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Pet cats, the better sentinels for indoor organic pollutants

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Pets are the most intimate companions of humans, as pets and people share most of their lives indoors. Based on the connections between pet animals, humans, and the environment, pet cats and dogs are often recommended as sentinels for the detection of environmental contaminants and for comorbidity tracking. However, their suitability as sentinels is yet to be established. Persistent organic pollutants and environmental hormones have replaced particulate matter (PM2.5) and volatile organic compounds (VOCs) in causing indoor air pollution. This review summarises the differences in the types and concentrations of indoor organic environmental pollutants detected in pet dogs and cats. This includes an analysis of the main exposure routes of different types of pollutants. To identify which of the two pet species are better sentinels, cats and dogs were compared based on their metabolic capacities of various indoor organic pollutants. In addition to PM2.5, a range of organic compounds including polychlorinated biphenyls, polybrominated diphenyl ethers (PBDEs), bromophenols, perfluoroalkyl substances, organochlorine pesticides, fungicides, polycyclic aromatic hydrocarbons, phthalic acid esters, organophosphate pesticides, organophosphorus flame retardants, and melamine have been detected in both dogs and cats. Pets often accumulate PBDEs from dust; however, traces of PBDEs are present in their diet. The indoor pollutant contamination levels in internal cats were generally higher than those in dogs. Cats accumulate organic pollutants associated with indoor environments, but they are sensitive to their toxicity because, unlike dogs, cats cannot metabolise most of the accumulated pollutants. Moreover, cats share similar clinical symptoms of thyroid diseases in humans. Based on the above observations of detection of indoor organic pollutants, it could be said that cats are better sentinels than dogs.

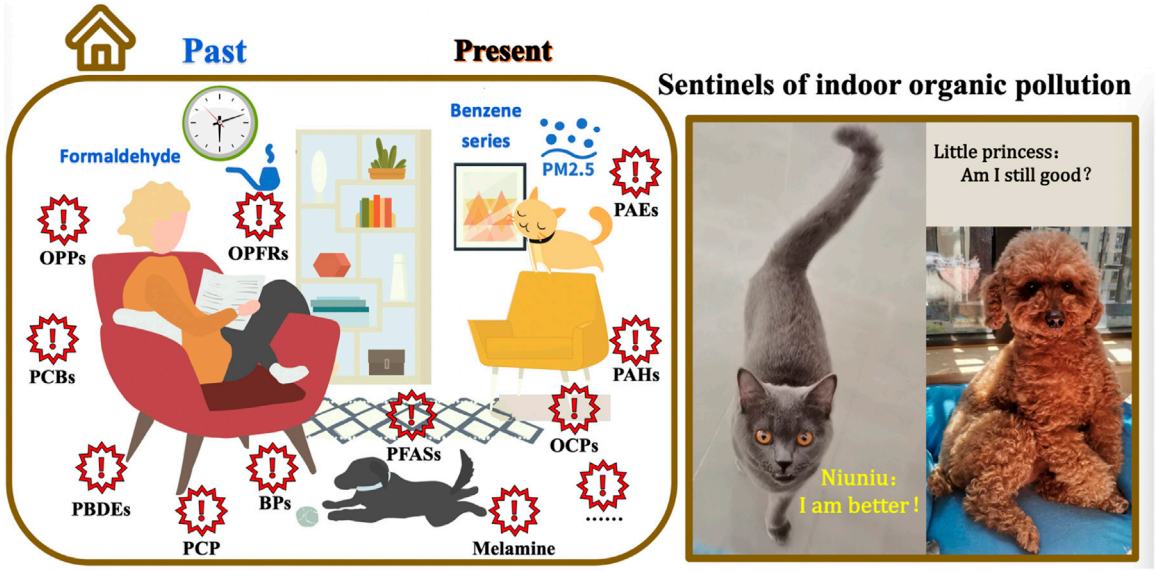
KEYWORDS

companion animal, serum, endocrine disrupting chemicals (EDCs), indoor, sentinel

1 Introduction

The indoor environment consists of air (gas and particles) and surfaces (walls, ceilings, and floors) (Bui et al., 2016). People spend almost 90% of their lifetime indoors in their homes, workplaces, and schools; therefore, the quality of the indoor environment has a profound influence on human health (Mercier et al., 2011). The persistent drive to improve living standards over the past decades have led to the recent focus on indoor air quality (Leung, 2015). Indoor air assessments include the detection of formaldehyde, benzene compounds, and particulate pollution of PM2.5 as pollutants. However, the influx of consumer products and building materials in recent decades has introduced several novel contaminants such as plasticisers (phthalic acid esters, PAEs), pesticides, polychlorinated biphenyls (PCBs), and polybrominated diphenyl ethers (PBDEs) in indoor environments (Mercier et al., 2011; Tran et al., 2020; Yue et al., 2020). Human exposure to elevated levels of toxic volatile organic compounds (VOCs), semi-VOCs, and fine particulate matter (PM2.5) may induce asthma, allergies, cardiovascular diseases, neurological diseases, cancers, foetal malformations, and abortions (Li et al., 2008; Kotzias, 2021). Annually, over five million people die prematurely due to indoor air pollution caused by building material emissions and activities such as cooking and smoking (McAuley et al., 2012; Tran et al., 2020; González-Martín et al., 2021).

The key attribute of sentinel species is that they react to environmental hazards before humans do. Pets fulfil most of the criteria for sentinel species and have served as valuable sentinels for human exposure to various indoor contaminants since the turn of this century (EWG, 2008; Venier and Hites, 2011; Guo et al., 2012; Ali et al., 2013). Although they live in the same environment as humans, pets have a shorter life span and tend to catch illnesses easily as they are more sensitive to pollutant exposure (EWG, 2008). For example, endocrine toxins are of particular concern for cats, especially PBDEs, plastics, and bisphenol A (BPA)-related thyroid toxins, because thyroid disease is the main cause of pain and death worldwide in cats (EWG, 2008). However, dogs and cats frequently consume food contaminated with chemicals associated with processing and packaging, resulting in cumulative health risks. However, a number of studies have detected heightened levels of organic contaminants in indoor air and household dust, suggesting that exposure to processed and packaged food may not be the main reason for indoor air pollution (Venier and Hites, 2011). Pets share indoor environments with humans, and spend most of their lives indoors that they mirror human exposure and are affected by the toxins similar to their human owners (Ali et al., 2013; Braouezec et al., 2016; Poma et al., 2020). Worldwide occurrence of bladder cancer in dogs is similar to that of the same disease in humans; similarities observed in non-ovariectomised pet dogs have provided insights into the relationship between the environment and breast cancer in humans (Hayes et al., 1981; Sévère et al., 2015). Although pets live in man-made environments, there is little comparison between cats and dogs in terms of the sources of their exposure to these pollutants, or their metabolic capacities to deal with those pollutants (Ruiz-Suárez et al., 2015a). The unique roles played by cats and dogs in our lives may make them exceptional sentinels for humans in terms of exposure to indoor pollutants and the resultant health problems that they



GRAPHICAL ABSTRACT

experience (Dye et al., 2007). The present study compares the contamination levels of different indoor organic pollutants in pets. It discusses the rationality of using cats and dogs as sentinels and attempts to determine the most suitable sentinels for different types of indoor pollutants.

