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Persistent Organic Pollutants in Human Adipose Tissue from Normal pregnant Women A Review of Global Trends and Preliminary Data for Singapore

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Abstract:

Persistent Organic Pollutants (POPs) are man-made chemicals that have an intrinsic resistance to natural degradation processes, and are therefore environmentally persistent. Introduction of POPs into the environment from anthropogenic activities has resulted in their widespread dispersal and accumulation in soils and water bodies, as well as human and ecological food chains where they are known to induce toxic effects. Due to their ubiquity in the environment and lipophilic properties, there is mounting concern over the potential risks of human exposure to these POPs via the ingestion and inhalation pathways. This has led to the establishment of a worldwide research program to determine prevailing levels of POPs in the population and investigate the health risks associated with background exposure. This paper reviews the state of knowledge regarding residual levels of POPs in human adipose tissue worldwide, and provides preliminary data on the levels of key POPs in female adipose tissues collected in Singapore. Organochlorine pesticides (OCPs) were found to have a comparable level to levels reported for Poland in 2001, with a mean of 0.98 μg/g and 0.84 μg/g on a lipid weight basis respectively. For total polychlorinated biphenyl (PCB) congeners, the meanconcentration of 34 ng/g (lipid weight basis) is lower than values reported from Japan in 1980 and Belgium in 2000. Polybrominated diphenyl ethers (PBDEs) are present at similar levels to Belgium in 2000, at 3.7 and 4.7 ng/g (lipid weight basis) respectively.

Keywords- review, persistent organic pollutants, human adipose tissue, global trends, Singapore

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INTRODUCTION

Environmental xenobiotic compounds that are both persistent and bioaccumulative have the potential to induce adverse effects on human health. Persistent organic pollutants (POPs) are a group of compounds that are prone to long-range atmospheric transport and deposition, and readily undergo biomagnifications in food chains. The global ubiquity of POPs became apparent following their detection in even remote regions of the Earth, including Polar Regions, at levels posing risks to both wildlife[1] and humans[2].

Of the numerous POPs that are prevalent in our environment, a "black list" (www.pops.int) of POPs has been designated under the diplomatic signing of the Stockholm Convention in 2001. The compounds include: pesticides, namely: Aldrine, DDT, Dieldrine, Endrine, Heptachlor, Chlordane, Mirex and Toxaphen; industrial compounds, namely: Hexachlorobenzene and Polychlorinated Biphenyls (PCBs); and other chemical byproducts, namely: Polychlorinated dibenzodioxins (PCDD) and Polychlorinated dibenzofurans (PCDF) - a general name "dioxins" is used for PCDDs and PCDFs. These POPs are known to be particularly toxic with a strong propensity for biomagnifications, and have been associated with both carcinogenic and endocrine disrupting effects in a range of biota. Although the effects on human health from environmental exposure to these POPs remains unclear, there is growing concern over elevated concentrations of a broad spectrum of POPs in a range of human tissues, including blood, adipose tissue and breast milk. Due to their lipophilic properties, POPs readily accumulate in human adipose tissue following ingestion of contaminated foodstuffs, and serves as a useful matrix for comparing accumulated levels in different countries.

POPs may be introduced into the environment from a variety of emission sources and anthropogenic activities. Point, area and line sources include releases from industrial installations, domestic premises, traffic, waste disposal operations such as incinerators and landfills, and activities such as crop stubble burning and the spreading of sewage sludge on land. Area sources can also include the release of POPs from secondary sources such as contaminated land masses or water bodies which have accumulated POPs from both historical and ongoing deposition. The release of POPs into the environment may be subject to partition coefficient (Kow). The BAF and the BCF measure the concentration of a substance in a living organism relative to its concentration in the surrounding medium. The criteria to determine the propensity of an organic chemical to undergo bioaccumulation was specified in the Toxic Substance Management Policy (TSMP) under the Canadian Environmental Protection Act of 1999 (www.ec.gc.ca), which states that a BCF or BAF which exceeds 5,000, or the logarithm of the Octanol-water partition coefficient (Log Kow) of the chemical substance exceeds 5 indicates a propensity to accumulate in lipids. As a result of elevated hydrophobicity (Kow >5), POPs are readily concentrated and retained in the lipid tissues of biota. Humans, typically at the top of the food chain accumulate the highest concentrations of these hydrophobic and persistent compounds.

