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Human Health Effects of Polychlorinated Biphenyls: From Exposure Pathways to Clinical Evidence

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***Abstract*—Polychlorinated biphenyls (PCBs) are synthetic chlorinated hydrocarbons that were once widely used in electrical equipment, building materials, and industrial fluids due to their chemical stability and dielectric properties. These same characteristics, however, have rendered PCBs among the most persistent environmental pollutants. Due to their resistance to degradation, PCBs persist in soils, sediments, and food webs, remaining detectable in human tissues even decades after their production ban. Both legacy PCBs and unintentionally produced congeners continue to circulate globally, leading to persistent, low-level human exposure. Their lipophilic nature promotes accumulation in adipose tissue and breast milk, enables placental transfer, and results in biological half-lives measured in years, creating a multigenerational toxic burden. This review summarizes recent evidence on the health impacts of PCBs on humans. We highlight clinical and epidemiological findings from the past decade, integrated with mechanistic insights that explain how PCB exposure contributes to disease. Particular emphasis is placed on carcinogenesis, reproductive and developmental toxicity, endocrine disruption, neurodevelopmental and neurodegenerative outcomes, immune dysfunction, and cardiometabolic disorders. Across these domains, converging mechanisms, including oxidative stress, endocrine receptor interference, calcium imbalance, immune modulation, and epigenetic alterations, offer biological plausibility for the observed health outcomes. Despite global bans, PCB residues persist as a significant public health concern. The persistence of PCBs underscores the need for continued biomonitoring, mechanistic research, and effective risk communication. Insights from PCB toxicology provide a framework for managing other persistent organic pollutants, reinforcing the importance of proactive environmental health strategies to safeguard future generations.**

***Keywords*—Persistent organic pollutants; Exposure pathways; Bioaccumulation; Toxicological mechanisms; Toxic health outcomes**

I. Introduction

Polychlorinated biphenyls (PCBs) are synthetic chlorinated hydrocarbons produced in large volumes throughout the mid-20th century for use in electrical transformers, capacitors, construction materials, and various industrial applications. Structurally, they consist of a biphenyl backbone with one to ten chlorine substitutions, resulting in 209 possible congeners with diverse physicochemical properties [1]. Their chemical stability, non-flammability, and dielectric performance made them attractive for industry, but the same traits underlie their environmental persistence and tendency to bioaccumulate [2]. By the late 1970s, mounting evidence of toxicity had prompted national bans, and the Stockholm Convention of 2004 established a global framework for the elimination of PCBs. Nevertheless, substantial reservoirs of legacy PCBs persist in soils, sediments, and aquatic ecosystems, while non-legacy congeners continue to be unintentionally generated in the manufacture of pigments and dyes, ensuring that exposure persists in many regions [3]. Consequently, PCB residues are still detectable in the food chain and in human tissues worldwide, decades after formal production ceased.

From a health perspective, PCBs are classified as persistent organic pollutants (POPs) with well-documented multisystem toxicity. The International Agency for Research on Cancer has classified PCBs as Group 1 human carcinogens, reflecting robust epidemiological and mechanistic evidence. Earlier mass poisoning events, most notably the Yusho incident in Japan (1968) and the Yu-Cheng incident in Taiwan (1979), provided dramatic evidence of acute toxicity, with chloracne, hepatic dysfunction, and reproductive abnormalities as hallmark features [4]. Long-term, low-level exposures have been associated with cancers, endocrine and reproductive disorders, neurodevelopmental deficits, immune dysfunction, and cardiometabolic disease [5]. Mechanistically, PCB congeners act through multiple toxicological pathways. Dioxin-like congeners, which adopt a planar structure, bind the aryl hydrocarbon receptor (AhR) and trigger oxidative stress, dysregulated gene expression, and inflammatory cascades [6]. Non-dioxin-like congeners, in contrast, disrupt intracellular calcium signaling through ryanodine receptor activation, alter thyroid hormone transport, and interfere with steroid receptor pathways. Hydroxylated metabolites (OH-PCBs) introduce an additional layer of complexity, as they can

mimic endogenous hormones, compete for transport proteins, and disrupt metabolic enzyme systems [7]. Together, these mechanisms create a strong biological rationale for the wide spectrum of health outcomes associated with PCB exposure.

In recent years, advances in biomonitoring and epidemiology have clarified the scope of these risks. High-resolution analytical methods now permit precise, congener-specific quantification in human blood and tissues [8], while large longitudinal cohorts have linked PCB biomarkers to cancer, thyroid dysfunction, neurocognitive impairment, and cardiovascular disease [9]. At the same time, mechanistic studies continue to uncover roles for PCBs in oxidative stress, mitochondrial injury, immune cell dysfunction, and epigenetic reprogramming. The purpose of this review is to integrate these contemporary findings, with a focus on evidence. We organize the discussion by health outcome, including cancer, reproductive and developmental toxicity, endocrine disruption, neurotoxicity, immunotoxicity, and cardiometabolic disease, and highlight mechanisms that connect PCB exposure to clinical manifestations. Particular attention is given to vulnerable populations such as infants, pregnant women, and occupationally exposed groups. Finally, we outline future research priorities, including the roles of mixtures and metabolites, gene–environment interactions, and the influence of climate-driven re-mobilization of legacy pollutants.

II. Chemical Properties and Environmental Persistence of PCBs

PCBs consist of two benzene rings joined by a single bond, with up to ten chlorine substitutions ($C_{12}H_{10-n}Cl_n$). Variation in the number and position of chlorine atoms at the ortho, meta, and para sites produces 209 congeners, each with distinct physicochemical properties (Fig. 1) [2].