2 Pollutants of concern

The leading sources of indoor chemical contamination include the degradation products of building materials or volatile compounds of coating components (Mercier et al., 2011). Other sources include products for home cleaning, cosmetics, biocides, textiles, house furnishings, electronic devices, and other household products; indoor activities such as cooking, smoking, and burning incense and candles; the intrusion of materials from outdoors. Exposure routes to indoor pets may include consumption of contaminated drinking water and canned/commercial food; inhalation of contaminated air, and licking of dust (EWG, 2008; Mensching et al., 2012; Aeluro and Kavanagh, 2021). Documented organic pollutants in pet dogs include bromophenols (BPs), dioxin-like PCBs (DL-PCBs), non-dioxin-like PCBs (NDL-PCBs), organochlorine and organophosphate pesticides (OCPs and OPPs), organophosphate esters (OPEs), polycyclic aromatic hydrocarbons (PAHs), PBDEs, perfluoroalkyl substances (PFASs), melamine and derivatives and novel brominated flame retardant (NBFRs). In Spain, these have been found in plasma and hair samples (Ruiz-Suárez et al., 2015b; González-Gómez et al., 2018). Cats have been investigated as possible sentinels for exposure to various chemicals that are potentially harmful to humans, including flame retardants (bromine, phosphate, etc.,) (Poutasse et al., 2019; Aeluro and Kavanagh, 2021), perfluoroalkyl substances (Bost et al., 2016), and chlorinated pollutants (Ruiz-Suárez et al., 2015a).

2.1 Polybrominated diphenyl ethers

PBDEs are known for their ubiquitous use as flame retardants; they pose health risks by disrupting endocrine function. PBDEs have been in use since the 1970s in many household products including electronic goods, synthetic textiles, polyurethane foam, or thermoplastics (Birnbaum and Staskal, 2004; Shaw and Kannan, 2009). As described by Hites (2004) and Dye et al. (2007), commercial materials incorporate “penta”, a composite that includes tetrabrominated BDE-47, pentabrominated BDE-99 and -100, and hexabrominated BDE-153 and -154; “octa”, which mainly contains septa- and octabrominated, as well as hexa- and nonabrominated congeners; “deca”, which includes the maximally brominated congener 209 and includes minimal nonabrominated congeners (Hites, 2004; Dye et al., 2007).

In Pakistan, levels of PBDEs in cat serum were found to be significantly higher than those in human serum (Ali et al., 2013). The Centers for Disease Control and Prevention (CDC) and the Environmental Working Group (EWG) conducted studies that found a 23-fold increase in the accumulation of PBDEs in cats than that found in people (EWG, 2008). The levels of PBDE in California house cats were approximately 50-fold higher than those found in local residents (EWG, 2008; Guo et al., 2012). In Georgia, Massachusetts, and North Carolina during 2005 and 2006, ΣPBDE levels in cat serum were 20–100 times higher than the median levels in U.S. adults. More domestic cats than feral cats die from feline hyperthyroidism (FH), as excessive PBDE serum burdens have been found in domestic cats compared with wild cats; PBDE has been correlated FH development (Poutasse et al., 2019).

In Sweden, decabromobiphenyl (BB-209, discontinued in 2000) is present across all cat serum samples at levels comparable to those of decabromodiphenyl ether (BDE-209) (Norrgran et al., 2015); BDE-209 is the major congener in cat serum (Nomiyama et al., 2022). In South Africa, BDE-209 is the dominant brominated flame retardant (BFR) in pet cat hair, followed by bis(2-ethylhexyl)-3,4,5,6-tetrabromo-phthalate (BEH-TEBP) and 2-ethylhexyl-2,3,4,5-tetrabromobenzoate (EH-TBB) (Brits et al., 2019). In the case of the recently banned BDE-47, the characteristics of contamination were very similar between humans and cats, although the concentrations were higher in cats (Henríquez-Hernández et al., 2017). Dog serum samples were dominated by tetra-to hepta-BDE congeners, as well as decabromodiphenylethane and hexabromocyclododecane (Venier and Hites, 2011). Hyperthyroid cats have higher serum concentrations of BDE-99, BDE-153, and BDE-183 than euthyroid cats (Norrgran et al., 2015). However, a study conducted using 20 obese and 20 normal house dogs showed that although BDE-47 and BDE-99 were the predominant congeners in both groups, the serum levels of total BDEs and thyroxine and thyroid-stimulating hormone levels could not be correlated (Srebočan et al., 2019). PBDEs in cat serum were present in the following order of concentration: BDE-99 > 47 > 153, yet in the sera of dogs and humans, they were BDE-47 > 99 > 153 and BDE-47 > 153 > 99, respectively (Ali et al., 2013).

Hydroxylated metabolites of PBDEs (HO-PBDEs), formed through oxidation involving cytochrome P450 enzymes have structural similarities with thyroxine; residual concentrations of HO-PBDEs have been reported in human blood; their toxicity levels vary based on the location of the hydroxyl group (Qiu et al., 2009; Lin et al., 2019). Exposure to technical penta- and octa-BDEs resulted in high concentrations of endocrine-disrupting chemicals such as 2,4,6-tribromophenol and 6-OH-BDE47, which are the most common species in cat serum, indicating that hydroxylation, dehalogenation, and ether bond rupture are the main metabolic pathways in serum (Norrgran et al., 2012). In 17 Swedish homes, MeO-BDE68 was the only methoxylated

PBDE detected in cat serum, whereas HO-PBDEs were not detected in cats or dogs, suggesting that the formation of BPs as metabolites may also be due to diphenyl ether bond cleavage of PBDEs in cats and dogs (Mizukawa et al., 2017). The observation of significantly higher levels of ∑BPs than ∑HO-PBDEs in the serum of both cats and dogs suggests that there are other sources of contamination than diphenyl ether bond cleavage, which is believed to be a substantial metabolic pathway for PBDEs and BP formation in humans (Ali et al., 2013). After incubation in PBDE mixtures (BDE-47, BDE-99, and BDE-209), the detection of 2,4,5-TBPh metabolised from BDE-99 in the microsomes of dog liver, similar to that in humans, showed hydroxylation at the 1′ carbon atom of the ether bond in dogs (Mizukawa et al., 2017). The 100% detection rate of BB-209 but low or absent hydroxylated PBDE metabolites in cat serum suggests the difficulty in metabolising BB-209 (Norrgran et al., 2015). Decabromodiphenylethane (DBDPE) was present at high levels in dust and food samples but was not detected in serum samples, which indicates poor bioavailability of DBDPE in cats (Norrgran et al., 2017). However, there was no correlation between HT cats and ΣPBDE levels, suggesting age- or disease-dependent changes in the metabolism of PBDE (Dye et al., 2007).