For many years, residues of POPs have been readily detected in the human adipose tissue of individuals in a number of countries, including those living in Europe, Asia, Africa and North America. In this study, we review available data from the scientific literature for levels of POPs in human adipose tissue from various countries around the world. We also report the first such data from Singapore and evaluate levels in the global context.

2. Pesticides

Toxicity and persistence of pesticides are useful properties for killing their target organisms, but these qualities also cause problems for humans and the environment. The occurrence of organochlorine pesticides (OCPs) in the environment and subsequently in the food chain of humans and wildlife has been noted since the early sixties[4]. Toxicological investigations have shown that several pesticides are carcinogenic in animals, thus raising concern over human exposure[3]. To date, this concern has led to several OCPs being restricted or banned for agricultural and/or disease vector control (WHO 1989). However, secondary emissions of pesticides accumulated in various environmental compartments, including soils, sediments and water bodies are expected to persist long into the future. Subsequent transport via the atmospheric pathway ensures that pesticides are widely dispersed from their source, regionally and even globally, thereby representing a ubiquitous threat to human health and wildlife.

Dichlorodiphenyltrichloroethane (DDT) and its derivatives have now been found in most environmental media, and constitute the dominant OCPs found in human tissues, most notably adipose tissue. DDT is organochlorine compound which was first synthesized in Germany in 1874. DDT's insecticidal properties were realized in 1939, and subsequent commercial use began in 1945. In the human body, DDT is first dechlorinated to tetrachlorodiphenylethane (DDD) which is water soluble and less toxic to human health. Another group of DDT derivatives includes dichlorodiphenyldichloroethanes (DDEs), which can readily accumulate in human

adipose tissue, and represents a significant health threat due to its long half-life. DDE may be accumulated via metabolism of DDT in the organism itself, or upon ingestion of DDE tainted foodstuffs[4]. Amongst the isomers in the DDE group, anti-androgenic 4,4'-DDE is the most abundant, with concentration in the human adipose ranging from 3.5 to 3229 ng/g on a lipid basis in Greenland[9]. In contrast, other pesticides, including Aldrin, Dieldrin, Lindane, Heptachlor and Heptachlor Epoxide, are generally present at low concentrations or below analytical detection limits at the ng/g level (lipid weight basis)[10] .

Geographically, no major differences in the levels of DDT have been reported for human adipose tissues. Based on available data (Table 1), mean concentrations of _DDTs in Asian regions is approximately 2.8ug/g, and for Europe and Greenland about 3.3ug/g and 4.0mg/kg, respectively. The highest levels of _DDT, with a mean of 84.3ug/g in 1989 and 25.7ug/g in 1991 respectively, were found in persons living in a Veracruz city, Mexico[11]. Statistical analysis showed that age was positively correlated to levels of several OCPs in adipose tissues of persons living in British Columbia[13], but no statistically significant differences were found between sexes[12].

Reporting on the storage of the separate isomers in fat tissue among the Hexachlorocyclohexanes (HCHs) group of pesticides, investigators found that beta-HCH was the predominant isomer. Due to its greater stability and lipophilic property, beta-HCH is metabolized very slowly and thus eliminated from the human body at a lesser rate than other HCH isomers[5]. Eighteen years of monitoring data from Holland between 1968 to 1986 showed that beta-HCH concentrations were persistent over time, with little evidence of a marked decline[5].

Hexachlorobenzene (HCB) is another important pesticide contaminant[5] in human tissues. HCB enters the environment via agricultural use as a fungicide and as an industrial byproduct. It is chemically stable and highly recalcitrant in the environment. Due to its persistence, specifically in the tissues of animals of a higher trophic status, HCB continues to pose a threat to public health. In the early seventies, in the Netherlands, an increase of HCB in human adipose tissues was associated with an increase in HCB concentrations in consumer products of animal origin[5]. Following a ban in some EU countries in the 1970s', there has been a gradual decrease of HCB concentrations in adipose tissues of Dutch citizens, the concentration of HCB (0.7mg/kg) in 1986 was half (1.3mg/kg) that reported in 1968/69[5].

