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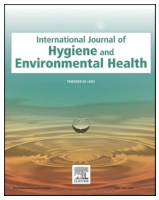

Inhalation and dermal absorption as dominant pathways of PCB exposure for residents of contaminated apartment buildings

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ABSTRACT

Applications of polychlorinated biphenyls (PCBs) in buildings and their persistence in indoor environments have led to cases of current and highly elevated exposure in humans, despite the global cease of production decades ago. Personal exposure to PCBs was assessed among residents in a social housing estate in Denmark containing both contaminated (n = 67) and non-contaminated (n = 23) apartments. Samples and estimated daily intakes (EDIs) were assessed for 15 PCB congeners, and body burden, which was limited by the dietary data availability, was compared across 7 indicator PCBs, with its sum (PCB_{sum7}) often applied in European regulation of PCBs. Median PCB_{sum7} EDI across measured pathways for exposed residents was 101 ng· (kg bodyweight)$^{-1}$· day^{-1}, with the majority of exposure (60%) coming from inhalation of contaminated indoor air. Calculated from both PCBs measured in indoor air and on hand wipes, dermal absorption estimates showed comparable results and served as a secondary exposure pathway, accounting for 35% of personal exposure and considering selected assumptions and sources of physical-chemical parameters. Estimates revealed that diet was the primary PCB source among the reference group, accounting for over 75% of the PCB_{sum7} EDI across exposure routes. When evaluating overall EDIs across the two study groups and including dietary estimates, PCB exposure among exposed residents was around 10 times higher than the reference group. Solely within the exposed population, pathway-specific body burdens were calculated to account for exposure across years of residence in contaminated apartments, where lower chlorinated PCBs were dominant in indoor air. Among these dominant congeners, estimated body burdens of PCB-28 and -52 were significantly correlated with measured serum ($r_s = 0.49, 0.45$; $p < 0.001$). This study demonstrates that inhalation and dermal absorption serve as dominant exposure pathways for residents of apartments contaminated with predominantly lower chlorinated PCBs and suggest that predictions of body burden from indoor environment measurements may be comparable to measured serum PCBs.

1. Introduction

Polychlorinated biphenyls (PCBs) were used as flame retardants, plasticizers, and dielectric fluid, among other applications, with production starting in the 1930s (IARC, 2016). Consisting of 209 distinct congeners of varying numbers and positions of chlorine atoms, PCBs were manufactured in mixtures, the most common being the Aroclor and Clophen series, until production largely ceased in the 1980s. They were particularly lauded for their high chemical stability, which inadvertently led to their environmental persistence, also in indoor environments (Audy et al., 2018). When the Stockholm Convention on Persistent Organic Pollutants (POPs) went into effect in 2004, PCBs were listed as one of the initial "Dirty Dozen". They are classified as carcinogenic to humans (Group 1 in IARC framework) and are considered to be endocrine disruptors, neurotoxic, and immunotoxic with effects on cardiovascular and reproductive health (ATSDR, 2000; Heilmann et al., 2010; IARC, 2016).

Within the Stockholm Convention treaty, all use of PCBs is required to be phased out by 2025, with total elimination of PCBs by 2028 (UNEP, 2017). However, historic applications, such as elastic sealants and fluorescent lighting ballasts, are still present in buildings currently in use, which contribute to ongoing contamination of indoor environments and to continued human exposure to PCBs. With these continuing exposures in mind, the Danish Health Authority established two

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recommended action values in 2009 for PCBs in indoor air: an air concentration ≥300 ng PCB_{total}/m^3 is considered a possible health risk and an action plan is needed, and ≥3000 ng PCB_{total}/m^3 requires immediate action (Jensen, 2013). PCB_{total}, as defined by the Danish Health Authority, is the sum of seven indicator congeners (PCB-28, -52, -101, -118, -138, -153, and -180) measured in air multiplied by a corrections factor of 5, based on practice and action levels first established in Germany (Jensen, 2013). Notably, current Danish recommended action values only consider the inhalation pathway of PCB exposure for individuals living in contaminated homes. These recommendations resulted in increased awareness and screening of residential buildings, particularly among social housing organizations and municipalities, throughout Denmark.

In one such social housing estate, Brøndby Strand Parkerne, several high-rise apartment buildings were built using PCB-containing sealants. The indoor environment in some apartment buildings within the estate has been examined thoroughly, through dust, air, and surface wipe samples, and residents' exposure has been characterized using samples from their apartments in addition to hand wipes and serum samples (Andersen et al., 2020; Frederiksen et al., 2020). Both environmental and personal samples have indicated that residents' exposure was high and exceeded Danish Health Authority limits, but relative contributions of exposure pathways to internal dose measurements have not yet been determined. The relationship between PCB exposure to internal dose is supported by the wealth of studies conducted among individuals living or working in environments with high indoor air PCB concentrations, which correlated with similarly elevated serum levels and particularly for lower chlorinated PCBs (Ampleman et al., 2015; Herrick et al., 2011; Kraft et al., 2018, 2021; Liebl et al., 2004; Meyer et al., 2013). Given the measured air concentrations and prevalence of lower chlorinated PCBs measured previously in these apartments, dermal absorption is likely to also be an important pathway for PCB exposure, although few studies have evaluated its contribution to personal exposure. While dermal absorption has long been considered important, the difficulty in its measurement has led to body burden contributions to be largely underestimated for many semi-volatile organic compounds, including PCBs (Weschler and Nazaroff, 2012). For those living and working in non-contaminated buildings, diet and particularly fish consumption are likely to be the primary sources of personal exposure to PCBs (Fromberg et al., 2011).

Herein, we examined which pathway of exposure contributed most to personal exposure to PCBs for residents of contaminated buildings compared to those living in non-contaminated apartments. Samples had been previously collected and analyzed for 15 PCB congeners. Dietary exposure was estimated based on available data for specific congeners (8 out of the 15). Sum of the seven indicator PCBs, PCB_{sum7}, was used for comparisons of relative exposure contributions and body burden measurements.

2. Methods

2.1. Population and recruitment

Located about 15 km southwest of Copenhagen, Denmark, Brøndby Strand Parkerne is a housing estate containing 12 fifteen-story apartment buildings, with 4–5 apartments per floor, and several shorter apartment buildings. Erected between 1969 and 1974, only the first 5 high-rise buildings were constructed with PCB-containing sealants, which were placed around light façade elements indoors and on the enclosed balconies and windows outdoors (Andersen et al., 2020). Based on analysis of sealant materials, congener patterns indicate that they likely contained Clophen A-40 or Aroclor 1248, both of which are dominated by lower (mostly tetra-) chlorinated PCBs (Andersen et al., 2020; Takasuga et al., 2006). The remaining seven high-rises as well as surrounding smaller apartment buildings were constructed without use of PCB-containing sealants. Additional details about the buildings and PCB content have been published previously (Andersen et al., 2020; Frederiksen et al., 2020).