Pet cats are exposed to PBDEs primarily through their diet (PBDEs are present in food and liquids) and the indoor environment, mostly dust particles (Peterson, 2012); there is a correlation between cat serum and household dust in terms of PBDE congener concentrations and patterns (EWG, 2008; Guo et al., 2012; Norrgran et al., 2017). The correlation between dust PBDE and serum total thyroxine (T4) concentration also suggests that ingestion of household dust is the most likely primary route of exposure (Dye et al., 2007), as well as a significant exposure route for people, particularly young children (Harrad et al., 2010; Mercier et al., 2011; Dirtu et al., 2012).

2.2 Polychlorinated biphenyls

PCBs, banned in the 1970s and 1980s were ubiquitous, carcinogenic, teratogenic, and toxic compounds; PCBs were used as coolants, insulating fluids in transformers and capacitors, stabilising additives in polyvinyl chloride (PVC), and as plasticisers in sealants; they are also present in many other applications (Audy et al., 2018; Sari et al., 2020). Some PCBs metabolites are more polar than their parent compounds; therefore, they are easily retained in the tissues and organs of both humans and wildlife, resulting in greater toxicity effects (Hovander et al., 2002).

"Outdoor" cat groups showed a lower PCB burden than "indoor" cat groups (up to 1,446 ng/g in serum) (Serpe et al., 2018). In Atlanta, Georgia, PCB levels in the serum of healthy dogs were ~5 ng/g (Schilling et al., 1988). ∑PCBs concentrations in Japanese human serum were ~3.7 times and ~7.5 times higher than those in cats (160 pg/g) and dogs (79 pg/g) (Kunisue and Tanabe, 2009). In southern Italy, PCB concentrations in adipose tissues were higher in dogs than in cats. The concentrations of all PCB congeners in human serum samples were only correlated with age; PCB-153, PCB-138, and PCB-180 were the top three predominant congeners (Park et al., 2007). The profile of PCB-138, PCB-153, and PCB-180 in pet animals of Taranto City were similar to that in humans, where significantly heightened concentrations of dioxin-like PCBs were detected in pets compared to the other locations (Storelli et al., 2009). In cat (*Felis silvestris catus*) sera from Paris, the concentrations of ∑19PCBs were 2,799 ± 944 ng/L, while those in cat food and indoor air was 1.7 ng/g and 1.5 ng/m^3, respectively (Braouezec et al., 2016). The intake volumes of PCBs due to dust ingestion and consumption of commercial dry pet foods were at the level of ng/kg; however, considering the third pathway of inhalation exposure, the model-based calculated indoor exposure of ∑6PCBs for Italian cats was higher than the reference dose (Serpe et al., 2018). Cats suffering from acromegaly induced diabetes mellitus, whose PCB profiles were similar to those of humans, showed a significantly higher PCB concentrations in the plasma compared to other cats; this indicated that endocrinopathy inhibited their capacity to metabolise persistent organic pollutants (Dirtu et al., 2013).

Cytochrome P450 monooxygenases catalyse PCBs into hydroxylated polychlorinated biphenyls (OH-PCBs), which interfere with thyroid hormone homeostasis (Kowalik and Sechman, 2022). ∑OH-PCBs concentrations in dogs were 3- and 10-fold higher than those in cats and humans, respectively, indicating that animals, especially dogs, might be at risk from OH-PCBs (Kunisue and Tanabe, 2009); the high OH-PCBs/PCBs ratios in dogs (~16–~300-fold) suggest a greater metabolic capacity for PCBs (Kunisue and Tanabe, 2009; Ali et al., 2013). Significant negative correlations were detected between the concentrations of PCBs, OH-PCBs, and thyroid hormone (THs) (Nomiyama et al., 2022). Decreased levels of total T4 and 3,3′,5-triiodo-L-thyronine (T3) in the serum of PCB-exposed dogs were negatively correlated with PCB concentrations; this indicates that PCB exposure enhanced TH excretion by increasing TH uptake and TH conjugation enzyme activities in dog liver, but not in that of cats, because of species-specific PCB and TH metabolism (Takaguchi et al., 2019).

2.3 Bromophenols (exogenous source)

BPs are not only metabolites of PBDEs but also natural products of marine origin and man-made chemicals. As important phenolic contaminants, BPs are widely distributed in water bodies, indoor air, sediments, waterborne organisms, and human tissues, exhibiting significant adverse effects in humans, especially in terms of the disruption of thyroid

hormone homeostasis and sex hormone steroidogenesis (Wang et al., 2020). 2,4,6-tribromophenol (2,4,6-TBP), as a reactive FR intermediate or as a wood preservative, can disrupt the function of neuroendocrine cells and the endocrine system as a whole, and has a negative impact on dehaloperoxidase-haemoglobin (Zhao and Franzen, 2013). The analysis of serum samples from Pakistan showed that 2,4-dibromophenol (2,4-DBP), 2,4,5-tribromophenol (2,4,5-TBP), and 2,4,6-TBP were the most frequently detected BPs in cats, higher than those in dogs, while 2,3,4,5,6-pentabromophenol (2,3,4,5,6-PBP) was the major BP in dog serum (Ali et al., 2013). In samples from Japanese veterinarian hospitals, BP concentrations were higher in cat blood than in dog blood, while similar BP profiles between pet blood samples and commercial pet food suggested that diet might be a substantive route of exposure to pet BPs (Mizukawa et al., 2017). The detected median value of 2,4,6-TBP in the serum of cats is about 25-fold higher than that in cat foods, which further suggests that food intake could be an important pathway for internal accumulation of BPs (Norrgran et al., 2017).