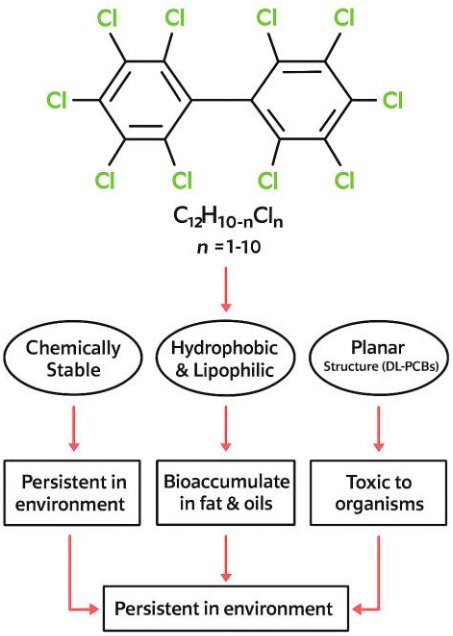

Fig. 1. *Key chemical properties and environmental behaviors of PCBs.*

Increasing chlorine substitution enhances lipophilicity and thermal stability, while simultaneously reducing water solubility and vapor pressure. These characteristics made PCBs attractive for industrial uses requiring durability and non-flammability. Once released, PCBs exhibit remarkable resistance to degradation. Their stability against heat, acids, and bases, combined with limited microbial biodegradability, allows them to persist for decades. Lower-chlorinated congeners are relatively volatile and undergo long-range atmospheric transport, whereas highly chlorinated congeners display low volatility, strong hydrophobicity, and a high affinity for soils and sediments. This partitioning into organic matter creates long-term environmental reservoirs that gradually reintroduce PCBs into the biosphere [3]. For example, atmospheric monitoring continues to detect PCB deposition in remote Arctic regions, even decades after production bans [10].

The toxicological significance of PCBs is strongly linked to their structural subclasses. Dioxin-like congeners, typically characterized by no or a single ortho substitution, adopt planar conformations that bind to the AhR, eliciting dioxin-like toxic responses. Highly toxic congeners in this category include PCB-126, PCB-77, and PCB-169 [11]. Non-dioxin-like congeners, containing multiple ortho-substitutions, are non-planar and do not activate AhR directly. Instead, they exert toxicity through alternative pathways, such as the perturbation of ryanodine receptor–mediated calcium signaling and the disruption of thyroid hormone binding proteins [12]. These divergent mechanisms indicate that no single congener or pathway can fully predict the toxicological profile of PCB mixtures.

Bioaccumulation is another defining property of PCBs. Their strong lipophilicity drives preferential storage in adipose tissue and breast milk, with human half-lives for some congeners extending over years to decades [13]. Biomagnification further amplifies their persistence, as PCBs accumulate along aquatic food webs, reaching peak concentrations in long-lived predatory species and ultimately entering the human diet through fish, seafood, meat, and dairy products [14]. Notably, serum PCB levels in humans often reflect cumulative dietary intake of higher-chlorinated congeners such as PCB-118, -138, -153, and -180, which are widely used as marker compounds in epidemiological studies. Although degradation can occur through processes such as photolysis or reductive dechlorination in anaerobic sediments, these pathways are typically slow and incomplete. Estimates suggest that the majority of the global PCB inventory produced in the 20th century remains present in some environmental compartment [2]. This persistence guarantees continued human exposure, including in populations geographically distant from original manufacturing or use sites.

III. Routes of Human Exposure to PCBs

Understanding the pathways by which PCBs enter the human body is essential for interpreting epidemiological evidence and designing effective public health interventions. Although production ceased decades ago in most countries, several exposure routes remain highly relevant. The principal pathways include dietary ingestion, inhalation, ingestion of contaminated dust or soil, dermal absorption, and maternal transfer (Figure 2). Each pathway contributes differently depending on geography, occupation, lifestyle, and age.

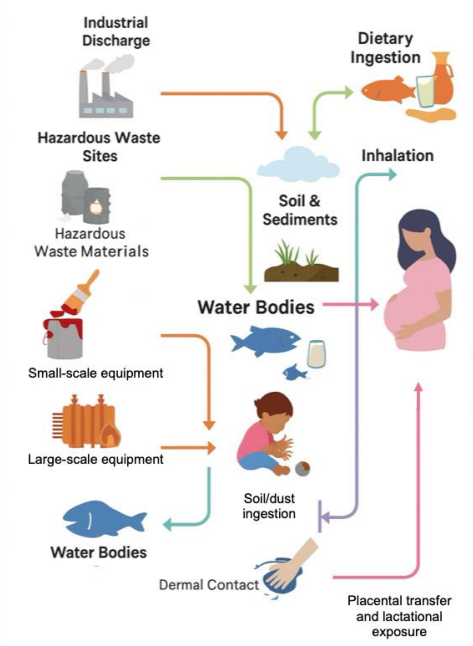

Fig. 2. *Schematic representation of major environmental sources and exposure pathways of PCBs in humans.*

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A. Dietary Uptake from Contaminated Foods

Dietary intake remains the predominant exposure pathway for the general population. PCBs bioaccumulate in fatty animal tissues and biomagnify up aquatic food webs, which makes fish and seafood the primary dietary sources. Numerous studies in contaminated regions, such as the North American Great Lakes, have shown that frequent consumption of locally caught fish is strongly linked to elevated PCB body burdens [15]. Even in the absence of obvious pollution "hot spots," background dietary intake contributes substantially to chronic exposure. A state-of-the-science review estimated that approximately 85–90% of total adult PCB intake comes from food, particularly fish, dairy, and meat products [3], [16]. Recent biomonitoring data corroborate this conclusion. For example, anglers consuming fish from the Upper Niagara River were found to carry serum PCB levels exceeding national averages, with several species (carp, bass, bullhead) still containing residues above safety thresholds decades after bans [17]. Moreover, high-fish-consuming populations, such as subsistence fishing communities or certain Indigenous groups, consistently exhibit PCB concentrations far above background levels [18].