3. Dioxins, Furans and Polychlorinated Biphenyls

Dioxins (PCDDs) and furans (PCDFs) are polyhalogenated aromatic hydrocarbons of high toxicity. There are a total of 210 different congeners; 75 dioxin congeners and 135 furan congeners, of which 17 are potentially toxic. Dioxins and furans are now found prevalently in air, water and soil in almost all natural environments. PCDD/Fs are strongly bound to organic matter, where half-life in soil has been estimated at 10 to 20 years[47]. PCDD/Fs enter the environment primarily as unintentional by-products of combustion and chemical processes. Waste incinerators have been identified as one of the major sources in the urban environment, and others include uncontrolled combustion such as backyard burning. Car exhaust emissions, especially from cars using leaded gasoline with halogenated scavengers also contain considerable levels of PCDD/Fs.

From studies conducted in the 1980s', persons inhabiting industrial areas were found to have higher adipose tissue dioxin levels[46]. For example in rural China values of 0.142ng/g of dioxin were reported in 1984- almost ten times less than levels reported for the industrialized areas of Japan, Canada and New York, USA (Table 2). PCDD/F levels at 1.75 ng/g were measured in adipose tissues samples from certain areas of South Vietnam in 1984 – ten times higher than concentrations measured in the north of the country. Over 170 kg of 2,3,7,8-TCDD, a congener of PCDD, was sprayed onto vegetation as a defoliant from fixed wing airplanes or helicopters to certain areas in the south of Vietnam during the war between 1962-1970 and is the direct cause of the high PCDD/F levels found in human adipose tissues[48].

The National Human Adipose Tissue Survey (NHATS) was first conducted by the United States Environmental Protection Agency in 1987. An average concentration of 5.38 pg/g of 2,3,7,8-TCDD was reported in the adipose tissue of the US population, increasing from 1.98 pg/g in children under 14 years of age to 9.40 pg/g in adults over 45 years. Due to the different health risks associated with individual PCDD/F congeners, the International Equivalency (I-TEQ) factor was established by the North Atlantic Treaty Organization (NATO) in 1989. More recently, in 1998, the World Health Organization specified Toxic Equivalency Factor (WHO-TEQ) values with slight different weighing coefficient for PCDD/Fs toxicity compared with those from I-TEQ, and as such is not directly comparable for risk evaluation in human health.

A mean level of PCDD/PCDFs has been reported at 35.6 pg I-TEQ/g lipids in adipose tissue of citizens of France. Samples analyzed in 1999 were found to have

Table 1: Levels of pesticides in human adipose tissue from various countries. Mean concentrations (ug/g on a lipid weight basis)

Country & Sampling Time	p,p'-DDE	p,p'-DDD	p,p'-DDT	_DDTs	_-HCH	_-HCH	_-HCH	_HCHs	References
Japan 1986-1987	√	√	√	2.4	√	√	√	1.8	Kashimoto T., et al., 1989[15]
Greenland	3.1	–	0.9	4.0	–	–	–	–	Éric Dewailly et al., 1999 [9]
Denmark	1.8	–	0.3	2.1	–	–	–	–	Éric Dewailly et al., 1999 [9]
Finland	2.150	–	–	3.476	–	–	–	–	Hattula et al. 1976 [5]
Finland	0.360	–	–	0.383	–	–	–	–	Mussalo-Kuuhamaa, et al 1984 [6]
Finland	0.535	–	–	0.557	–	–	–	–	Mussalo-Kuuhamaa, et al 1991 [7]
Finland	0.567	0.006	0.011	0.584	–		0.178	0.204	Smeds A, Saukk O P, 2001 [8]
Mexico 1988	–	–	–	17.45	–	–	–	_	Waliszewski SM, et al,.1996 [10]
Mexico 1991	–	–	–	14.60	–		–	–	Waliszewski SM, et al,.1996 [10]
Northern Italy 1989	0.395	–	0.064	0.459	–	0.213	0.104	0.317	Gallelli G, et al., 1995 [11]
Poland 1990	√	√	√	15	√	√	√	0.25	Tanabe S., et al 1993 [16]
Poland 1997-2001	0.770	–	0.072	0.842	–	0.064	–	–	Strucinski P, et al., 2002 [13]
Spain 1991	√	–	√	4.4	–	√	–	1.53	Gómez-Catalán J., et al., 1995 [17]
South Vietnam 1991	√	√	√	4.9	√	√	√	0.03	Nakamura H., et al., 1994 [18]
Korea 1994-1995	1.0	0.007	0.056	1.1	0.002	0.18	0.0003	0.182	Youn-Seok Kang, et al., 1997[19]
Belgium 1996-1998	0.471	0.019	0.097	0.587	–	0.004	0.0052	0.0045	A. Pauwels., et al., 1997 [20]
Singapore 2003-2004	0.789	0.045	0.143	0.977	0.033	0.214	0.025	0.272	This study