2.4 Pthalic acid esters

PAEs have been broadly used in the manufacturing of plastics since the 1930s, including cosmetics, paints, pesticides, chemical fertilisers, adhesives, and plasticisers, and they are often detected in air, freshwater, sediment, and soil (Net et al., 2015). PAEs are a major public health concern because they are endocrine disrupters, carcinogens, teratogens, and mutagens. Exposure to PAEs increase the risk of fertility damage, cancer, and long-term diseases such as heart disease and diabetes (Ma et al., 2019). In the serum of cats living in Paris, France, PAEs had the highest level of organic pollutants compared with PCBs and PBDEs, at 107 ± 98 μg/L for ∑9PAEs, which was higher than that in human serum (11,600–18500 ng/L) (Inoue et al., 2005; Braouezec et al., 2016). In the case of cat food and indoor air of the same locations, concentrations of ∑9PAEs were 2,292 ng/g and 848 ng/m^3, respectively (Braouezec et al., 2016). In Italy, diethylhexyl phthalate (DEHP) found in cat food samples ranged from 2,500 μg/kg to 8,700 μg/kg (Ezõerskis et al., 2007)—much higher than that found in samples from Paris. PAEs in the human body are often hydrolysed and rapidly metabolised into their primary monoester metabolites (mPAEs) before being excreted as urine (Elliani et al., 2020; Cheng et al., 2021). However, no relative information regarding mPAEs in the urine of cats or dogs has been reported. Based on the data of PAE concentrations in different resources, pet food and indoor air are considered the main, but not the only two pathways of PAE exposure for pet cats and dogs.

2.5 Perfluoroalkyl substances

PFASs are versatile substances often included in consumer products, including water- and oil-proofing agents, inks, varnishes, waxes, lubricants, leather, paper, textiles, and fluoropolymers. When released from these products into the indoor environment, humans are exposed to PFASs (Shoeib et al., 2011). Perfluorooctane sulfonate (PFOS) and its salts were included in the list of persistent organic pollutants (POPs) under the Stockholm Convention of 2009; in 2019, perfluorooctanoate (PFOA), its salts, and related compounds were also added to the list (Meng et al., 2021). PFAS prevalence and geometric means in the serum samples of 72 cats were very similar to those in human sera in the U. S. population; thus, domestic cats were recommended as effective sentinels for understanding primary PFAS exposure routes, especially indoor sources relevant to children (Bost et al., 2016). The CDC and EWG also found 2.4-fold higher levels of PFAS in dogs compared to average levels in humans (EWG, 2008). In paired household dust and cat serum samples collected from Sweden, significant correlations between PFOA and perfluoroundecanoic acid (PFUnDA) were observed, while PFOS and PFOA were the two most abundant PFAS (Weiss et al., 2021). In the U.S., the highest PFAS concentration in the serum of indoor cats was that of perfluorohexane sulfonate (PFHxS); total PFAS levels were found in pet subjects affected by indoor life and higher body weight (Bost et al., 2016). The disturbed upregulation of thyroid hormone (TH) or thyroxine (T4) concentration has been used to deduce FH, and the significant positive correlation between serum total T4 and PFNA in pet cats suggested clinical consequences in cats (Weiss et al., 2021).

2.6 Organophosphorous flame retardants

OPFRs are chemicals produced at high volumes (>1,000 tons/year produced in Europe) that are incorporated into many household and industrial materials and finished products such as room furniture, buildings, automotive industries, and textile industries (Cristale et al., 2017). OPFRs are ubiquitous in South African indoor environments and are the major FRs contributing to more than 97% of the total FR concentration (Brits et al., 2019). Cats are exposed to bioavailable household FRs; elevated tris(1,3-dichloro-2-isopropyl) phosphate (TDCIPP) exposure is associated with FH. Cats can wear silicone pet tags to evaluate interventions to prevent health risks and act as sentinels for FR and endocrine-disrupting chemicals (Poutasse et al., 2019). Similar detection frequency, detection pattern, and concentration levels in both species from Spain showed that pet cats are effective sentinels of

human exposure to OPFRs (Henríquez-Hernández et al., 2017).

2.7 Pesticides (fungicide and insecticide)

Pentachlorophenol (PCP) is the main metabolite of hexachlorobenzene (HCB), which has been widely used as a fungicide and wood preservative (Lv et al., 2020). The concentrations of PCP, the most important phenolic compound detected in dogs, were significantly lower than those in cat serum in Pakistan (Ali et al., 2013). In Italy, insecticides and pesticides are the main cause of indoor poisoning (72.3%); such poisonings in an urban environment affected 55% of dogs and 69% of cats (Bertero et al., 2020). Lower levels of OCPs were detected in dog serum than in human serum in samples from Pakistan, whereas for OCPs and their metabolites, the highest concentration levels of serum in cats were ~22 times higher than those in dogs (2,435 ng/g lw in cats and 110 ng/g lw in dogs) (Ali et al., 2013). In the fat tissues of cats and dogs from southern Italy, p,p′-DDE was the most common DDT component (95.0%), whereas in dogs, these compounds were only present in two specimens (Storelli et al., 2009). In Japanese pets, the levels of organochlorines in the genitals of dogs were lower than those in the genitals of cats; however, the residue levels in the diets of cats and dogs were similar, implying that the accumulation and elimination mechanisms of these contaminants differ between these two pet types (Kunisue et al., 2005). In hair samples from cats and dogs, the order of importance of organohalogenated contaminants (OHCs) was ∑OCPs > ∑novel BFRs > ∑PBDEs > ∑PCB, whereas in paired hair-serum samples for cats (N = 12), ∑DDTs were significantly correlated (Lin et al., 2019). Median OCP levels in eastern Romania were 1,300 ng/g in indoor dust, indicating that the consumption of indoor dust is also a substantial route of exposure to OCPs in specific regions, although food intake is believed to be a substantial exposure route to OHCs in pets, in particular PCBs and OCPs (Dirtu et al., 2012).

2.8 Polycyclic aromatic hydrocarbons

For over 40 years, PAHs have been ubiquitous POPs, carcinogens, and teratogens released as by-products due to the incomplete combustion or pyrolysis of organic matter and fossil fuels (Albinet et al., 2007). PAHs in food come from environmental deposits or from food processing including smoking, drying, roasting, and barbecuing (Duedahl-Olesen, 2013). With the exception of benzo(a)pyrene, phenanthrene, fluorene, fluoranthene, and 2-naphthol were detected in almost all samples of dog blood, but their levels were highly variable and different from those found in human blood, which were 332.7 and 307.5, 68.1 and 33.7, 35.1 and 70.1, 86.6, and 59.4 ng/g lw (median values), respectively (Ruiz-Suárez et al., 2015b).