B. Environmental Sources and Inhalation

Although PCB production was banned decades ago, environmental reservoirs continue to act as significant long-term sources of exposure. PCBs re-enter soil, water, and air through multiple processes, including leakage from obsolete electrical transformers and capacitors, inadequate hazardous waste management, illegal dumping, and incineration of PCB-containing materials [19]. Once released, PCBs do not readily degrade; instead, they cycle repeatedly between environmental compartments. Field studies provide clear evidence of this persistence and cycling. In Turkey, measurements in industrial areas reported PCB concentrations of approximately 1,300 pg/m³ in ambient air, 169 ng/L in water, and 71 ng/g in sediments, illustrating active volatilization and redeposition between media [20]. Lighter congeners, owing to their higher volatility, can travel long distances in the atmosphere. Monitoring in the Arctic consistently documents annual deposition fluxes of 100–300 ng/m²/year, with no evidence of a clear downward trend despite global bans, highlighting the role of long-range transport [10]. Aquatic environments act as key sinks. PCBs accumulate in sediments, where they persist for decades, and then re-enter the food web through benthic organisms, progressively biomagnifying in higher trophic levels [21]. On land, PCBs in soils may be taken up by crops or bind to dust particles, creating additional exposure routes for nearby residents [3]. Together, these findings demonstrate that environmental contamination is not static but dynamic, with reservoirs serving as ongoing sources of human exposure. Both indoor and outdoor air constitute additional relevant pathways. Buildings constructed prior to PCB bans often contain caulks, paints, and sealants that slowly release PCBs into

indoor air. Concentrations indoors can exceed outdoor levels by a factor of 10. In occupational settings, such as the demolition of contaminated structures, exposure can be substantial. For example, demolition workers experienced a more than fourfold increase in blood PCB levels within a year, even when they used protective equipment [22]. These observations highlight the importance of considering inhalation as a significant exposure pathway in both residential and occupational settings.

C. Soil, Dust, and Dermal Contact

Children are especially vulnerable to incidental ingestion of contaminated dust or soil. Hand-to-mouth behavior, combined with PCBs adhering to household dust particles, has been recognized as a contributor to early-life exposures in polluted regions [23]. Although this pathway is generally minor compared with diet and inhalation, it may be locally significant near hazardous waste sites or in residences with PCB-containing building materials. Dermal absorption is typically less important, as intact human skin provides substantial resistance to PCB penetration. However, occupational contexts such as electrical maintenance, transformer disposal, or dredging of contaminated sediments may entail direct dermal contact with PCB mixtures, leading to measurable uptake [24]. These occupational exposures historically accounted for some of the highest PCB body burdens recorded.

D. Maternal Transfer and Intergenerational Exposure

PCBs accumulated in maternal adipose tissue can cross the placenta and accumulate in the fetus during pregnancy. They are also secreted in breast milk, creating an additional transfer route during early life [25]. Although breastfeeding remains strongly recommended for its numerous health benefits, these findings highlight the persistence of PCBs in human populations and their potential for intergenerational transmission. Studies consistently demonstrate that PCB concentrations in cord blood and breast milk correlate with maternal body burdens, directly linking maternal diet and environment to neonatal exposure [14], [26].

IV. Health Effects of PCBs on Humans

A. Carcinogenic Effects

Recent evidence indicates that PCB exposure contributes to carcinogenesis across multiple organ systems. Epidemiological investigations, including large cohort studies and case–control analyses, have linked PCBs to elevated risks of cancers of the liver, gastrointestinal tract, lung, skin, and hematopoietic system. For example, a Danish cohort of approximately 38,600 residents chronically exposed to PCB-contaminated indoor air from aged building materials exhibited significantly higher overall cancer incidence, including nearly a threefold increase in liver cancer risk and elevated hazards for pancreatic and brain tumors [27]. Similarly, a 50-year follow-up of Japan's Yusho poisoning cohort found excess mortality from liver and lung cancers, with liver cancer mortality especially increased among female patients and lung cancer among male patients [28]. In Italy, a case–control study conducted in the heavily polluted city of Brescia found that individuals with the highest serum PCB levels had nearly 1.8-fold greater odds of developing hepatocellular carcinoma compared with those with the lowest levels [1]. In China, a large 10-year prospective study of adults with type 2 diabetes found that higher serum concentrations of non-dioxin-like PCBs were associated with significantly greater incidence of gastrointestinal cancers (particularly liver and colorectal tumors) as well as lung cancer [29]. These cancer risks were particularly pronounced among participants with unhealthy lifestyles such as poor diet, smoking, or physical inactivity, suggesting a potential synergistic effect. The link with lung cancer is further supported by the Yusho cohort's elevated lung cancer mortality, as well as by mechanistic studies implicating PCB-induced chronic respiratory inflammation and oxidative stress [28], [30]. Cutaneous melanoma is among the most consistently observed malignancies associated with PCB exposure. A Canadian case–control study of women found that high plasma concentrations of several PCB congeners (e.g., PCB-138, -153, -180) were associated with more than a twofold increase in melanoma odds [31], corroborating earlier findings of elevated melanoma risk in PCB-exposed occupational cohorts. In contrast, a general-population study in northern Italy found no significant association between current plasma PCB levels and melanoma incidence [32], suggesting that exposure timing or modifying factors such as UV radiation and genetic susceptibility may influence risk. Beyond melanoma, immunologically related cancers such as non-Hodgkin lymphoma (NHL) have also been examined. An Israeli case–control study observed that elevated serum concentrations of higher-chlorinated congeners, particularly PCB-146 and PCB-156, were associated with roughly 70–75% higher odds of NHL [33], consistent with the immunosuppressive properties of PCBs. Although some studies and meta-analyses have yielded mixed results, and IARC still classifies the human evidence for NHL as limited, recent findings suggest a modest increase in NHL risk associated with PCB exposure [34]. Additional observations include possible links to meningioma and testicular cancer in the aforementioned Danish cohort, although those associations were based on small case numbers and require confirmation in further studies [27].