Study participants were recruited from Brøndby Strand Parkerne, with the exposed group formed by residents in the first 5 high-rise apartments and the reference group residing in other apartment buildings on the premises. Additional recruitment details were described previously in Frederiksen et al. (2020). All participants gave informed consent prior to providing any personal information and were informed of their individual results, upon request. Study protocols and related materials were approved by the Regional Ethics Committee (H-16041946) and reported to the Data Protection Agency through University of Copenhagen (SUND-2017-03).

2.2. Sample collection and analysis

Samples from participants' home environment (e.g., air, house dust, surface wipes) and personal samples (e.g., hand wipes, serum) were collected between October and December 2017. Further details regarding sample collections and results were reported for the home environmental samples in Andersen et al. (2020) and for personal samples in Frederiksen et al. (2020). In brief, active air samples were collected from each apartment's living room over a 24-h period, dust samples from participants' vacuum cleaners, and hand wipes by wiping both palms with an isopropyl alcohol wipe. All samples were stored at −20 °C following collection until extraction and analysis, except the dust, which was stored in a refrigerator until handling (i.e., sieving to <75 μm, then analyzed). All samples were analyzed for the 7 indicator PCBs and further PCB-8, -11, -18, -31, -44, -66, -74, -99 and -105, for 15 PCB congeners, total. This set of 15 PCBs, listed in Table S1 with relevant physicochemical properties, were compared across matrices. Dietary information was available for 8 of these congeners. As such, a subset of 7 indicator PCBs and their sum (ΣPCB-28, 52, 101, 118, 138, 153, 180), or PCB_{sum7}, for which all information was available, was used for comparisons of estimated daily intake (EDI) considering possible pathways of exposure.

2.3. Calculations

Potential pathways relevant to human exposure to PCBs include inhalation, dermal absorption, diet, dust ingestion, and hand-to-mouth contact. Given the physical-chemical properties of the PCBs, dust ingestion and hand-to-mouth contacts are anticipated to play comparatively minor roles but are still evaluated here (**SI Section S.3**). For each pathway of exposure, a pathway-specific EDI was calculated using concentrations measured in the corresponding environmental or personal sample. All EDIs [$ng \cdot (kg\ bodyweight)^{-1} \cdot day^{-1}$] were normalized to participants' body masses, which were self-reported.

2.3.1. Treatment of questionnaire data

Several parameters for EDI calculations were taken from questionnaires administered to all study participants. These data included an array of questions regarding their home environment, behavior, potential past exposures, height, weight, and dietary habits. To determine personal exposure with more specificity to individual behavior, cofactors such as exposure duration were calculated from questionnaire responses, or imputed if missing, based on estimations from the Danish Health Authority or US Environmental Protection Agency Exposure Factors Handbook. Exposure duration was calculated as the fraction of time spent per day in the home, which was determined from questionnaire data. If missing (n = 18), exposure duration was replaced with an estimate of activity factors based on age group (18-<65 years: 15.8 h/day; ≥65 years old: 19.6 h/day) (USEPA, 2011c). If body weight was missing (n = 1), this was imputed to 70 kg, as recommended by the Danish Environmental Protection Agency guidance document (Höglund et al., 2012).

2.3.2. Inhalation pathway

Exposure via inhalation was estimated from indoor air concentration (C_{air}) measured in participants' homes (ng/m^3). Inhalation rate was the recommended mean long-term exposure value for inhalation of combined males and females, based on age (range: 12.9–16 m^3/day) (USEPA, 2011a). The absorption fraction for PCBs from air in the lungs was assumed to be 100%, and measured air concentrations in residences were assumed constant and consistent within the various rooms of a single apartment.

$$EDI_{inhalation} = \frac{C_{air} \times inhalation\ rate \times exposure\ duration \times absorbance\ fraction}{body\ weight} \quad (1)$$

2.3.3. Dermal absorption

Exposure to PCBs through dermal absorption was estimated in two ways – from indoor air concentrations and from a hand wipe, which was taken from both palms. Hand wipes potentially capture dermal uptake via air and surface contact, whereas estimations from air concentrations likely reflect transdermal transport from chemicals in the gas phase in indoor air. Dermal uptake can also vary based on behaviors such as handwashing, which is likely to remove some particles and PCBs on the skin surface; however, this is not considered here, and participants were asked to not wash their hands for at least 30 min prior to study visits.

In calculating an EDI, air and hand wipe concentrations were used to determine a gas-phase concentration of individual PCBs and a concentration in surface skin lipids, respectively, and then calculate the transdermal flux, J, of transport from the boundary layer (air) or skin surface lipids (hand wipe) to the dermal capillaries at steady state (Weschler and Nazaroff, 2012, 2014). J was applied to total body surface area, which was estimated by age and sex of the participant and ranged from 1.69 to 2.15 m^2 (USEPA, 2011b). For calculating dermal exposure, measurements of PCB concentrations in air and on wiped hands are assumed to represent daily (24-h) exposure, where people are exposed on a continual basis via a personal cloud effect for air (e.g., clothing contribution) or via a constant skin surface PCB concentration. As such, the exposure duration is assumed to be 24 h/day.

$$EDI_{dermal} = \frac{J \times total\ body\ surface\ area \times exposure\ duration}{body\ weight} \quad (2)$$

Additional details for calculating the transdermal flux, J, for each method are included in Supplementary Information (**SI Section S1 and S2**).