2.9 Melamine

The most important organic nitrogen compounds used as FR additives are melamine and derivatives including melamine oxalate, melamine phosphate, melamine phthalate, and melamine cyanurate (Thirumal et al., 2010). Melamine and its derivative concentrations (i.e., ammeline, ammelide, and cyanuric acid) in the urine samples indicated that they were not significantly different between cats and dogs in the U.S. (205 ± 196 ng/ml for cats and 173 ± 215 ng/ml for dogs), and the calculated cumulative daily intake was 10-5-fold below the tolerable daily intake; however, age and sex were key predictors of the levels of the target chemicals in all pets (Karthikraj et al., 2018).

2.10 PM2.5 (the carrier and combination of various organic pollutants)

"Toxicity of PM 2.5" in early research has been put forward (Wang et al., 2011) because PM2.5 consists of diverse chemicals including but not limited to PAHs, OCPs, and PCBs. Therefore, the chemical composition of PM2.5 determines its health effects and cytotoxicity (Kim et al., 2019). A hypercellular response in the lower airways of dogs was associated with poor indoor air quality, including unacceptable levels of PM2.5 (>35 μg/m^3) or increased levels of VOC (>1 ppm) in the most frequently visited areas of the home by dogs (Lin et al., 2020). Based on the investigation of the National Taiwan University Veterinary Hospital, dogs with respiratory disease were those exposed to incense burning than were control dogs, but the household level of PM2.5 did not significantly differ between dogs with and without respiratory disease. For cats, unacceptably high levels of household PM2.5 (>35 μg/m^3) were significantly correlated with respiratory disease (Lin et al., 2018). Cotinine was not detected in dog urine (Yorkshire terriers) that was not exposed to cigarette smoke, whereas exposed dogs tested positive for the compound. Dogs exposed to smoke showed elevated levels of macrophage and lymphocyte concentrations; moreover, anthracosis was observed in the macrophage cytoplasm (Roza and Viegas, 2007).

3 Discussion

Non-human animals are considered sentinels in environmental monitoring and tracking of infectious diseases; this is based on knowledge of the connections between animals, humans, and the environment, through a paradigm of "One Health shared risk" (Rabinowitz et al., 2008; Dórea et al., 2011).

TABLE 1 Indoor organic pollutants in cat serum/blood.

Sample	Contaminant	Component	Test group	Concentration (average value, a and/or median value, M)	References
Serum	PBDEs	∑PBDEs	Young cats	1.85–10.3 ng/ml (A 4.3, M 3.5)	Dye et al. (2007)
Serum	PBDEs	∑PBDEs	Old non-hyperthyroid cats	2.23–27.2 ng/ml (A 10.5, M 5.9)	
Serum	PBDEs	∑PBDEs	Old hyperthyroid cats	3.0–39.5 ng/ml (A 12.7, M 6.2)	
Blood	PCBs	∑PCBs		160 pg/g (A)	Kunisue and Tanabe, (2009)
Blood	OH-PCBs	∑OH-PCBs		840 pg/g (A)	
Blood	PCP	PCP		2,200 pg/g (A)	
Serum	PBDEs	∑PBDEs		631–22537 ng/g lw (A 4505, M 2904)	Guo et al. (2012)
Serum	PCBs	∑PCBs		15.1–566 ng/g lw (A 203, M 170)	
Serum	OCPs	∑OCPs		0–10534 ng/g lw (A 1016.7, M 391)	
Serum	PBDEs	∑15PBDEs	Euthyroid cats	470–16000 ng/g lw (M 2850)	Mensching et al. (2012)
Serum	PBDEs	∑15PBDEs	Hyperthyroid cats	370–51000 ng/g lw (M 2517)	
Serum	PBDEs	∑PBDEs		1–1,280 ng/g (A 72, M 6.1)	Ali et al. (2013)
Serum	BPs	∑BPs		20–335 ng/g lw (A 125, M 110)	
Serum	NBFRs	BTBPE		<1–9 ng/g lw (A 1.6, M < 1)	
Serum	PCBs	∑OH-PCBs		<0.5–21.5 ng/g lw (A 5.3, M 2.2)	
Serum	OCPs and metabolites	p,p′-DDE		1–2,150 ng/g lw (A 282, M 95)	
Serum	OCPs and metabolites	∑DDTs		<1–2,175 ng/g lw (A 297, M 111)	
Serum	OCPs and metabolites	∑OCPs		46–2,435 ng/g lw (A 475, M 345)	
Serum	PFASs	PFOS		LOQ–121 ng/ml serum (A 8.89, M 9.05)	Bost et al. (2016)
Serum	PFASs	PFHxS		LOQ–235 ng/ml serum (A 6.91, M < LOQ)	
Serum	PFASs	∑PFASs		LOQ–412.99 ng/ml serum (A 21.97, M 14.7)	
Serum	PBDEs	∑9BDEs		38.4–81.3 ng/L (A 56.1)	Braouezec et al. (2016)
Serum	PCBs	∑19PCBs		1,406–4,279 ng/L (A 2797)	
Serum	PAEs	∑9PAEs		81,000–451000 ng/L (A 107,000)	
Serum	PAEs	Di-n-butyl phthalate		79,900 ng/L (A)	
Serum	PAEs	Di-iso-butyl phthalate		53,200 ng/L (A)	
Serum	PAEs	Di-iso-nonyl phthalate		43,800 ng/L (A)	
Serum	PAEs	Di-ethylhexyl phthalate		32,830 ng/L (A)	
Blood	PBDEs	∑PBDEs		<4.2–490 pg/g (M 180)	Mizukawa et al. (2016)
Blood	BPs	∑BPs		91–1,300 pg/g ww (M 260)	Mizukawa et al. (2017)
Serum	PCBs	∑PCBs		20.08 ± 78.31 ng/g fat (M 0.35)	Henríquez-Hernández et al. (2017)
Serum	BDEs	∑BDEs		5.48 ± 4.35 ng/g fat (M 4.45)	
Serum	OPFRs	∑OPFRs		1,049.88 ± 558.93 ng/g fat (M 909.25)	
Serum	PBDEs	BDE-209		32 pmol/g lw (M)	Norrgran et al. (2017)
Serum	PBDEs	BB-209		24 pmol/g lw (M)	
Serum	PCBs	CB-153		28 pmol/g lw (M)	
Serum	OCPs and metabolites	4,4′-DDT		30 pmol/g lw (M)	
Serum	OCPs and metabolites	4,4′-DDE		42 pmol/g lw (M)	
Serum	BPs	2,4,6-TBP		73 pmol/g lw (M)	
Serum	MeO-PBDEs	2′-MeO-BDE68		58 pmol/g lw (M)	
Serum	MeO-PBDEs	6-MeO-BDE47		42 pg/g–1206 pg/g serum (M 125)	
Serum	Chlorinated phenol	Chlorinated phenol		68 pmol/g lw (M)	
Serum	PCBs	∑PCBs		32.4–1,446 ng/g lw (A 264, M 175)	Serpe et al. (2018)
Serum	PFASs	PFOS		680–5,000 pg/ml (A 2300, M 2300)	Weiss et al. (2021)
Serum	PFASs	PFDA		130–1,500 pg/ml (A 500, M 430)	
Serum	PFASs	PFOA		<LOQ-15000 pg/ml (A 1800, M 1100)	
Serum	PFASs	∑PFASs		1.6–2.3 ng/ml (A 7.6, M 6.4)	