Mechanistic research offers strong biological plausibility for the epidemiological associations described above. PCBs and their metabolites generate reactive oxygen species (ROS) that can damage DNA, proteins, and lipids; chronic oxidative stress, in turn, activates NF-κB and other pro-inflammatory signaling pathways that promote aberrant cell proliferation and survival [5]. For example, experiments with a quinone-type PCB metabolite showed that it significantly enhanced breast cancer cell aggressiveness by stimulating aerobic glycolysis and upregulating matrix metalloproteinases (MMPs), consistent with a pro-tumorigenic metabolic shift [30]. Epigenetic data provide further support: in vitro exposure of human blood monocytes to common congeners such as PCB-

118 and PCB-153 induced global DNA hypomethylation, a hallmark of genomic instability closely associated with carcinogenesis [35]. Endocrine disruption is also a notable feature of PCB toxicity – certain PCB congeners can weakly mimic estrogens or antagonize androgens, while others interfere with thyroid hormone signaling, collectively resulting in hormonal imbalances that could promote hormone-responsive tumors [31]. Finally, PCBs modulate the immune system by activating the AhR, which alters the function of immune cells. Through AhR-mediated pathways, PCBs can suppress tumor-surveillance mechanisms while provoking chronic inflammation in tissues, both of which facilitate tumor initiation and progression [33]. In the liver, sustained PCB exposure may lead to steatohepatitis that progresses to cirrhosis and ultimately hepatocellular carcinoma. In the stomach, PCB-induced oxidative stress and immune dysregulation may contribute to gastric tumorigenesis, whereas in lymphoid tissues, chronic immunosuppression may increase susceptibility to lymphoma.

B. Reproductive and Developmental Toxicity

Accumulating clinical and epidemiological evidence demonstrates that even low-level exposures can impair fertility, elevate the risk of adverse pregnancy outcomes, and compromise neurodevelopmental trajectories in children. In men, exposure to PCBs has been associated with reduced semen quality and altered hormone profiles. A study of young men from Brescia, Italy, a region with a history of PCB contamination, reported that higher seminal PCB concentrations were strongly associated with reduced sperm motility and increased abnormal morphology. Men in the highest quartile exhibited a 19% reduction in motility and 23% fewer morphologically normal sperm relative to those in the lowest quartile [1]. Consistent results have been reported elsewhere; in a U.S. cohort, serum PCB-153 was inversely correlated with testosterone concentrations, suggesting interference with androgenic pathways [36]. Maternal exposure has also been shown to influence male developmental outcomes. A Danish birth cohort revealed that women living in PCB-contaminated housing had sons with a significantly higher risk of cryptorchidism (OR ~1.7), supporting the hypothesis that PCBs disrupt androgen signaling during fetal development [37].

Female reproductive function is similarly vulnerable. In women undergoing fertility evaluation, serum concentrations of PCB congeners 118, 138, 153, and 180 were associated with suppressed luteal-phase estradiol and gonadotropin levels, consistent with impaired ovarian function [36]. Strong associations have also been reported between PCB exposure and pregnancy loss. In Turkey, women who experienced spontaneous abortions had markedly higher blood levels of PCB-101, -52, and -138, with odds ratios ranging from 2 to over 16 compared to women with successful pregnancies [38]. Similarly, in an Italian IVF cohort, women from a highly polluted region had a live birth rate of only 33%, compared with 73% in women from a low-exposure region, despite equivalent fertilization and implantation outcomes [39]. Adverse effects extend to fetal growth. A large U.S. prospective cohort found that maternal PCB exposure in early pregnancy was significantly associated with reduced birth weight [40]. The ALSPAC cohort in the United Kingdom confirmed these findings, reporting lower infant birth weight among PCB-exposed mothers, particularly in lower socioeconomic groups [41]. Although most studies have not identified strong associations with preterm birth, consistent evidence of reduced birth weight indicates that intrauterine growth is a primary target of PCB toxicity.

Neurodevelopment is another critical area of concern. A longitudinal study of a Slovak cohort found that higher cord blood concentrations of dioxin-like PCBs predicted reduced IQ scores at age six, with decrements of 1.5–2 points across full-scale, verbal, and performance domains [42]. Continued exposure in childhood was also linked to further reductions in verbal ability and cognition, reinforcing the persistence of developmental impacts. Associations with behavioral outcomes, including ADHD, remain inconsistent: some cohorts report elevated symptom scores [43], whereas others report null associations [44]. Mechanistic studies provide further substantiation for these observations. In Israel, maternal PCB burden was associated with reduced neonatal thyroid hormone levels [45]. In contrast, a large Chinese cohort (>2,200 mother–infant pairs) found that maternal dioxin-like PCB exposure was significantly associated with reduced thyroid hormone levels in cord blood [46]. Complementary findings from Iran revealed that higher PCB levels in cord blood were associated with elevated pro-inflammatory cytokines (TNF-α, IL-6, IL-8) and oxidative stress biomarkers in neonates, indicating a combined endocrine and inflammatory mechanism [26]. Mechanistic studies further substantiate these observations. PCBs and their metabolites induce oxidative stress, disrupt gametogenesis, impair implantation, and compromise placental function. As endocrine disruptors, they antagonize androgen receptors, mimic estrogens, and interfere with thyroid hormone transport and metabolism [47]. Animal models also provide evidence for transgenerational effects: rats exposed in utero to Aroclor 1221 exhibited hormonal disturbances and increased body weight across F_2 and F_3 generations, even without further exposure, consistent with epigenetic inheritance of PCB-induced developmental programming [48].

C. Endocrine Disruption

PCBs are recognized as broad-spectrum endocrine disruptors, capable of interfering with thyroid, reproductive, and metabolic hormone systems. Recent human studies have provided compelling evidence that PCB exposure alters hormone homeostasis, often resulting in subtle but clinically relevant disturbances that can contribute to long-term disease risk. Altered thyroid function has been among the most consistently reported endocrine outcomes. In adults exposed to PCBs since childhood in Michigan, higher serum PCB burdens were associated with elevated free thyroxine

(FT_4) levels and an increased FT_3-to-FT_4 ratio, whereas thyroid-stimulating hormone (TSH) levels remained unchanged [7]. In a German pregnancy cohort, maternal PCB levels were linked to reduced maternal FT_3 and TSH and to altered thyroid hormone status in infants, indicating potential consequences for both maternal and neonatal thyroid physiology [49]. Mechanistically, hydroxylated PCB metabolites (OH-PCBs) bind to transthyretin, displacing endogenous thyroxine (T_4) and altering hormone transport and distribution. In parallel, dioxin-like congeners, such as PCB-126, function as potent AhR agonists, enhancing hepatic thyroid hormone metabolism and disrupting receptor-mediated signaling [50]. Together, these mechanisms align with epidemiological findings of hypothyroxinemia and altered thyroid hormone profiles in exposed populations.