"–"Not target compounds

"√" No value reported

Table 2: Levels of PCDDs, PCDFs and PCBs in human adipose tissue from various countries. Mean concentrations (ng/g on a lipid weight basis)

Country and sampling time	PCDD/Fs	PCBs	References
Korea 1994-1995	19[a](males) 16.5[a](Females)	–	Kang., et al., 1997[19]
France 1999	35.6[a]	–	Arfi., et al., 2001 [24]
Japan 1970-1971	31.6[a]	–	Choi., et al., 2002 [25]
Japan 1994-1996	31.5[a]	35.4[a]	Choi., et al., 2002 [25]
Japan 1998-1999	49[b]	17[b]	Takenaka., et al., 2002 [28]
Japan 2000	11.9[a]	15.3[a]	Choi., et al., 2002 [25]
India	14-46[b](males) 16-56[b](females)	_	Kumar., et al., 2001 [26]
Spain 1997-1998	31[a], 36.3[b]	25.2[b]	Schuhmacher., et al., 1999c [22] & Wingfors., et al., 2000 [23]
Spain 2002	9.2[a], 11[b]	10.8[b]	Marta., et al., 2004 [29]
Italy	2.81-13.2	_	Baklassarri., et al., 2002 [27]
North of Vietnam 1984	0.142	–	Schecter, A., et al., 1986 [48]
South of Vietnam 1984	1.749	–	Schecter, A., et al., 1986 [48]
China 1984	0.113	–	Ryan, J.J., et al., 1987 [47]
Japan 1984	1.667	–	Ryan, J.J., et al., 1987 [47]
Canada 1976	1.017	–	Schecter, A., et al., 1986 [48]
Canada 1980	0.915	–	Schecter, A., et al., 1986 [48]
USA, New York 1982-1983	1.047	–	Schecter, A., et al., 1986 [48]
Finland 1984	<0.002-7.70	–	Koistinen J., et al., 1995[31]
Belgium 1996-1998	_	373.1 (7 congeners)	A. Pauwels., et al., 2000 [20]
Belgium 2000	–	879.7 (35 congeners)	Covaci A., et al.,2002 [34]
Poland 1979	–	1200*	Shinsuke Tanabe., et al., 1993 50]
Poland 1990	–	1500*	Shinsuke Tanabe., at al., 1993 [50]
Japan 1980	–	3000*	Loganthan., et al., 1990 [51]
Japan 1981	–	3100*	Mori et al., 1983 [52]
Japan 1986-1987		775*	T. Kashimoto., 1989 [57]
Singapore2003-2004	–	34 (40 congeners)	This study

[a] PCDD/F and PCB toxic equivalents (TEQ) are given as I-TEQ.
[b] PCDD/F and PCB toxic equivalents (TEQ) are given as WHO-TEQ.
[C] TEQ of PCBs included in that of PCDD/Fs.
*: numbers of congeners not specified

Table 3: Mean levels of PBDEs (ng/g on lipid weight basis) in human adipose tissue from various countries. Mean concentrations (ng/g on lipid weight basis)

Country and sampling time	PBDEs	References
USA, California	28.9 (PBDE47)	Washam C., 2003 [32]
USA, San Francisco Bay	85.7 (PBDE47, 99, 153, 154)	Jianwen She., et al., 2002 [33]
Belgium 2000	4.75 (PBDE28, 47, 99, 100, 153)	Covaci A., et al., 2002 [34]
Swede	5.36 (PBDE17, 28, 47, 66, 100, 99, 85, 154, 153)	Meironyte Guvenius D., et al., 2001 [35]
Swede	11.7 (PBDE47,99,100)	Haglund, P., et al., 1997 [38]
Swede	3.8-16 (PBDE47)	Lindström., et al., 1998 [39]
Swede	5.0 (PBDE28, 47, 85, 99, 100, 153, 154)	Meironyté Guvenius D., et al., 1999 [40]
Finland	6.3-22 (PBDE47, 99, 153)	Strandman., et al., 1999 [41]
Spain	25.1 (PBDE47, 99, 153)	Meneses., et al., 1999 [42]
Singapore 2003-2004	3.7(PBDE47, 99, 100, 153, 154)	Current study