ww, wet weight; lw, lipid weight; PBDEs, poly brominated diphenyl ethers; PCP, pentachlorophenol; MeO-PBDEs, methoxyl polybrominated diphenyl ethers; BDE-209, decabromodiphenyl ether; BB-209, decabromobiphenyl; CB-153, 2,2'4,4',5,5'-hexachlorobiphenyl; 2′-MeO-BDE68, 2'-methoxy-2, 3',4,5'-tetrabromodiphenyl ether; 6-MeO-BDE47, 6-Methoxy-tetrabromodiphenyl ether; PCBs, polychlorinated biphenyls; HO-PCBs, hydroxylated polychlorinated biphenyls; OCPs, organochlorine pesticides; p,p′-DDE (4,4′-DDE), dichlorodiphenyldichloroethylene; DDT, dichlorodiphenyltrichloroethane; 4,4′-DDT (p,p'-DDT), 2,2-bis(4-Chlorophenyl)-1,1,1-trichloroethane; BPs, bromophenols; 2,4,6-TBP, 2,4,6-tribromophenol; NBFRs, novel brominated flame retardant; BTBPE, 1,2-bis(2,4,6-tribromophenoxy)ethane; PFASs, Perfluoroalkyl substances; PFOS, perfluorooctane sulfonate; PFHxS, perfluorohexane sulfonate; PAEs, phthalic acid esters; BDEs, brominated diphenyl ethers; OPFRs, organophosphorous flame retardants; PFDA, perfluorinated decanoic acid; PFOA, perfluorooctanoate.

Diseases such as hyperthyroidism, malignant lymphoma, chronic renal failure, and cancer, when at higher incidences in pets, are associated with lifestyle and the presence of various chemicals in the pet diet (Ali et al., 2013). Clearly, exposure to dust, dermal contact, or inhalation could be substantial routes of exposure for cats because they use activity areas in the house (rug, floor, sofas, etc.,) and engage in behaviours (grooming on and licking various materials) similar to toddlers (Dirtu et al., 2012). Evidence-based medicine indicates that not only are domestic cats the only species besides people frequently diagnosed with hyperthyroidism—known as toxic nodular goitre (TNG) in humans—but cats also share the clinical symptoms; hyperthyroid cats serve as animal models for TNG (Poutasse et al., 2019). However, in the case of dogs, owing to species-level differences, and differences in body size, lifestyle patterns including the amount of outdoor time (frequency of dog walking), consumption of varying amounts of commercial dry food and canned food, varying senses of smell, and differences in metabolic capacity, the levels of the same indoor organic pollutants detected in their bodies are unlikely to be similar to those of pet cats.

Variations of contaminant levels in serum, whole blood, hair, and urine between cats and dogs living in the same areas suggest differences in the sources of exposure and/or in the uptake and metabolic efficiencies of pollutants (Tables 1, 2; Supplementary Table S1). As shown in Tables 1, 2, the median concentrations of ∑PBDEs, ∑PCBs, ∑PFASs, ∑BPs, and ∑OCPs in the serum of cats were higher than those of dogs by approximately 1760-fold (Guo et al., 2012; Ali et al., 2013), ~58-fold (Schilling et al., 1988; Serpe et al., 2018), three-fold (Sévère et al., 2015; Bost et al., 2016), seven-fold (Ali et al., 2013; Mizukawa et al., 2017) and ~17-fold (Guo et al., 2012; Ali et al., 2013), respectively. These are stark differences between pet cats and dogs from different areas of the world, and the general accumulation patterns are ∑PBDEs > ∑PCBs > ∑OCPs > ∑BPs > ∑PFASs. However, in adjacent areas or regions, the median concentrations of ∑PBDEs, the average concentrations of ∑PCBs and ∑OH-PCBs in the blood samples, and the median concentrations of ∑OCPs and ∑OH-PCBs in the serum of cats were 1.8 times higher (Mizukawa et al., 2016), twice higher (Kunisue and Tanabe, 2009), 2.74 times lower (Kunisue and Tanabe, 2009), 15 times higher (Ali et al., 2013) and 2.73 times (Ali et al., 2013) lower compared with dogs. Except for the significantly higher accumulation of ∑OCPs in cats than in others, the similar multiple relationships of ∑OH-PCBs between cats and dogs verified the greater metabolic capacity of PCBs in dogs. In Supplementary Table S1, seen from other samples from pets, the average concentrations of ∑PCBs in adipose tissue, the median concentrations of ∑PBDEs and ∑OCPs in hairs of cats are about 4.8 (Storelli et al., 2009), 3.6 (Ali et al., 2013) and 1.5 (Ali et al., 2013) times higher compared with that in dogs in the same study areas. The net accumulation of ∑PBDEs was still higher than that of ∑OCPs, similar to the general trend observed in serum samples from different regions. In Supplementary Table S2, contaminants from different sources are listed. The median concentrations of ∑PBDEs in cat food and dog food range from 7.1 pg/g (wet food) (Mizukawa et al., 2016) to 1800 pg/g wet weight (ww) (Dye et al., 2007), and 4.2 pg/g (Mizukawa et al., 2016) to 1,080 pg/g ww (Venier and Hites, 2011), respectively. The median concentrations of ∑BPs in cat and dog food ranged from 110 to 270 pg/g ww and 12–20 pg/g ww (Mizukawa et al., 2017), respectively. Higher levels of contamination from cat food were observed. Moreover, in dust, one of the main sources, the median levels of ∑PBDEs, ∑PCBs and ∑PFASs were 782–17177 ng/g dry weight (dw), 97.4 ng/g dry weight (dw), and 280 ng/g dry weight (dw) separately. The exposure levels of the different pollutants varied significantly in the order ∑PBDEs > ∑PFASs >∑PCBs.