Reproductive hormone regulation is also susceptible to PCB disruption. Longitudinal studies have demonstrated that peripubertal PCB exposure influences pubertal development in a congener-specific and sex-specific manner. For example, in Russian boys, higher concentrations of non-dioxin-like PCBs predicted earlier onset and accelerated progression of puberty, whereas dioxin-like toxic equivalents were associated with delayed pubertal development [50]. Among women, PCB exposure has been associated with altered menstrual cycles, longer time-to-pregnancy, and higher miscarriage risk, while daughters of highly exposed mothers have been reported to experience irregular cycles and earlier menopause. In Faroese men, elevated PCB body burdens were correlated with higher luteinizing hormone (LH) and sex hormone-binding globulin, as well as increased total testosterone; however, bioavailable free testosterone remained unchanged. This pattern is consistent with pituitary compensation and subclinical testicular stress, supporting an anti-androgenic mode of action [51]. In vitro studies provide mechanistic corroboration: many PCB congeners antagonize the androgen receptor, whereas others weakly activate estrogen receptors, producing a net anti-androgenic effect in men and mixed estrogenic/anti-estrogenic effects in women [52].

Metabolic disruption has also emerged as a major endocrine consequence of PCB exposure. A nested case–control study in China reported that individuals in the highest quartile of serum non-dioxin-like PCBs had up to a threefold higher risk of developing type 2 diabetes within five years, accompanied by elevations in fasting glucose and triglycerides [53]. Data from the U.S. NHANES further revealed that men with high PCB-153 levels exhibited reduced testosterone and more markers of metabolic syndrome [54]. Mechanistic studies indicate that PCBs accumulate in adipose tissue, where they promote chronic inflammation and impair insulin signaling, partly through increased cytokine release such as TNF-α and IL-6. AhR-dependent pathways are particularly implicated: PCB activation of AhR in adipocytes and hepatocytes induces expression of CYP enzymes and inflammatory mediators, while pharmacological AhR antagonism has been shown to block PCB-induced glucose intolerance in experimental models [55]. Mitochondrial dysfunction and oxidative stress further contribute to insulin resistance, while recent studies indicate that PCBs can induce DNA methylation changes in pancreatic β-cell genes, impairing insulin secretion [53].

D. Neurotoxicity and Neurodevelopmental Effects

PCBs remain a major concern for the nervous system, with increasing evidence that both developmental and adult exposures contribute to neurological dysfunction. Developmental neurotoxicity is of particular concern, as prenatal and early-life stages are especially sensitive to PCB-induced disruption. Epidemiological and experimental studies consistently demonstrate adverse effects on cognition, behavior, and neuroendocrine signaling, and emerging evidence also suggests a role for PCBs in neurodegenerative processes later in life [56], [57].

Mechanistic studies have clarified how PCBs interfere with neurodevelopment. Non-dioxin-like congeners, which constitute the majority of human PCB body burdens, disrupt calcium (Ca^{2+}) signaling by sensitizing ryanodine receptors (RyRs). Even at very low concentrations, congeners such as PCB-11, -37, and -95 can trigger abnormal Ca^{2+} release from endoplasmic reticulum stores, resulting in excessive dendritic growth and branching in developing neurons [58]. Recent work has shown that PCB-37, at sub-nanomolar levels, simultaneously promotes dendritic arborization and caspase-dependent apoptosis in a CREB-dependent manner, thereby linking structural overgrowth with neuronal vulnerability [59]. These findings suggest that RyR-mediated Ca^{2+} dysregulation is a central pathway by which PCBs impair neuronal connectivity. Beyond Ca^{2+} signaling, PCBs also alter dopaminergic neurotransmission. Experimental models reveal that exposure reduces tyrosine hydroxylase activity and dopamine levels, while zebrafish studies confirm disrupted dopaminergic and GABAergic signaling accompanied by sensorimotor deficits [60]. Oxidative stress and inflammation constitute additional converging mechanisms: elevated levels of reactive oxygen species and pro-inflammatory cytokines (e.g., TNF-α, IL-6) have been observed in PCB-exposed neonatal cord blood and in animal models [26]. Thyroid hormone disruption represents another well-established mechanism. PCBs and their hydroxylated metabolites can displace thyroxine (T_4) from transthyretin, enhance its clearance, and reduce circulating T_4. Because thyroid hormones are critical for brain development, even transient reductions during gestation can have lasting cognitive effects [61]. Taken together, these mechanistic insights explain how PCBs interfere with multiple neurodevelopmental pathways simultaneously.

Epidemiological studies provide strong support for these mechanisms. A comprehensive review of studies published since 2015 concluded that prenatal and perinatal PCB exposure is consistently associated with poorer cognitive performance in children, particularly reduced IQ, memory, and learning scores, as well as attention problems [62]. Some evidence suggests sex-specific vulnerability, with boys showing greater PCB-related declines in certain cohorts. Associations with

ADHD and autism spectrum disorder remain inconsistent: whereas Japanese cohorts report associations between higher prenatal PCB burdens and elevated autism-related symptoms at 18 months, other studies have found no significant relationships [44]. Notably, the strongest and most reproducible findings relate to deficits in cognition, attention, and behavior regulation, rather than categorical diagnoses of neurodevelopmental disorders.

There is also growing concern that PCB exposure contributes to neurodegeneration later in life. A population-based cohort in northern Italy, a region with high PCB contamination, found that individuals in the highest tertile of serum PCB concentrations had more than twice the incidence of dementia compared with those with lower exposures [63]. Longitudinal analyses confirmed a dose–response relationship, with higher PCB body burdens linked to accelerated cognitive decline [64]. Although associations between PCBs and Parkinson's disease remain less consistent, experimental data demonstrate that PCBs damage dopaminergic neurons, and human studies have detected higher PCB concentrations in the brains and serum of Parkinson's patients [65]. One imaging study of PCB-exposed workers even suggested possible sex-specific susceptibility, with greater striatal dopamine transporter loss in women.