similar levels to those reported for other European countries, and the USA. No relation to sex or age of the tissue donor was apparent, and levels can be considered as representative of prevailing concentrations of these compounds in most industrialized countries[24]. There was no obvious trend of PCDD/F levels found in adipose tissue in citizens of Japan between 1970 and 1999[25, 28] (Table 2). However, in 2000, the concentration of PCDD/F was reported as only one-third of the amount in 1999[25]. This magnitude of decline seems unlikely over such a short time interval, but a longer term reduction is supported by the downward trend in emission levels of PCDDs and PCDFs following implementation of strict emission regulations and associated abatement technologies[30].

Polychlorinated biphenyls (PCBs) are a family of 209 chemical compounds for which there are no known natural sources. PCBs were widely used as coolants and lubricants in electrical components and paint additives until they were widely banned in the 1970s by most developed nations. During their manufacture and use, PCBs were released into the atmosphere via industrial emissions, weathering of PCB containing materials, and incineration of PCB-containing products. However, PCBs continue to be released into the environment from leakage of defunct equipment, leaching from landfills, and from previously contaminated soils and sediments. Recent reports have shown that oily fish and Scottish-farmed salmon contain particularly high levels of PCBs. PCBs have the potential to affect thyroid hormone functions, thereby impairing mental development[45].

In a survey of Dutch citizens, the levels of PCBs did not change over a 10 year period between 1973 and 1983, where the persistence of PCBs in fatty tissues remained at a level of 3 μg/g (lipid weight basis)[5]. With mean levels of PCBs among Poland's population measured at 1200 ng/g and 1500 ng/g (lipid weight basis) in 1979 and 1990 respectively, the data indicates the potential of PCBs to persist in human adipose tissue over time[50]. Although a ban ended the use of PCBs in Japan in the 1976, high levels of PCBs, up to 3000 ng/g (lipid weight basis) were detected in human adipose tissue in 1981. However, the concentration of PCBs was reduced to a mean level of 775 ng/g (lipid weight basis) within 36 human adipose tissue samples analyzed in Japan in 1987[57].

Due to similar mechanisms of toxicity to dioxins for some PCB congeners, including non-ortho, mono-ortho chlorine substituted biphenyls, these compounds are also referred "dioxin-like" compounds and rated in terms of toxic equivalency I-TEQ or WHO-TEQ. As shown in Table 2, recent studies conducted in Japan between 1994 and 2000 reported dioxin-like PCB congener I-TEQ or WHO-TEQ values which can be used to compare toxicity profiles with PCDD/Fs and assess exposure risk to human health[25,29]. An earlier study conducted in 1994 showed similar I-TEQ values of 31.5 ng/g and 35.4 ng/g (lipid weight basis) in human adipose tissues for PCDD/Fs and PCBs, which indicates a similar level of toxicity for PCDD/F and PCBs to human health respectively. In 2000, the levels of PCDD/Fs and PCBs with I-TEQ values were also comparable, but reduced to the levels of 11.9 ng/g and 15.3 ng/g (lipid weight basis) in human adipose tissues from Japan, which indicates less toxic effect on human health over six years. A separate study conducted from 1982-1989 on human adipose tissues obtained from donors in Atlanta, Georgia showed that dioxin-like PCB congeners in adipose tissue varied greatly between samples whereas PCDD and PCDF profiles were more consistent. Age was positively correlated with the concentrations of PCDD and PCBs in adipose tissue[33].

4. Polybrominated Diphenyl Ethers (PBDEs)

Rarely has there been so much discussion about a group of chemical products as the flame retardants (www.bsef.com). PBDEs are a specific group of flame retardants widely used in plastics, textiles, electronic circuitry and other materials to suppress combustion. Three industrial formulations of PBDEs are used widely as flame retardants (BSEF, 2001). Deca-BDE (consisting almost completely of BDE-209) is used mainly in thermoplastics and textiles; Octa-BDE (a mixture of hexa- to octa-BDE congeners) is used in acrylonitrile/butadiene/styrene plastics; and penta-BDE (a mixture of tetra-and penta-BDE congeners) is used mainly in polyurethane foam. In 2001, total worldwide demand of these three PBDEs reached 67 metric tons. Production of all PBDEs has escalated greatly over the last 20 years, and this has been accompanied by their emergence in a diversity of environmental and biological samples[36]. To date, the toxicology of PBDEs is still under investigation, but it has been established that PBDEs are environmentally persistent, bioaccumulative and toxic to human health[37]. The critical effects of penta-BDE are associated with neurobehavioral development at low doses (from a dose of 0.6mg/kg body weight) and, at higher doses, effects on thyroid hormone levels in rats and mice[44]. Due to concerns over rising levels of contamination in human breast milk and wildlife, as well as associations with thyroid dysfunction, penta- and octa-BDE were banned in the EU in mid-2003 (www.bsef.com). Deca-BDE has been classified as a possible human carcinogen and is known to interfere with brain development in rats[45].