Confronted with high levels of PBDEs from different sources, especially for cats eating more contaminated food than dogs, the high contamination level of PBDEs is understandable. Similar BDE-47/BDE-99 ratios in cats and in-house dust samples indicate that house cats could be a good model for assessing the exposure of people to chemicals in indoor dust (Dirtu et al., 2013). However, the oxidative metabolism of BDE-47 and BDE-99 is different in the microsomes of the liver of cats and humans, suggesting that cats are not suitable sentinels for internal exposure of humans to PBDEs, but cats are possibly a suitable sentinel for internal hexabromocyclododecane exposure in humans (Zheng et al., 2016). In addition, the fact that lower concentrations in dust and higher concentrations in pet bodies indicated that the accumulation of PCBs in pets is much higher than that of PFASs. It has been suggested that dogs are not suitable sentinels for human exposure to PAHs, PCBs, and organochlorine pesticides (Ruiz-Suárez et al., 2015a), which is supported by evidence of pollutant metabolism. The bioconcentration factors (BCFs) of dichlorodiphenyltrichloroethane and their metabolites (DDTs), hexachlorocyclohexane isomers, hexachlorobenzene, and chlordane compounds in dogs were substantially lower than those in cats, indicating that dogs may have a higher capacity to metabolise and eliminate these contaminants (Kunisue et al., 2005). 2,4,5-TBPh caused by incubation in PBDE mixtures is only found in dog liver microsomes, which are similar to human liver microsomes, implying species-specific metabolic capacities for PBDEs (Mizukawa et al., 2017). Hepatic sequestration of polychlorinated dibenzo-p-dioxins (PCDDs), dibenzofurans (PCDFs), and oxychlordane has been observed in dogs (Kunisue et al., 2005). Phenobarbital-induced isozymes and CYP2B and 2C subfamily enzymes are key to the metabolism of PCBs (especially CB 153) by forming an uncommon PCB 2,3-arene oxide intermediate (Duignan et al., 1987; Ariyoshi et al., 1992). However, cats barely metabolise PCB 153, and it is possible that cats may have different CYP2B and 2C (cytochrome P450 2 B and 2C) subfamily enzymes than dogs (Kunisue et al., 2005). The fact that dogs can metabolise many types of indoor organic pollutants, such as PBDEs, DDTs,

TABLE 2 Indoor organic pollutants in dog serum/blood.

Sample	Contaminant	Component	Test group	Concentration range (A and/or M)	References
Serum	PCBs	∑PCBs	Monroe County dogs	0.9–5.1 ng/g (M 3.0)	Schilling et al. (1988)
Serum	PCBs	∑PCBs	Comparison dogs	0.66–3.2 ng/g (M 1.7)	
Blood	PCBs	∑PCBs		79 pg/g (A)	Kunisue and Tanabe, (2009)
Blood	OH-PCBs	∑OH-PCBs		2,300 pg/g (A)	
Blood	PCP	PCP		150 pg/g (A)	
Serum	PBDEs	∑PBDEs		1.37–2.15 ng/g ww (A 1.76, M 0.86)	Venier and Hites, (2011)
Serum	PBDEs	∑PBDEs		0.6–4 ng/g lw (A 1.9, M 1.65)	Ali et al. (2013)
Serum	BPs	∑BPs		<5–150 ng/g lw (A 25, M 16)	
Serum	NBFRs	BTBPE		<1–3.7 ng/g lw (A 0.9, M < 1)	
Serum	PCBs	∑OH-PCBs		2.2–16.8 ng/g lw (A 7.8, M 6.0)	
Serum	OCPs and metabolites	∑OCPs		16–110 ng/g lw (A 32, M 22.5)	
Serum	PFAS	∑PFASs		0.5–30.8 ng/ml lw (M 4.91)	Sévère et al. (2015)
Blood	PBDEs	∑PBDEs		<4.2–300 pg/g (M 100)	Mizukawa et al. (2016)
Blood	BPs	∑BPs		2.7–2,700 pg/g ww (M 93)	Mizukawa et al. (2017)
Serum	PBDEs	∑PBDEs	Obese dogs	0.0059–0.1275 ng/g serum (A 0.0190, M 0.0084)	Srebočan et al. (2019)
Serum	PBDEs	∑PBDEs	Normal house dogs	0.0059–0.0400 ng/g serum (A 0.0112, M 0.0062)	

hexachlorocyclohexane isomers, hexachlorobenzene, and chlordane compounds, makes them less sensitive as indicators. It has been recommended that sentinels be defined as animals sharing a common environment that can be used to measure the exposure levels of contaminants when it is unethical or inconvenient to take human samples. Even if humans do not feed on pet food, contaminants in processed food are too serious to be ignored. The risk of indoor organic pollutants to pets is the basis for human health risk assessment. If the residual pollutants in pets can be easily metabolised, the detection of these biological materials will make it more complicated than the direct determination of indoor air and dust, which would result in a meaningless sentinel selection. Based on the differences in metabolic capacity and accumulation characteristics between the two pets, uncertainty due to body size, food intake, exercise habits, and other factors had not impaired that monitoring results of cats could help gauge the exposure risks for infants. Therefore, based on the findings in this review, it could be said that cats outperform dogs in the comparative screening of sentinels. It is important to have sufficient data on the interspecies comparison of metabolic capacity for environmental pollutions of cats and dogs, in the future.

Both whole blood and serum were used in blood-based testing for indoor organic pollutant contamination. For example, in one study, ∑PBDEs in the serum of cats and dogs were 1–1,280 ng/g and 0.6–4 ng/g lw (Ali et al., 2013), while for whole blood testing they were <4.2–490 pg/g (<0.0042–0.49 ng/g) and <4.2–300 pg/g (<0.0042–0.3 ng/g) (Mizukawa et al., 2016). Additionally, for BPs, they were 20–335 ng/g lw and <5–150 ng/g lw in the serum of cats and dogs (Ali et al., 2013), and 91–1,300 pg/g (0.091–1.3 ng/g) ww and 2.7–2,700 pg/g (0.0027–2.7 ng/g) in the whole blood of cats and dogs (Mizukawa et al., 2017). There was an order of magnitude difference in the data obtained from the two test substrates. Serum refers to transparent liquid precipitated on the surface of blood clots after plasma coagulation. Because of the higher specific gravity of whole blood than that of serum—due to the red blood cells, white blood cells, platelets, and various coagulation factors contained in whole blood which are absent in the serum—the results are bound to be affected. Because erythrocytes contain approximately 30%–40% solids, when 100 volumes of erythrocytes are diluted with 100 volumes of distilled water, this results in a far greater dilution than a two-fold dilution of the fluid fraction of the corpuscles (Collip, 1920). To ensure that the degree of dilution was much higher, and because of the interference of many blood cells and other substances in whole blood, it is possible that the measured value would be lower than the values in the serum samples and also lower than the actual data. This explains the reason for using serum samples in the majority of blood tests.

