hydrophobic congeners with a molecular weight lower than 1000 will readily be absorbed via the roots, whilst less hydrophobic congeners with a molecular weight lower than 1000 will readily be absorbed *via* the foliage. The concentration of the congener and the fat content of the plant as well as the pH and the organic matter concentration are important factors because high fat content plants tend to accumulate elevated amounts of PCBs (Zhang *et al.*, 2017).

8. Conclusion

Improper disposal, accidental spillage and discharge of PCB-containing materials or effluents associated to their long-range atmospheric transport have resulted in marine pollution as well as several devastating eco-toxicological effects on humans, animals, and ecosystems. The presence of PCBs in marine ecosystems reduces the abundance of aquatic animals. It is clear from this review that the most deleterious toxicological effects of PCBs are to humans from the consumption of mariculture products after their bioaccumulation and biomagnification through the aquatic food chain. Albeit aquatic animals' exposure to environmental pollutants such as PCBs can be reduced if preventive measures are taken to ensure good aquatic environment quality or conditions to which aquatic animals will be exposed. This can be done through frequent monitoring of PCB congeners especially dioxin-like congeners and close regulation of industrial wastewater discharges in aquatic systems. However, if environmental pollution already exists, one should limit the aquatic animals' exposure to polluted environments, provide healthy feed for aquatic animals and decontaminate the polluted environment. Sometimes, chemical adsorbents such as charcoal and activated carbon can be added to contaminated feed to facilitate secretion of contaminants by glands or cells and subsequent defecation. On the other hand, human health can be protected by following aquatic animal consumption advisories set by states and federal regulatory bodies as well as complying with the jurisdictions. Humans can also try as much as possible to avoid eating animal products that are known to accumulate POPs. Nevertheless, it should be noted that health effects of PCBs are not congruous. Indeed, an exposure to an identical PCB-containing element can render various: the differences may lie in the body organism, congener properties, or even environmental conditions.

One recommendation from this review is that, to protect the public and environment's health, the presence or occurrence of PCBs, their concentrations, distributions, and spatiotemporal trends in marine ecosystems need to be monitored to evaluate or assess their potential biological risks on humans and the biota. Indeed, evaluating the chemical contamination or toxicological assessment can help determining the amounts of PCBs that can bioaccumulate in aquatic animal tissues because they are proportional to the marine pollution status. Indirectly, this can also control human exposure to PCBs. Additionally, it must be emphasized that the analysis of PCBs in aquatic environments or aquatic biota is important to establish future directives, recommendations and decisions related to environmental conservation to avoid ecological and economical failures. Finally, active, and passive treatment technologies or activated carbon remedial techniques can be put in place to improve the quality of aquatic ecosystems. Yet, adsorption of PCBs to activated carbon guarantees a long-term efficiency for the biodegradation of PCBs in soils and sediments. This remediation strategy will not only reduce the bioavailability of pollutants but also the toxicity of the medium. However, the level of sorption relies on the properties or characteristics of the matrix.

Statements and Declarations

Availability of data and material

Not applicable.

Code availability

Not applicable.

CRediT authorship contribution statement

Prisca Stephanie Kandjo Ngoubeyou: Conceptualization, Funding acquisition, Writing – original draft. **Christian Wolkersdorfer:** Funding acquisition, Project administration, Supervision, Writing – review & editing. **Peter Papoh Ndibewu:** Conceptualization, Supervision, Writing – review & editing. **Wilma Augustyn:** Supervision, Writing – review & editing.

Declaration of Competing Interest

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

Data availability

No data was used for the research described in the article.

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