2.3.4. Diet

Diet is considered a major pathway of exposure for PCBs because of their persistence in the environment and accumulation in food chains. Estimates of dietary intake of PCBs from food was only available for 8 of the 15 PCB congeners measured in the samples (the 7 indicator PCBs and PCB-105) for the adult Danish population. These were estimated from multiple food sources for individuals in Denmark, ages 15–75 years, with food samples taken between 1998 and 2003 (Fromberg et al., 2011). PCB concentrations in food were used to calculate an intake per age group using a typical Danish diet from that time period. PCB exposure from food in Denmark is expected to have decreased slightly since then, whether from changes in PCB levels in food and/or fish consumption, and whole diet adult exposure estimates for summed PCBs were roughly 3 times lower when assessed in 2004–2011 (Duedahl-Olesen et al., 2020); however, Fromberg et al. (2011) was the most recent report of overall dietary exposure to individual PCBs in the Danish population. We assume here that dietary consumption of PCBs in food for the participants during this study period is similar to this report, and thus, the dietary estimates calculated here could be slightly overestimated. Gastrointestinal (GI) uptake is assumed to be 90%, which was estimated from studies assessing dietary absorption of PCBs (Andreas Moser and McLachlan, 2001; Aylward et al., 2014; Ritter et al., 2011).

$$EDI_{diet} = \frac{Dietary\ intake \times GI\ uptake\ fraction_{diet}}{body\ weight} \quad (3)$$

2.3.5. Pathway-specific body burden

For exposed residents, internal dose of PCBs was assessed using EDIs from each exposure pathway to predict body burden concentrations for individual congeners. The body burden concentrations (C_{PCB}(t) [ng/g lipid]) were calculated to reflect presumed body burden (i.e., presence of PCB in the body) from a specific exposure pathway at time of sampling, while considering the number of years participants were exposed in their homes and the half-life ($t_{1/2}$) of the congener. This was intended to serve as a back calculation to determine how much PCB (i.e., dose) could be attributed to a specific pathway of exposure and then compare to the measurement in serum, which incorporates all pathways of exposure. A constant dose by each pathway over time was assumed, even though certain exposures, like diet, are likely to have changed over time. This also includes the assumption that measurements (e.g., PCBs in indoor air, dust and hand wipes) and parameters (e.g., body surface area, food consumption, and lipid mass) are constant over time. Pathway-specific EDIs were converted to a daily dose (i.e., mass of PCB per day), then normalized to body lipid (BL), which was calculated based on BMI and age, as in Aylward et al. (2014) (**SI Section S.4**). Sums of these body burden concentrations were compared then to serum concentrations, as were previously reported in Frederiksen et al. (2020), to assess the validity of these calculations. These were also assessed via Spearman correlations. Serum measurements for specific congeners were considered if detection frequency was >70%, and non-detects in serum were imputed to the limit of quantification (LOQ)/2.

PCB body burdens were assessed in a one-compartment, first-order model, as outlined in Lorber (2008) and Aylward et al. (2014). Since the residents all lived in the homes for different lengths of time, and the samples as well as personal measurements were cross-sectional, time (t) as the exposure period was assessed based on the time residents reported living in the contaminated apartment. These calculations were only conducted among the exposed population, as the body burden at time 0, $C_{PCB}(0)$, which was defined as the baseline body burden of PCBs at the time of move-in to a contaminated apartment, was calculated from pathway-specific median EDIs from the reference population. The first-order dissipation rates of specific congeners in the body, k, was represented by $\ln 2/t_{1/2}$ [day^{-1}]. Half-lives used were intrinsic human elimination half-lives for individual congeners, by Ritter et al. (2011) for the majority of compounds. The half-life for PCB-101 was calculated by Schettgen et al. (2012) (Table S2).

$$Change\ in\ PCB\ over\ time = \frac{\partial C_{PCB}}{\partial t} = \frac{EDI_{pathway}}{BL\ (t)} - kC_{PCB}(t) \quad (4)$$

$$C_{PCB}(t) = C_{PCB}(0)^{*}e^{-kt} + \frac{EDI_{pathway}(t)}{BL\ (t)}{}^{*}\frac{1-e^{-kt}}{k} \quad (5)$$

A simulated example for an average exposed participant was also calculated for PCB-28, comparing exposure via inhalation and dermal uptake across the range of years lived in a contaminated apartment as reported within the study population.

3. Results & discussion

3.1. Study population & PCB measurements

Residents from both non-contaminated (n = 23) and contaminated buildings (n = 71) provided personal and environmental samples from their home environments. Four residents from contaminated buildings were excluded from analyses due to incomplete sample sets (i.e., missing dust, air, or hand wipes). As such, the total sample population was 90, with 23 from non-contaminated residences and 67 from contaminated ones. In general, characteristics of the study population, comparing the

exposed and reference groups, were very similar (Table S3), as previously observed by Frederiksen et al. (2020). These similarities expanded beyond physical characteristics of participants to years of living in their current residence and the time spent daily in their homes. As such, the primary difference between the groups was their exposure to PCBs via contaminant sources within their individual apartments. Notably, individuals in the exposed population had been living in contaminated apartments for an average of 15.5 years (Table S3). Two participants included in the reference population had briefly lived in a PCB-contaminated apartment within Brøndby Strand Parkerne (<5 years), but their move-out occurred >10 years prior to sampling. This also occurred among the exposed population, with some participants (n = 11) having lived previously in one contaminated apartment and moved to another contaminated apartment within the housing estate, hence the difference in years in current residence and exposed time period (Table S3). Questionnaire responses also reported that only one participant had worked directly with PCBs for a short time early in their career (i.e., over 30 years prior to study sampling), and nearly all others had not worked in a PCB-contaminated building to their knowledge, indicating that PCB exposure was likely attributed to the home environment.

Measurements in individual matrices have been previously reported in depth (Andersen et al., 2020; Frederiksen et al., 2020). Briefly, active air samplers, dust, hand wipes, and serum were analyzed for 15 PCB congeners. Among these matrices sampled from the exposed population, detection frequencies were >70% for individual congeners, with the exception of air for PCB-138 and -180, and serum for PCB-101. PCB congeners were detected notably less in reference population samples, with only 8 of 15 congeners detected in >70% of samples in both air and dust (Andersen et al., 2020). Previous reports of PCBs from hand wipes and serum examining home environments are limited, and in general, participants living in the contaminated apartments had lower levels in serum and on hand wipes than those who had been occupationally exposed (e.g., capacitor workers, e-waste recycling workers) and higher