The concentrations of contaminants detected in dog hair were higher in samples of Spain than those of Pakistan, and also higher in cat hair than in dog hair, which agrees with the results of serum samples (Supplementary Table S1). Hair has been selected as a non-invasive matrix because hair samples provide sample stability and information on compound exposure, and their considerable fat content enables the analysis of a wide range of pollutants (González-Gómez et al.,

2018). Meanwhile, non-invasive sampling not only minimises damage to living species or populations but also has more ethical acceptance and practical advantages, such as cost-effectiveness and easy applicability to both human and animal samples (Behrooz et al., 2020). In previous studies, POPs, heavy metals, trace elements, and other contaminants in the hairs of mammals, including wild cats, sheep, polar bears, hedgehogs, and crows, have been effectively determined (Jaspers et al., 2010; Arıkan et al., 2018; Behrooz et al., 2020; Drysdale et al., 2021; Gevao et al., 2022), suggesting that non-invasive tests are suitable for monitoring both fat-soluble and protein-binding pollutants. Hair is directly exposed to the environment and continuously accumulates contaminants from the indoor environment (Brits et al., 2019). The strong correlation of alkyl-OPFRs between hair and air in humans also demonstrates the reliability of analysing contaminants in hairs in the evaluation of indoor air quality (Kucharska et al., 2015). It is important to keep notice of the location of the sampling, for example, whether they were cut from the back, left and right sides, or under the pet's abdomen, because more contact with the ground or air could bring about error in results. Although the advantages of non-invasiveness for hair investigation are important, the accuracy of blood tests limits the popularisation of hair tests. Strong correlations between the levels of PFASs in blood and urine demonstrate the reliability of urine tests as a non-invasive method of analysis to detect indoor organic pollutants (Wang et al., 2018). Metabolites of melamine and PAEs are excreted mainly in urine; therefore, it is feasible to determine the concentration of contaminants in urine samples. Based on previous investigations, the concentrations of organochlorine insecticides, PCBs, and metals in the urine of cattle, sheep, and humans could be monitored (Jurjanz et al., 2008; Trejo-Acevedo et al., 2009; Kronberg, 2010), indicating that the application of urine tests as a non-invasive approach should be promoted in pets. Because faeces are the main route of excretion of POPs accumulated in the large intestine, the faeces of pets can also be introduced to assess the presence of indoor organic pollutants (Lee et al., 2018).

In all the above-cited studies, except for the ∑PBDEs in dry cat food (Dye et al., 2007), PFOS in cat serum (Bost et al., 2016), 1,2-bis(2,4,6-tribromophenoxy)ethane (BTBPE) in the serum of cats and dogs, BTBPE and DBDPE in the hair of cats and dogs (Ali et al., 2013), and the average concentrations of the target pollutants were all higher than the median values. Moreover, except for the research by Storelli et al. (2009), Braouezec et al. (2016), and González-Gómez et al. (2018), all studies provided the median values of the target pollutants. In the present study, severe data asymmetry, large data differences, non-normal distributions, and serious bias meant that the mean value could not reflect the characteristics of the data; instead, the median was used. Therefore, future studies should pay more attention to the significance of the median values, rather than automatically providing the mean values of datasets.

It has already been established that consuming contaminated food contributes to more than 90% of the total exposure to these compounds, including PAHs, PCBs, and organochlorine pesticides, and foods of animal origin are one of the main contributors (Ruiz-Suárez et al., 2015b). For example, ∑9BDEs in the serum samples of cats in Paris were almost 100-times higher than in cat food and indoor air, yet the intake of these compounds was approximately 100-times higher through food ingestion than by inhalation of indoor air (Braouezec et al., 2016). For PCBs and OCPs, dietary intake is considered a major exposure route compared with indoor dust ingestion (Dirtu et al., 2012). In addition to dietary intake, inhalation of contaminated air and dust are the main routes for pets to indoor exposure to organic contaminants (EWG, 2008; Mensching et al., 2012; Peterson, 2012; Aeluro and Kavanagh, 2021). Dust is a relevant exposure pathway for the ingestion of some PFASs in cats because their grooming behaviour has resulted in a high dust intake (Weiss et al., 2021). In contrast to previous studies, it has been shown that for MeO-PBDEs, PBDEs, and PBDE congeners, ingestion of household dust is the primary exposure route rather than diet, in the U.S., Sweden and elsewhere (Dye et al., 2007; EWG, 2008; Guo et al., 2012; Norrgran et al., 2017). It can be inferred that owing to the differences between pollutant types, the exposure routes directly affect the levels of contamination in pet bodies. The easiest route to pets for MeO-PBDE, PBDE, and PBDE congeners is dust, while food intake is the main route for PAHs, PCBs, and organochlorine pesticides. As shown in Supplementary Table S2, PBDEs in dry food are higher than those in canned/wet food, according to Dye et al. (2007) and Mizukawa et al. (2016), but the opposite is true for BPs (Mizukawa et al., 2017). Cats consuming canned food have a greater risk of hyperthyroidism than cats that feed on foods stored in other containers (Edinboro et al., 2004). The risks associated with daily intake of commercial food (dry, wet, and canned food) should not be ignored.

4 Conclusion

Based on the aggregated results presented in this review, the most commonly detected indoor organic pollutants in pet cats and dogs include OHCs such as PCBs, BFRs, BPs, PFASs, OCPs, PAEs, OPPs, OPFRs, PAHs, melamine, and special solid PM2.5, which are suspended in the air for a long time. Based on the differences in lifestyle and body functions between cats and dogs, and, above all, variance in the ability to metabolise pollutants, the overall contamination levels in the serum, whole blood, hair, and urine in cats were higher than those in dogs. Although the exposure routes of all target pollutants were mainly food, PBDEs were mainly found in household dust. Dogs have the ability to metabolise most accumulated indoor organic pollutants; therefore, they are not sensitive enough and are not suitable sentinels for humans. Cats share some clinical

symptoms with humans, and the levels of pollutants they accumulate are well correlated with those in the indoor environment. We conclude that, in addition to being a qualified companion, cats are good sentinels for indoor organic pollutants.

Author contributions

Conceptualization, TM and ZD; Formal analysis, TM; Funding acquisition, TM; Investigation, PW; Methodology, TM and TW; Project administration, YL; Resources, ZD; Software, PW; Validation, TM; Visualization, YL; Writing–original draft, TM; Writing–review and editing, YL. All authors have approved the submitted version of the manuscript.

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Conflict of interest

PW was employed by Jiangsu Rainfine Environmental Science and Technology Co., Ltd.

The remaining authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Publisher's note

Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fenvs.2022.1023818/full#supplementary-material

SUPPLEMENTARY TABLE S1
Indoor organic pollutant in other samples of cats and dogs.

SUPPLEMENTARY TABLE S2
Indoor organic pollutant in food and dust.

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