E. Immunotoxicity

PCBs are established immunotoxicants that can both suppress protective immune responses and promote autoimmune phenomena. Epidemiological and experimental evidence consistently demonstrates that PCB exposure dysregulates immune function across the human lifespan, beginning as early as the prenatal period. One of the clearest demonstrations of PCB-induced immunosuppression is evident in studies of vaccine efficacy. In the Faroe Islands, children with higher prenatal PCB exposure displayed markedly lower antibody titers to diphtheria and tetanus following routine immunizations, with a subset failing to reach protective levels despite full vaccination [66]. In Slovakia, higher cord blood concentrations of PCB-153 were associated with reduced antibody responses to infant tuberculosis vaccination. In contrast, Inuit infants in Arctic Canada with elevated prenatal PCB burdens experienced increased rates of otitis media and lower respiratory infections during their first year of life [67]. Experimental studies provide mechanistic support: dioxin-like congeners such as PCB-126 activate AhR, leading to thymic and splenic atrophy, reduced T-cell cytokine production (e.g., TNF-α, IFN-γ, IL-2), and oxidative stress in immune tissues [68]. Non-dioxin-like PCBs show more complex dose–response effects. For example, low-dose exposure can suppress inflammatory cytokines, while high-dose exposure paradoxically enhances them, as demonstrated in a wound-healing mouse model. Moderate exercise attenuated some of these effects, suggesting that lifestyle factors may modulate PCB-induced immune disruption [69]. Overall, human and animal data converge on the conclusion that background PCB exposures are primarily immunosuppressive, resulting in diminished vaccine efficacy and increased infection risk. Importantly, reduced immune surveillance may also increase long-term cancer risk, particularly for malignancies such as non-Hodgkin lymphoma [2], [70].

Paradoxically, PCBs are also implicated in the promotion of autoimmunity. Epidemiological studies have found associations between serum PCB concentrations and the presence of autoantibodies. In the U.S. NHANES cohort, higher serum levels of dioxin-like congeners were associated with increased prevalence of antinuclear antibodies [71]. In Slovakia, residents of a PCB-contaminated district had significantly elevated anti-thyroid peroxidase and anti-thyroglobulin antibodies compared with those in a less contaminated area [72]. More recently, a Swedish case–control study reported that hydroxylated PCB metabolites (notably 4-OH-CB187 and 3-OH-CB153) were associated with an increased risk of multiple sclerosis, providing direct evidence that long-term PCB exposure may contribute to clinical autoimmune disease [73]. Mechanistic data lend credibility to these associations. Quinone metabolites of PCBs are highly reactive, capable of alkylating proteins and generating ROS. In mice, these metabolites induced lymphocyte apoptosis and skewed T-helper differentiation toward a Th1-dominant profile, a combination that promotes the release of autoantigens and pro-inflammatory signaling [74]. PCB quinones also induce neutrophil extracellular trap (NET) formation via ROS-dependent pathways, thereby exposing nuclear autoantigens to the immune system and lowering tolerance thresholds [75]. Furthermore, AhR activation by dioxin-like congeners promotes differentiation of Th17/Th1 subsets and impairs regulatory T-cell function, thereby enhancing autoimmune susceptibility [76].

Emerging evidence indicates that immune dysregulation may begin as early as the prenatal period. Some cohorts have reported smaller thymus sizes and reduced NK cell counts in infants with higher in utero PCB exposure, although the results are not entirely consistent. Importantly, reduced vaccine responses observed in early life have been shown to persist into school age, underscoring the long-lasting impact of prenatal exposure to PCBs [77]. The immunotoxic effects of PCBs are not limited to humans. Wildlife chronically exposed to PCBs, including fish, seals, and marine mammals, show altered cytokine expression, thymic atrophy, and heightened infection risk [78], [79]. These cross-species findings reinforce the conclusion that PCB-induced immune dysregulation is mechanistically conserved across vertebrates. Table 1 summarizes detailed results from representative human and animal studies.

TABLE I. SUMMARY OF HUMAN AND ANIMAL STUDIES DEMONSTRATING IMMUNOTOXIC EFFECTS OF PCBS.

Study / Population	Exposure Assessment	Observations	Mechanistic / Health Implications
Faroe Islands birth cohort (Heilmann et al., 2006)	Cord blood PCB-153 (tertiles, n≈119)	Top tertile: 24% of children below protective antibody threshold to diphtheria vs. 5%	Prenatal PCB exposure reduces vaccine-induced

Study / Population	Exposure Assessment	Observations	Mechanistic / Health Implications
		in lowest; significantly lower mean titers to diphtheria/tetanus	humoral immunity
Slovakia cohort (Protano et al., 2024)	Cord blood PCB-153	Reduced BCG antibody titers; effect persisted into school age	Long-lasting suppression of adaptive immunity
Inuit infants, Canada (Dewailly et al., 2000)	Maternal/cord PCB burden	1.5–1.9-fold higher risk of otitis media and respiratory infections in first year	Increased infection susceptibility due to impaired immunity
NHANES, U.S. adults (Dinse et al., 2016)	Serum dioxin-like PCBs	ANA prevalence higher (OR ≈ 1.8, top vs. bottom quartile)	Subclinical autoimmunity
Slovak residents (Langer, 2015)	Serum PCBs, polluted vs. control districts	Markedly higher anti-TPO and anti-Tg antibody prevalence in polluted area	Autoimmune thyroiditis linked to PCB burden
Swedish MS case–control (Vaivade et al., 2025)	Serum OH-PCBs (4-OH-CB187, 3-OH-CB153)	Increased multiple sclerosis risk (OR > 2)	Hydroxylated metabolites implicated in CNS autoimmunity
Animal, mice (Du et al., 2019)	PCB-126 exposure	Thymic/splenic atrophy; ↓ TNF-α, IFN-γ, IL-2	AhR-mediated suppression of T-cell immunity
Animal, mice (Pillai et al., 2020)	PCB exposure + exercise	Low-dose PCB suppressed cytokines, high-dose enhanced; exercise partially mitigated	Dose-dependent immune modulation, lifestyle influence
Animal, mice (Peng et al., 2022a,b)	PCB quinone metabolites	Lymphocyte apoptosis; Th1 skewing; excessive NET formation	ROS-driven apoptosis and NETosis expose autoantigens
Mechanistic, in vitro/in vivo (Eti et al., 2022)	AhR agonism by PCB-126	Expansion of Th17/Th1; impaired Tregs	Lowered tolerance threshold, autoimmune activation
Birth cohort (Merza et al., 2025)	Cord blood PCBs	↑ TNF-α, IL-6, IL-8; oxidative imbalance in neonates	Prenatal immune activation and oxidative stress
Infants (Kolijn & Langerak, 2023)	In utero PCB/dioxin exposure	Smaller thymus, reduced NK cell counts	Developmental immune impairment
Wildlife (marine mammals, rodents) (Walker et al., 2024; Sun et al., 2025)	Long-term environmental PCB contamination	Pro-inflammatory gene expression; thymic atrophy; ↑ NF-κB	Conservation of immunotoxic mechanisms across species