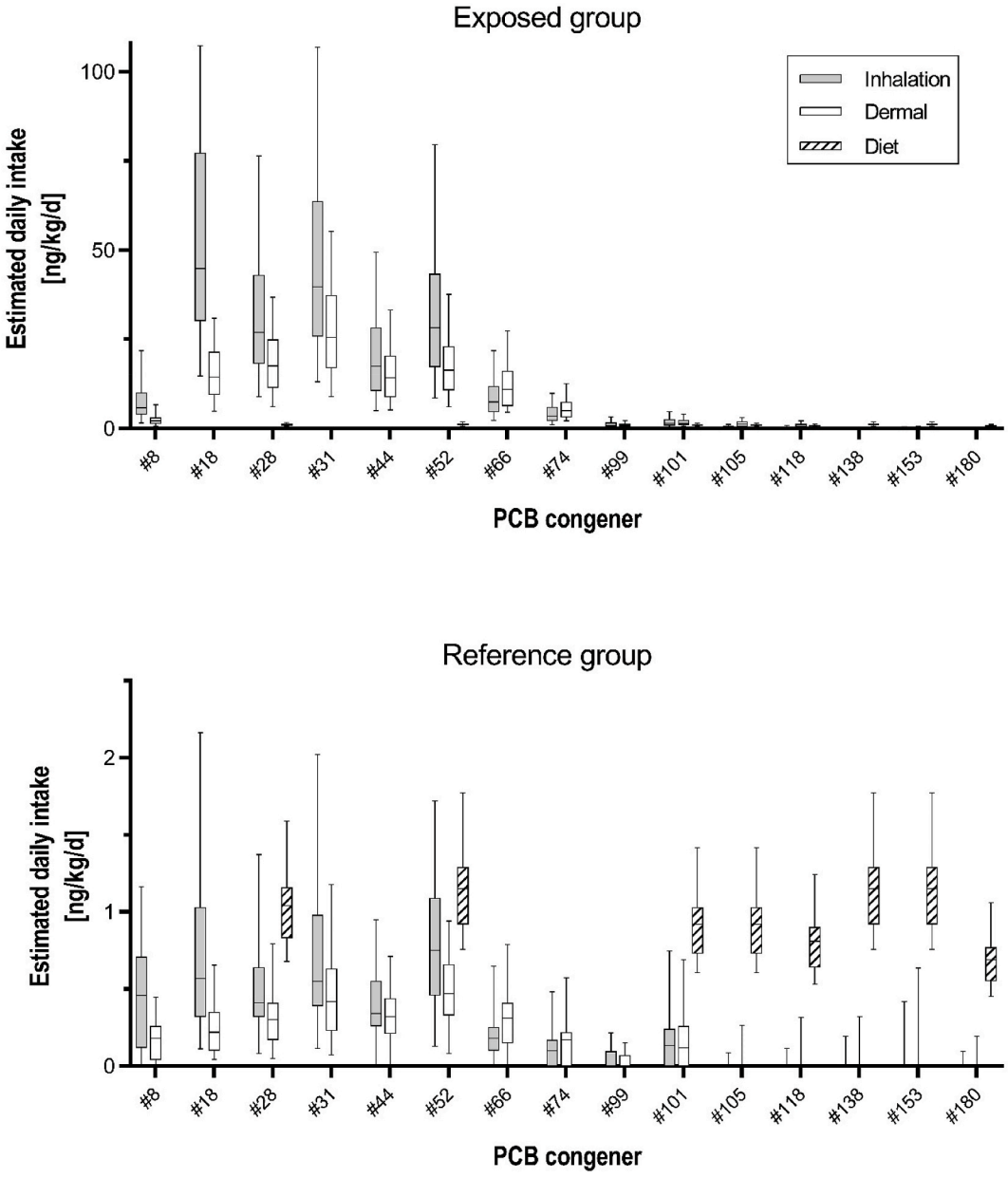

Fig. 1. Estimated daily intakes (interquartile range and 5-95% interval) for specific PCBs from inhalation, dermal absorption, and diet are shown for the exposed (above) and reference (below) populations. Dietary exposure was only available for PCB-28, 52, 101, 118, 138, 153, and 180.

serum levels than most previous evaluations in general populations (Frederiksen et al., 2020).

3.2. Dominant pathways of PCB exposure

3.2.1. Exposed population

Among the exposed population, inhalation was the most prominent pathway of exposure for PCBs, accounting for roughly 60% of the estimated daily intake (EDI) of PCB_{sum7} (Figure S1). For all individual PCBs up to PCB-101, inhalation of contaminated air was the largest contributor to PCB EDI, with a median inhalation EDI for PCB_{sum7} of 57 ng kg bw^{-1}·day^{-1} (Fig. 1). The large variation in EDI observed among the exposed participants, particularly for inhalation (Fig. 1), is due to the differences in both the PCB contamination level of the apartment and individual participant behaviors (e.g., time spent in the home daily). In addition, the median inhalation-specific exposure estimates over the course of a year was 1690 μg yr^{-1} for PCB_{sum7} and 5340 μg yr^{-1} for the 15 PCB congeners measured. When comparing these estimates to those calculated in Ampleman et al. (2015), this study would be among the highest observed exposure estimates in studies of contaminated environments and ambient air. Specifically, estimates from this study were similar in magnitude to residents in other studies of contaminated Danish homes (median = 1100 μg yr^{-1} for 24 measured PCBs) and slightly lower than workers in contaminated schools in Germany (median = 10000-36000 μg yr^{-1} for 6 PCBs, which overlap with PCB_{sum7}).

Dermal uptake was observed to be a significant secondary pathway of exposure, accounting for about 35% of the EDI for PCB_{sum7} (Figure S1). Together with inhalation, the two pathways accounted for about 95% of the EDI for PCB_{sum7}. The role of dermal absorption has often been neglected or underestimated (Weschler and Nazaroff, 2012), and thus, estimates and risk assessment of PCB exposure have relied on inhalation alone (Jensen, 2013). The calculations shown here demonstrate that while inhalation is the primary pathway of exposure, the dermal contributions can be substantial for some congeners and add significantly to overall PCB exposure. This is worth considering in terms of risk assessment and regulatory action as well as remediation, given how elevated the total EDIs were for exposed residents.

For the higher chlorinated PCBs (PCB-138, -153, −180), diet still played a prominent role, contributing about 7% to the PCB_{sum7} EDI and a large share of the calculated intake for these individual congeners. However, estimated total exposure and overall concentrations of these congeners in environmental samples were low, compared to the lower chlorinated PCBs, and thus, these constituted minor components of total PCB exposure overall.

The other two pathways (hand-to-mouth contact and inadvertent dust ingestion) contributed minimally to overall exposure, less than 0.5% each of PCB_{sum7} EDI (Figure S1). For both of these pathways, exposure is expected to be low based on physical-chemical properties of PCBs allowing for increased partitioning of PCBs to air compared to dust or being particle-bound (Andersen and Frederiksen, 2021). Given the high PCB content measured in the indoor dust of the contaminated apartments, these exposure pathways would likely be more important among children, who are closer to the ground, consume more dust inadvertently, and more frequently touch their hands to their mouths. This was observed in other previous evaluations, where non-inhalation sources such as diet were considered to be more prominent exposure pathways to PCBs for toddlers living in presumably non-contaminated residences, as compared to adults (Harrad et al., 2006). Individuals under age 18 were not included here; however, it is worth noting that children in various stages of development could have different relevant exposure pathways for PCBs while living in contaminated homes.