PBDE congeners 2,2',4,4'-tetraBDE (BDE-47), 2,2',4,4',5-pentaBDE (BDE-99), and 2,2',4,4',5,5'-

hexaBDE (BDE-153) were detected at high levels in human adipose tissue samples from Sweden in 1999 and, combined, constituted 87-96% and 84-94% of the total sum of PBDEs in liver and adipose tissue, respectively[40]. BDE-47 has been identified as the predominant congener in the adipose tissue of contemporary California women, where concentrations ranged between 5.2 and 196 ng/g of lipid, with a median value of 28.9 ng/g of lipid among 32 breast adipose tissue samples analyzed in 1995. These levels were between 3 and 10 times higher than those measured in similar samples from Japan, Sweden, Germany, and Norway[32, 33, 34, 39, 41]. The authors speculated that high levels may be due to California's flammability regulations which demand the use of treated polyurethane foam and textiles used in furnishings. A relatively high concentration of PBDE-47 in the adipose tissue of a 74-year-old Swedish male was reported at 8.8 ng/g lipid on a weight basis in 1994[38], which indicates the strong propensity of PBDE to bioaccumulation in the human body over the lifetime of an individual. In four Swedish reports, the sum concentrations of PBDEs did not show any clear change in adipose tissue samples between 1997 and 2001[35,38,39,40].

5. Preliminary data on POPs levels in female adipose tissue in Singapore

Approximately ten grams of adipose tissues were procured via biopsy from sixteen volunteer expectant mothers admitted to the National University Hospital, Singapore for cesarean section delivery. The volunteers ranged in age from 22 to 41 years, where all donors were in a healthy condition without any prenatal complications. The study was approved by the Institutional Ethics Review Board, Singapore and informed written consents were obtained from the women antenatally.

This study represents the first of its kind in Singapore, where similar data for the wider region of Southeast Asia are sparse. The sample preparation and analytical procedure is outlined in a previous manuscript[56]. Data are presented in Tables 1 & 2 for comparison to similar data obtained from other countries. DDT and its derivatives were the dominant POPs present in adipose tissue with a concentration range from 7 to 2928ng/g, and a mean of 977ng/g on a lipid weight basis which is comparable to the value from Finland[7] and Poland 2001[13]. A total of 41 congeners of PCBs were analyzed and ranged in concentration from 7 to 71ng/g with a mean of 34ng/g on lipid weight basis. These levels are much lower than reported in Japan in 1980[51,52] and Belgium in 2000[34]. A sum of five PBDE congeners (PBDE-47, 99, 100, 153, 154) were present at a concentration range of 0.5 to 12.3ng/g, and a mean of 3.6ng/g (lipid weight basis) in adipose tissue which is comparable to values obtained from Belgium citizens in 2000[34].

6. Conclusions

Among the pesiticides, OCPs of the DDT group are the most prevalent contaminants in human adipose tissues worldwide. Typically, levels of PBDEs are still the lowest amongst the major groups of POPs prevailing in the environmet, but levels in biological matrices, including human adipose tissues are rising around the world due to large scale production and usage of these compounds. Clearly, it will remain a challenge for the scientific community to continue monitor and evaluate the risk of current and emerging POPs in our environment, and ultimately to eliminate the most harmful compounds and identify safer alternatives. Historical data on the known effects of POPs on the wildlife and human health clearly advocates the adoption of a precautionary principle prior to the intentional or incidental emission of novel POPs into the environment. Analysis of local samples in Singapore, has demonstrated the prevalence of POPs in adipose tissues. Although concentrations are comparable to those observed elsewhere, longer term monitoring of a larger cross section of the popluation is required to establish temporal trends and potential risks to human health.

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