ABBREVIATIONS: ANA, ANTINUCLEAR ANTIBODY; AHR, ARYL HYDROCARBON RECEPTOR; BCG, BACILLE CALMETTE–GUÉRIN; IL, INTERLEUKIN; MS, MULTIPLE SCLEROSIS; NET, NEUTROPHIL EXTRACELLULAR TRAP; NK, NATURAL KILLER (CELL); PCB, POLYCHLORINATED BIPHENYL; ROS, REACTIVE OXYGEN SPECIES; TPO, THYROID PEROXIDASE; TG, THYROGLOBULIN; TNF-A, TUMOR NECROSIS FACTOR-ALPHA; TREG, REGULATORY T CELL

F. Metabolic and Cardiovascular Effects

PCBs disrupt glucose and lipid metabolism, contributing to the development of cardiovascular disease. Multiple human cohort studies have associated PCB body burdens with insulin resistance and the onset of type 2 diabetes. In a six-year prospective study, each unit increase in serum PCB-118 was associated with significant increases in fasting glucose and insulin, along with a 0.66 increase in HOMA-IR. Individuals in the highest exposure category had nearly a 75% increased risk of developing diabetes [80]. This effect was most pronounced in participants with high polygenic risk scores, underscoring the interaction between genetic susceptibility and environmental exposure. Similar associations were observed in a ten-year follow-up cohort in Iran, where higher total PCB concentrations predicted incident type 2 diabetes, with dioxin-like congeners conferring up to a 4.8-fold increased risk [9]. PCBs also alter lipid metabolism and contribute to the development of dyslipidemia. A population-based study of nearly 3,800 adults in China found significant positive associations between serum PCB concentrations and levels of total cholesterol, triglycerides, and LDL-cholesterol [81].

Mechanistic research has identified several pathways underlying these metabolic outcomes. PCBs accumulate in adipose tissue, where they trigger chronic low-grade inflammation and secretion of cytokines such as TNF-α and IL-6, both of which impair insulin signaling. Experiments with PCB-153 demonstrate NF-κB activation and reactive oxygen species generation, leading to altered hepatic and pancreatic gene expression and reduced glucose uptake and insulin secretion [82]. Dioxin-like congeners such as PCB-126 act through AhR pathways, activating endothelial and immune cells, increasing pro-inflammatory cytokines (IL-6, IL-1β), and upregulating adhesion molecules including ICAM-1 and VCAM-1. In rodent studies, chronic PCB-126 exposure exacerbated adiposity, worsened insulin resistance, increased circulating triglycerides and cholesterol, and caused oxidative damage in pancreatic islets [83]. Further evidence implicates mitochondrial dysfunction and epigenetic modifications: PCB exposure alters DNA methylation in pancreatic β-cell genes, impairing insulin secretion, and induces transcriptional changes via PXR and CAR in hepatocytes [53], [55].

The metabolic consequences of PCB exposure directly contribute to increased cardiovascular risk. In the Aragon Workers' Health Study, dietary PCB

exposure was significantly associated with higher coronary artery calcium scores, a marker of subclinical atherosclerosis [84]. In Sweden, elevated serum PCB levels predicted greater carotid plaque burden and cardiac remodeling, including increased left ventricular mass. Experimental evidence provides further support: mice exposed to PCB-126 for ten weeks developed significantly larger atherosclerotic lesions in the aortic root, demonstrating a direct causal role in atherosclerotic progression [83]. Hypertension has also been consistently linked to PCB exposure. In a northern Italian cohort living in a highly polluted area, participants in the highest PCB strata had approximately double the risk of incident hypertension compared to those with lower exposure, with the association strongest for highly chlorinated congeners such as PCB-138, -153, and -180 [63]. Recent meta-analyses corroborate these findings, confirming that both total PCB levels and dioxin-like congeners are significant predictors of hypertension risk [6]. These metabolic interactions between PCBs, inflammation, and cardiovascular outcomes are summarized in Fig. 3.

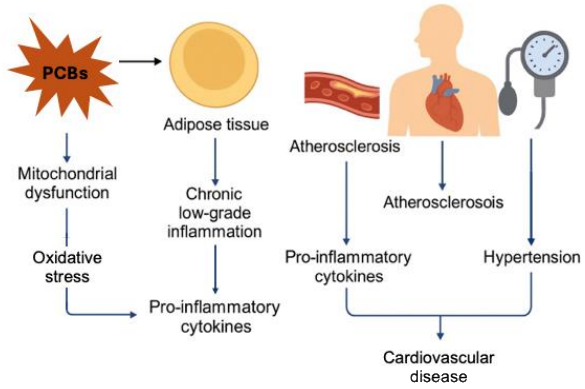

Fig. 3. Metabolic interactions between PCBs, adipose tissue, inflammation, and cardiovascular outcomes.