3.2.2. Reference population

Among the reference population, diet was the primary pathway of exposure, as evidenced by having the most prominent EDI among individual congeners and PCB_{sum7} (median PCB_{sum7} for diet = 7 ng kg bw^{-1}·day^{-1} and for all other pathways = 2 ng kg bw^{-1}·day^{-1}). PCB exposure via inhalation was the second most prominent pathway, particularly for lower chlorinated PCBs (Figure S1). Previous studies have estimated that up to 90% of human exposure to persistent organic pollutants (POPs) including PCBs are from dietary sources, and particularly fish (Darnerud et al., 2006; Fromberg et al., 2011). This is reflected in the reference group exposure and relative contributions of individual pathways, with roughly 80% of the EDI of PCB_{sum7} attributed to diet (Figure S1). The estimated intake via diet was also similar to EDIs reported in previous studies evaluating dietary exposure to PCBs across Europe and in Canada in the 1990s and 2000s (Aylward et al., 2014). The smaller contributions of PCB exposure from inhalation and dermal absorption are likely due to background air concentrations in the reference apartments. While notable, these air concentrations are comparable to measurements conducted in other uncontaminated Danish homes (Frederiksen et al., 2012), and adult inhalation exposure estimates are similar to air measurements in other non-contaminated indoor environments (e.g., homes, cars, offices) (Harrad et al., 2006). Again, hand-to-mouth contact and inadvertent dust ingestion contributed minimally, if at all, to PCB exposure among the reference population (Figure S1), which can be attributed to very low levels of PCBs measured on hands and in dust in non-contaminated environments. This demonstrates that there are still sources of PCB exposure outside of diet, and people are likely exposed to them on a daily basis, albeit minimally.

3.2.3. Comparison of study populations and relevant regulations

Among the exposed group, median PCB EDI across the 15 congeners, excluding dietary contributions, was 281 ng kg bw^{-1}·day^{-1}. Compared to similar reference group estimates (median Σ_{15}PCBs = 6 ng kg bw^{-1}·day^{-1}), residents living in contaminated apartments were exposed to PCB levels around 40 times higher than the reference group. Dietary contributions were excluded within this calculation due to the lack of dietary information available for several of these congeners. When including diet for the PCB_{sum7}, which constituted 7 ng kg bw^{-1}·day^{-1} for both groups and thus incorporating all measured pathways of exposure, the median PCB EDI was 101 ng kg bw^{-1}·day^{-1} for the exposed and 9 ng kg bw^{-1}·day^{-1} for the reference group. As such, with the inclusion of diet, the residents of contaminated apartments still experienced roughly 10 times higher exposure or daily intake across the sum of indicator PCBs. This further emphasizes the importance of monitoring indoor environments and underlines how contamination sources within residences can contribute heavily to personal exposure.

Currently in Denmark, there is no set tolerable daily intake (TDI), and as previously mentioned, current recommended action values rely on indoor air levels based on an older German TDI of 1 μg kg bw^{-1}·day^{-1}. This German TDI was established from a toxicological study with long-term exposure of rats to a technical PCB mix and typically compared to air concentrations by multiplying 5 times a sum of 6 indicator PCBs, which overlaps with the seven here (Jensen, 2013). With a median measured EDI across all routes of exposure for PCB_{sum7} of 101 ng kg bw^{-1}·day^{-1} (which is about 505 ng kg bw^{-1}·day^{-1} after accounting for the corrections factor), nearly all of the exposed population falls below the established German TDI, with only individuals above the 95th percentile exceeding the limit. The French Food Safety Authority (AFSSA) TDI recommends that the sum of 6 indicator PCBs should not exceed 10 ng kg bw^{-1}·day^{-1} (AFSSA, 2007; Duedahl-Olesen et al., 2020), which was based on the World Health Organization (WHO) discussion of PCB TDI and does not leave any room for exposure from sources other than food. A similar TDI of 20 ng kg bw^{-1}·day^{-1} for Aroclor 1254 was recommended by the WHO, although the same TDI had been proposed for the sum of all 209 congeners (AFSSA, 2007; Faroon et al., 2003; Jensen, 2013). In this case and with the consideration of multiple pathways of exposure, all of the exposed group would exceed AFSSA values. With the stringency of the AFSSA TDI, a few of the reference group also exceeded the AFSSA limit with background

concentration exposures to PCBs.

3.3. Dermal absorption from air and hand wipes

The EDI for dermal absorption was calculated using both PCB concentrations from indoor air and hand wipes from participants' palms, based on equations described in Weschler and Nazaroff (2012). EDIs calculated using both methods were very similar, and the EDIs from indoor air are presented for comparisons to other exposure pathways. Among the exposed population, the two differently derived EDIs were highly correlated ($r = 0.94$, $p < 0.0001$, Figure S2) for the 15 PCB congeners assessed. Their median values were also very similar, indicating that the two methods could be interchangeable and are strongly related (Table S4). Although less congeners were detected in indoor air of reference apartments (i.e., lacking detection of the higher chlorinated PCBs), the same trend was observed among the reference participants as well (Table S4).

These two methods of calculating dermal absorption estimate the same outcome; however, the hand wipe is expected to yield a higher estimate than indoor air because the wipe, particularly of the palms, would capture surface contact in addition to partitioning from air to skin. A skin wipe without the potential for surface contact, such as a wipe of the backs of hands, would likely better reflect dermal absorption from air concentrations, although the two wipes have been shown to be correlated (Yang et al., 2019). From a small number of exposed residents in this study ($n = 6$), both wipes of palms and backs of hands were collected and analyzed separately (Frederiksen et al., 2020; SI). Palm wipes contained slightly higher concentrations of PCBs than the back-of-hand wipes; however, there was not a clear trend across the measured PCBs. Similarly, median dermal EDIs were not consistently higher among hand wipe estimates compared to indoor air, and an obvious trend was not evident (Table S4). The high correlation between the two separate measures across congeners, which cover a wide range of physicochemical properties, could be because the individuals' skin surfaces are approximately at equilibrium with the air in the contaminated apartments. This is assumed in the calculations in Weschler and Nazaroff (2012). The concurrence between the two methods could indicate that a measurement of PCBs in indoor air or skin wipe would yield similar results for the consideration of personal exposure. Any differences between these two calculation methods are likely due to assumptions made regarding PCB concentrations on the hands (i.e., skin surface lipids on palms are likely to be a thinner layer than average body skin lipid thickness) as well as possible differences in estimating parameters (e.g., mass-transfer coefficient, for transdermal permeation from indoor air, and partitioning coefficients, such as Henry's Law constant). Nonetheless, because the hand wipes could be incorporating surface contact with air-to-skin partitioning, the dermal EDI calculated from indoor air was used here in all comparisons to the other pathways of exposure for PCBs.

Previous estimations of dermal absorption from skin wipes or other samples have been calculated frequently using a dermal absorbance fraction, utilizing a range of factors from 14% (Wester et al., 1993) to near 100%, including congener-specific absorbance fractions (Ertl and Butte, 2012; Garner and Matthews, 1998). However, Kissel (2011) pointed out that fractional absorption misrepresents dermal absorption, due to skin loading conditions and flux considerations. In this case, there is likely an 'infinite supply' of PCBs to the skin (i.e., more PCB than can be absorbed by a person's skin). As such, conditions for dermal absorption would likely be flux-limited, based on Kissel's argument. Fractional absorption would not be appropriate for our assessment, particularly with the conditions of contaminated residences as the exposure is likely continuous; thus, we instead utilized transdermal flux to determine PCB partitioning through the skin and into the dermal capillaries for our calculations of dermal absorption.

3.4. Body burden in exposed residents

Pathway-specific body burdens were calculated for the seven indicator PCBs and their sum for the exposed population, with the reference group exposure medians serving as the baseline measurement, $C_{PCB}(0)$. This allowed for comparison to serum, as in a pathway-specific exposure contribution to PCB presence in serum over time. Similar to results from the EDI calculations, inhalation exposure contributed the most to PCB body burden among exposed participants, and PCB-28 was the most prominent congener of the seven indicators (Fig. 2). Again, dermal absorption accounted for significant exposure as well (Fig. 2). As expected, diet appeared to play a larger role among the higher chlorinated PCBs (PCB-118 and larger); however, this contribution was small compared to the overall PCB sum relative to the body burden of the lower chlorinated PCBs.

Numerous studies have investigated associations between indoor air and serum and have demonstrated that PCBs in indoor air are positively and significantly associated with serum PCB concentrations, particularly for lower chlorinated congeners (Ampleman et al., 2015; Kraft et al., 2018, 2021; Meyer et al., 2013). However, no studies to the authors' knowledge have previously conducted a similar assessment, evaluating pathway-specific body burden including dermal uptake and based on PCB half-lives and cumulative exposure over time. One such evaluation was conducted by Lorber (2008) for the flame retardants polybrominated diphenyl ethers, where dermal contact with dust was considered. This lack of assessments is likely due to the fact that it has been rare to find such high contamination in home environments and elevated serum levels among residents (Frederiksen et al., 2012, 2020; Meyer et al., 2013). Other studies of PCB exposure have largely focused on diet, specific occupational exposures or exposures within contaminated schools or office buildings (Ampleman et al., 2015; Aylward et al., 2014; Herrick et al., 2016; Kraft et al., 2018).

As PCB-28 was the most abundant individual contributor of measured congeners to overall PCB body burden, inhalation and dermal pathways of uptake into the body were modeled based on median indoor air concentrations and compared across a range of residence times (1–45 years) for this congener specifically. Assuming constant air concentrations and consistent daily exposure, body burden of PCB-28 appeared to plateau at roughly 20 years of living in a contaminated apartment (Figure S3). Uptake of PCB-28 from living in a contaminated home is fastest during the first decade of living in that residence. The median years of residence in the exposed group was 15.5 years, and about one-third of the residents had lived in a contaminated apartment for over 20 years (Table S3). This suggests that for roughly one-third of residents within the exposed group, body burdens of PCB-28 would have stabilized by the time of sampling, based on solely inhalation and dermal absorption estimates.

Further, as the two largest contributors to the overall PCB_{sum7} body burden across pathways, PCB-28 and -52 were significantly correlated with their corresponding measured serum concentrations ($r_s = 0.49$, 0.45; $p < 0.001$), while the other individual congeners were, in general, positively correlated (Fig. 3). When compared to PCB-52, the estimated body burden sum for PCB-28 congregated more closely to the 1:1 line, indicating that PCB-28 body burden estimates better matched the actual measured serum values. The calculated PCB_{sum7} body burden was also significantly and positively correlated with the summed PCBs measured in serum ($r_s = 0.45$, $p < 0.001$; Fig. 3); however, this is likely driven by the dominance of the body burden of PCB-28 and more limitedly PCB-52. A lack of correlation was observed for PCB-101 to −138 ($r_s = -0.01$-0.15), and relatively weak ones for PCB-153 and -180 ($r_s = 0.21$, 0.25) (Fig. 3), pointing to a general underestimation of body burden by the model. This may be due to the restriction of having evaluated diet over residence time rather than residents' age, as these congeners have long half-lives and are typically attributed to dietary exposure across a lifetime. Such an underestimation could indicate the need for inclusion of a lifelong dietary exposure estimate within future

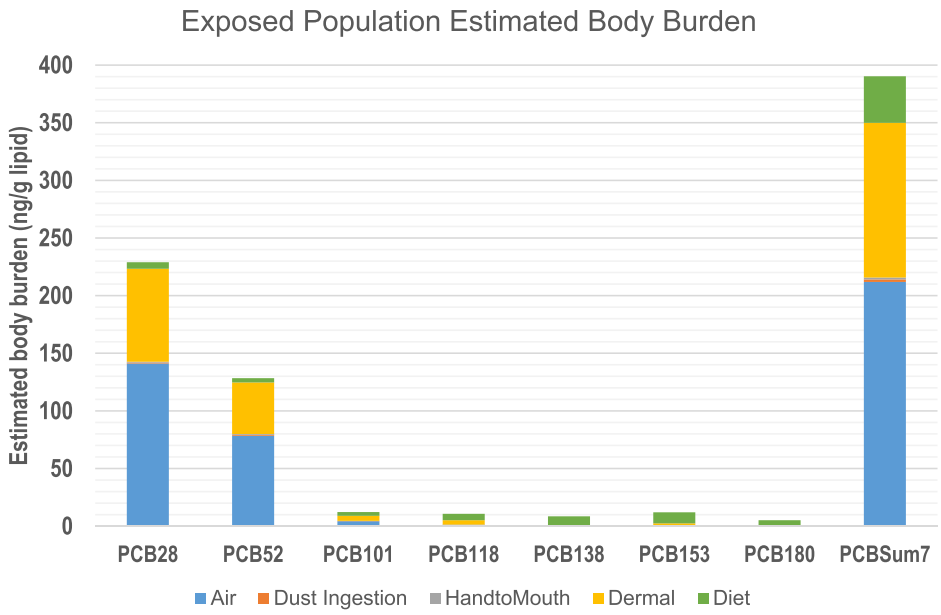

Fig. 2. Median pathway-specific body burdens were calculated for each PCB congener and sum of 7 indicator PCBs for the exposed population. These were normalized to individual estimation of body lipid mass and account for the time participants lived in the exposed apartments, indicating a body burden at the time of sampling while assuming a constant EDI over time

models.