V. FUTURE PERSPECTIVES

Recent advances have substantially expanded our understanding of PCB toxicology, yet several critical knowledge gaps remain. A key priority is to clarify the toxicological relevance of PCB mixtures and their metabolites, since humans are rarely exposed to single congeners. Real-world exposures involve complex mixtures of dioxin-like and non-dioxin-like congeners, as well as hydroxylated and other metabolites, which may act additively, synergistically, or even antagonistically. Emerging studies demonstrate that hydroxylated PCBs accumulate in human brain tissue in an age- and region-specific manner, with higher levels observed in adult cortex than in younger samples [85]. Other studies indicate that these metabolites interfere with host–microbiome interactions, such as inhibition of intestinal α-glucosidase activity, thereby disrupting glucose metabolism through previously unrecognized pathways [86].

Another critical area for future research is the role of genetic and epigenetic modifiers of PCB toxicity. Epidemiological studies have documented wide inter-individual variability in disease outcomes, suggesting that host susceptibility plays a decisive role. Evidence from long-term follow-up of highly exposed worker cohorts indicates that even after serum PCB concentrations decline, biomarkers of liver dysfunction, hormonal imbalance, and metabolic disruption persist [13]. Genetic polymorphisms in receptors such as AhR and ESR1 are associated with differential risks of vascular and neurological diseases in exposed populations [87]. Epigenetic studies further reveal that PCB exposure induces long-lasting modifications in immune- and neuron-related genes, raising the possibility that prenatal exposures may 'program' disease susceptibility later in life [88], [89]. Longitudinal birth cohorts with repeated biospecimen collection will be essential to determine whether such epigenetic signatures can serve as predictive biomarkers [90], [91].

On the translational front, preventive and therapeutic strategies remain insufficiently explored. Nutritional interventions such as supplementation with omega-3 fatty acids, selenium, and antioxidants have shown promise in counteracting PCB-induced oxidative stress and supporting thyroid function [92]. Experimental studies suggest that non-absorbable fat substitutes such as Olestra accelerate PCB elimination by interrupting enterohepatic circulation [93]. Small clinical trials confirm increased fecal PCB excretion after Olestra intake, although broader validation is required. Lifestyle interventions such as weight loss may reduce long-term body burdens but can paradoxically mobilize PCBs from adipose stores, temporarily elevating serum levels [94]. Combining dietary strategies with pharmacological or binding agents to capture mobilized PCBs in the gut may therefore offer a safer and more effective means of detoxification.

Environmental remediation remains an urgent and unresolved challenge. Decades after their ban, PCBs continue to cycle between soil, sediments, water, and air. Novel approaches including bacterial bioremediation through reductive dechlorination, advanced chemical degradation technologies, and improved sediment management are being investigated [19]. Climate change introduces an additional layer of complexity: warming, ice melt, and permafrost thaw are expected to remobilize legacy PCBs stored in Arctic reservoirs, reintroducing them into marine and terrestrial food webs [95], [96]. Sustained biomonitoring of fisheries and staple foods will be essential to protect public health, especially in communities reliant on traditional diets [97].

Finally, ensuring equity and fostering international collaboration are essential. While PCB levels have declined in many high-income countries, they remain elevated or even rising in regions with delayed bans, inadequate waste management, or continued use of PCB-containing equipment [19]. Strengthening global monitoring capacity, transferring remediation technology, and safely disposing of remaining stockpiles are critical steps. Public education is equally critical: communities require clear guidance on minimizing exposure, including safe fish consumption

practices and proper handling of PCB-containing building materials [98], [99].

In summary, the future of PCB research and management lies in an interdisciplinary approach that combines molecular toxicology, genomics, epidemiology, and environmental engineering. By focusing on mixtures and metabolites, identifying genetic and epigenetic susceptibility factors, developing safe interventions, and addressing global disparities in exposure, the field can move toward protecting vulnerable populations and mitigating the long-term health burden of PCBs. Moreover, the lessons learned from PCBs offer a framework for addressing other persistent organic pollutants, reinforcing the need for proactive strategies to safeguard environmental and human health in an era of accelerating industrial and climate change.

VI. Conclusion

PCBs remain a significant global health concern decades after their production and use were banned. Their persistence in the environment, bioaccumulative potential, and long biological half-lives ensure continued human exposure through diet, inhalation, and maternal transfer. Epidemiological evidence from the past decade has strengthened associations between PCB exposure and a wide spectrum of health outcomes, including cancer, reproductive and developmental toxicity, endocrine disruption, neurodevelopmental impairments, immunotoxicity, and cardiometabolic disease. Importantly, these findings are no longer confined to highly exposed occupational cohorts or poisoning episodes but are increasingly observed at background exposure levels in the general population.

Mechanistic research has identified convergent pathways underlying this diversity of effects. Oxidative stress, endocrine receptor interference, disruption of calcium homeostasis, immune modulation, mitochondrial dysfunction, and epigenetic reprogramming all contribute to the complex toxicological profile of PCBs. Such mechanistic insights provide strong biological plausibility for epidemiological observations and explain why PCBs act as multisystem toxicants. The persistence of PCBs in soils, sediments, air, and biota underscores the necessity of continued biomonitoring and public health surveillance. Vulnerable populations such as pregnant women, infants, and individuals with high dietary or occupational exposures require particular attention. Global disparities in exposure and remediation capacity demand international collaboration to ensure that communities in lower-resource settings are not overlooked.

Reducing the health burden of PCBs will require coordinated strategies that integrate molecular toxicology, epidemiology, clinical medicine, and environmental engineering. Research into the effects of mixtures and metabolites, the identification of genetic and epigenetic susceptibility markers, and the development of interventions to lower body burdens will be critical. Equally important are sustained efforts in environmental remediation and effective risk communication to reduce ongoing exposure. Ultimately, insights from PCB toxicology extend beyond this single class of chemicals, providing both a cautionary example of how industrial compounds can persist for decades with widespread and long-lasting health consequences, and a framework for addressing other persistent organic pollutants. Continued vigilance, innovation, and international cooperation are crucial to safeguard human health and the environment from the legacy of PCBs and emerging persistent contaminants.

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