Despite several promising positive correlations, there are evident gaps in the actual numerical body burden values, particularly for PCB-52, which may be caused by a number of factors. The half-life parameter used here could have influenced body burden estimations, as studies have reported a wide range of intrinsic PCB half-lives. We utilized half-life estimations from Ritter et al. (2011) and Schettgen et al. (2012), as they agreed with each other and a number of other studies; however, it is possible that overestimations of the lower chlorinated PCB half-lives and under-estimations of the larger PCB half-lives could have led to the gaps observed for comparing body burden to serum measurements. For instance, a more recent paper by Esser et al. (2021) suggested a half-life of 0.8 y for PCB-52; compared to the half-life of 2.6 y estimated by Ritter et al. (2011), this would yield a much smaller body burden and possibly better estimate serum measurements. For the higher chlorinated PCBs, it is possible that some of the older and longer-term residents may have consumed more PCB in their food (e.g., fish) years before, and thus the dietary estimates presented here were underestimated for the higher chlorinated PCBs. Further, air data was not available for some of the higher chlorinated PCBs, which could have contributed to lesser estimations of exposure to these larger PCBs, since we utilized indoor air concentrations for the calculations of body burden from dermal absorption. However, the magnitude of the dermal estimated doses, when compared between air and hand wipes, suggests that such dermal estimations were likely very small (Table S4). Further explanation for observed differences could include assumptions included in the model, such as assuming constant environmental conditions, equivalent intakes daily, and consistent and maximum flux for dermal uptake throughout the exposure period. In general, the exposure estimates from living in the contaminated homes reflected overall residents' PCB exposure and internal dose measurements, indicating that measurements in home environments could be adequate for future risk assessment.

3.5. Study limitations

Although extensive measurements were conducted among study participants and their respective residences, evaluation of how they have been exposed to PCBs and which route contributed most is not without limitations. First, inherent to calculation of an EDI are a series of behavioral parameters, which were not all measured or queried via questionnaire and thus approximated with the best available information. These variables, such as inhalation rate and total body surface area, were taken from the USEPA Exposure Factors Handbook with estimations based on age and sex of participants.

In addition, recruitment of study participants resulted in a skewed overall study population, which limited our ability to conduct more extensive and sophisticated analyses to compare the two exposure groups. Extensive efforts were made to recruit both exposed and reference participants; following recruitment, all interested participants were included within the study, resulting in the sample sizes here. While this may limit our analyses in comparing the study groups, stratification or limiting analyses to only exposed individuals allowed us to evaluate and compare the relative exposures and estimated intake. As such, we do not expect the sample size inequality to affect the internal validity of our results. We acknowledge, nonetheless, that the sample sizes did not allow us to investigate the impact of other behavioral parameters (e.g., smoking) since very few individuals in both study groups indicated that they were current smokers (Frederiksen et al., 2020).

Estimations of dermal uptake here assumed absorption from the total body skin surface and did not take the role of clothing or other linens which come in contact with the skin (e.g., blankets, sofas coverings, and bedsheets) into consideration. Previous work in the same apartments had assessed nine types of clothing fabrics for absorption of PCB-28 and -52 and indicated that clothing hanging in the apartment can serve as a reservoir for PCBs, thereby potentially contribute to continuous dermal absorption when individuals are not in their contaminated apartment (Morrison et al., 2018). Laundering of clothes and linens could remove a large portion of PCB content following contamination; however, these materials acting as contributors or barriers to exposure depend on time between washings and their exposure to PCB-contaminated air (Kolarik and Morrison, 2022). Based on this assessment, we assumed absorption from the total skin surface as a conservative estimate, while noting that not enough is known currently about the role of clothing as a source or barrier to dermal uptake.

Further, all estimations of body burden assume constant exposure for each pathway as integrated over the exposure period, which is

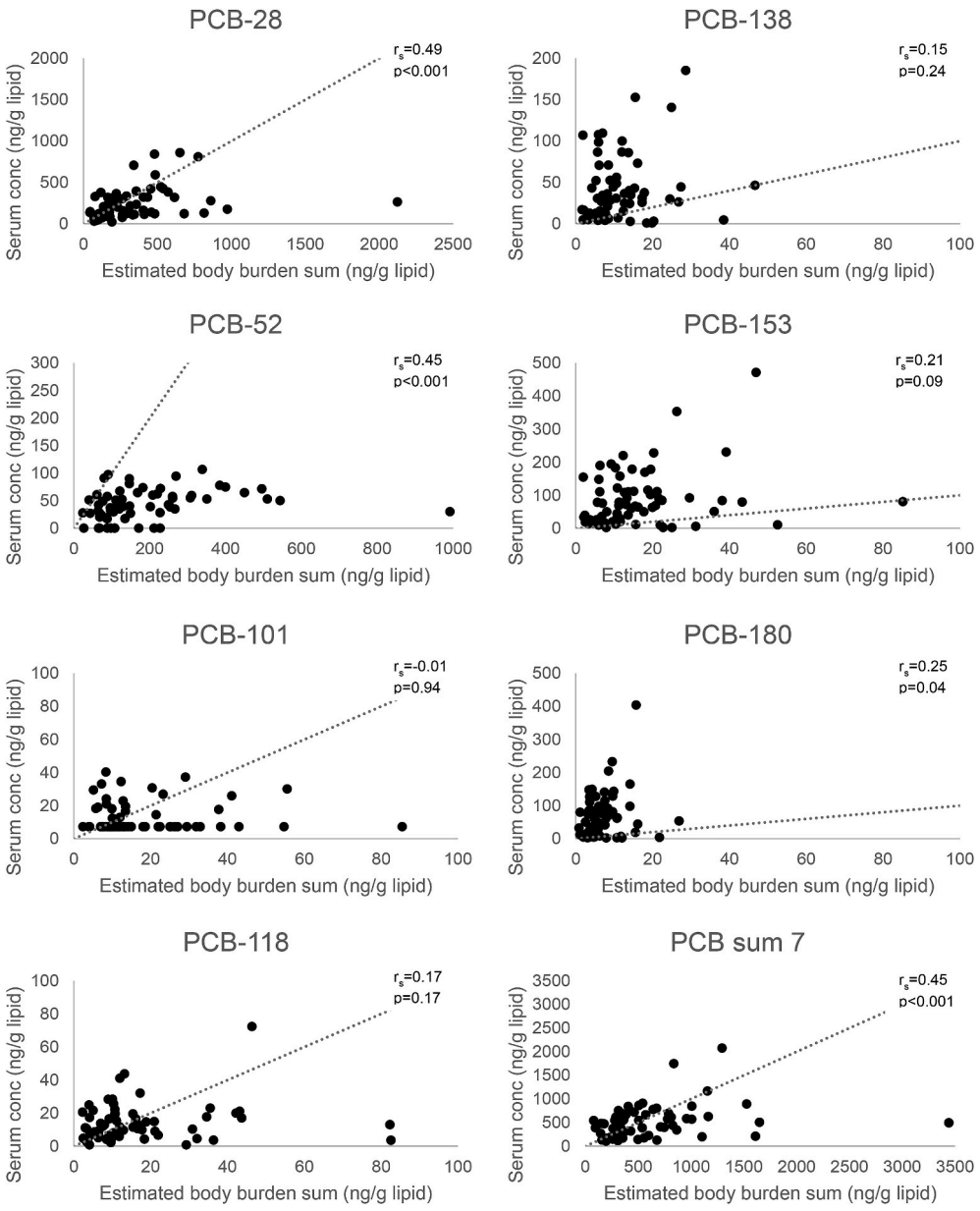

Fig. 3. Estimated body burden of individual exposed residents from all calculated pathways, normalized to individual body lipid mass, was compared to serum measurements for individual and summed PCBs. A 1:1 trendline is presented, demonstrating where body burden over- or under-predicted measured serum levels.

inherently the entire time in which participants have lived in their current residence. Samples within the homes were taken at one time point and then used to estimate exposure retrospectively. While the sample measurements themselves had a high level of validity, PCBs in indoor dust can be fairly consistent over time ($t_{1/2}$ = 5–18 years) whereas indoor air concentrations may fluctuate with temperature and season (Andersen et al., 2021; Whitehead et al., 2014). As such, these measurements may not be representative of many years of exposure, particularly if certain residents moved into the apartments when they were first built. Body burden calculations also assume similar body lipid mass, which is likely to fluctuate throughout a person's life by virtue of age and other factors. Other behaviors reported by participants at time of sampling such as time spent at home could also shift over time, and equating exposure period to number of days exposed does not take into account any time that participants spent away from their homes (e.g., holidays, visiting friends and family).

With regard to diet, calculations of dietary exposure were based on PCB content in food measured in Denmark in the late 1990s to early 2000s (Fromberg et al., 2011). PCB content has been slowly decreasing in food, including in fish and seafood, since PCBs have not been actively applied in decades (Saktrakulkla et al., 2020), and this has also been the case for dietary exposure in Denmark (Duedahl-Olesen et al., 2020). Thus, between the decreasing PCB content in food and less fish consumption, our estimations of dietary PCB exposure are potentially overestimations of the actual dietary contribution. This effect could be modulated by age and habits of residents, including dietary consumption prior to living in the contaminated apartments. We restricted the consideration of dietary intake only to the years in which residents lived in the apartments, for the purpose of comparing pathways of PCB exposure while recognizing that this assumption could underestimate lifetime dietary exposure, particularly for some of the higher chlorinated PCBs. Dietary information was only available for 8 of 15 PCB congeners; however, it is likely that the lower chlorinated PCBs were not as abundantly present in food as some of the higher chlorinated ones, such as PCB-138 and -153, and thus may not contribute significantly to overall exposure (Fromberg et al., 2011). The relative importance of exposure

pathways will be somewhat dependent on which congeners are included. In addition, exposure via diet did not consider potential deposition of PCBs onto food surfaces (i.e., partitioning of PCBs from air, dust, or airborne particles to food) while in contaminated apartments prior to consumption of the food.

Finally, physicochemical properties of the PCB congeners and any necessary adjustments with temperature (25 °C to skin surface temperature, 32 °C) were estimated primarily using SPARC software. In particular, we relied on Henry's Law constant, H, and log K_{oa} for calculating dermal absorption. Previous work has demonstrated that there are systematic inconsistencies across PCB congeners for certain experimental and modeled estimates of these parameters, which have been discussed in the context of air and dust partitioning (Andersen and Frederiksen, 2021). Here, we have utilized SPARC exclusively for calculating physicochemical properties, due to their consistency with other published values for PCBs (Li et al., 2003). So, any uncertainty within these properties, specifically with Henry's law constant as it relates to dermal uptake from indoor air, could have an effect on the magnitude of these results; however, the trends observed are not likely to deviate tremendously and dermal estimations from air tracked closely with hand wipe data, suggesting that these parameters would not impact the interpretation of our results here.

4. Conclusion

Here, we present estimations of daily intake and pathway-specific body burden of a suite of PCBs for residents of contaminated apartments and a related reference group. With high PCB concentrations in indoor air of contaminated residences, particularly of lower chlorinated PCBs, inhalation was the primary pathway of exposure for residents, and inhalation and dermal absorption combined accounted for roughly 95% of total estimated daily intake of the sum of 7 indicator PCBs. Among the reference group, diet was the primary pathway of exposure, confirming that general exposure to PCBs still comes predominantly from food sources. Assessments of body burdens across relevant pathways for PCB-28 and -52, as well as PCB_{sum7}, from these exposure estimations were significantly correlated to serum measurements, suggesting that these models could potentially predict internal dose over time. However, differences in the values between body burden and measured serum suggest that more detailed environmental and behavioral characterization, including lifelong dietary consumption, in exposure assessment as well as further investigations into PCB half-lives and partitioning characteristics should be considered. This evaluation also highlights the importance of considering dermal absorption for future risk assessment and remediation measures, which has been largely neglected in exposure assessment, but could contribute substantively to overall exposure.

Credit author statement

Stephanie C. Hammel: Formal analysis, Methodology, Investigation, Visualization, Writing - original draft. Helle Vibeke Andersen: Conceptualization, Methodology, Project administration, Investigation, Funding acquisition, Writing-review & editing. Lisbeth E. Knudsen: Writing-Review & Editing, Project administration. Marie Frederiksen: Conceptualization, Methodology, Project administration, Investigation, Funding acquisition, Visualization, Writing-review & editing.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijheh.2022.114056.

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