Why are there Different Age Related Trends for Different Chemicals?

Jochen F. MUELLER and Leisa-Maree L. TOMS

National Research Centre for Environmental Toxicology, The University of Queensland, 39 Kessels Road, Coopers Plains, QLD 4108, Australia

(Received 28 February 2010; accepted 29 May 2010)

Abstract—Persistent organic pollutants (POPs) such as dioxins, PCBs, persistent organochlorine pesticides (OCPs) and polybrominated diphenyl ethers (PBDEs) as well as perfluorinated compounds (PFCs) and triclosan are ubiquitous in the human population. In Australia, we have pooled and subsequently analysed over 10 000 human serum samples for the determination of these chemicals by age group (0–0.5; 0.6–1; 1.1–1.5; 1.6–2; 2.1–2.5; 2.6–3; 3.1–3.5; 3.6–4; 4.1–6; 6.1–9; 9.1–12; 12.1–15; 16–30; 31–45; 46–60 and >60 years) and gender. The results of this analysis were then used to assess the trends of these different chemicals as a function of age, gender and to a lesser extent region. Our data demonstrate clear chemical specific age trends. In particular we demonstrate that for the traditional POPs there is an increase in body burden with age whereas the opposite is true for chemicals such as PBDEs. For PFCs we find chemical specific age trends that vary from compound to compound. For triclosan we show that no apparent age trend is observable. The results of the study and its implications to the collection and archiving of samples for retrospective analysis are discussed.

Keywords: age trends, dioxins, polybrominated diphenyl ethers, perfluorinated compounds, Australia

INTRODUCTION

Humans are exposed to a wide range of chemicals throughout their life. A range of chemicals including those that are covered by the Stockholm Treaty on Persistent Organic Pollutants have a tendency to accumulate in biota and humans due to their physico-chemical properties (i.e. lipophilicity and persistence). Studies have shown that concentrations may vary substantially between individuals for a given chemical related to the exposure of the individuals to that given chemical (Ryan et al., 2002; Schecter et al., 2003). A key question is whether general trends can be found between populations or within a given population that allows us to obtain a better understanding of exposure and exposure pathways. The aim of this study was to determine whether or not trends in chemical concentration by age, gender and region of residence are apparent for dioxins and
dioxin-like PCBs, persistent organochlorine pesticides (OCPs), polybrominated diphenyl ethers (PBDEs), perfluorinated chemicals (PFCs) and triclosan.

MATERIALS AND METHODS

Samples

De-identified human blood serum samples were collected and pooled in 2002/03, 2004/05 and 2006/07. The serum samples were obtained from Sullivan and Nicolaides Pathology from surplus stored sera that had been collected as part of routine pathology testing. The first two collection periods used samples from 5 regions of Australia while the last collection was from south east Queensland, Australia, only. Participants' age and gender, and the date of blood collection were available for each sample; gender was not known for the cord blood samples. Prior to pooling, all samples were stratified according to age and gender. The age groups for the 2002/03 and 2004/05 were 0–4 (2004/05); <16 (2002/03); 5–15 (2004/05); 16 to 30 (2002/03 and 2004/05); 31 to 45 (2002/03 and 2004/05); 46 to 60 (2002/03 and 2004/05); and >60 (2002/03 and 2004/05) and for 2006/07 were: cord blood; 0–0.5; 0.6–1; 1.1–1.5; 1.6–2; 2.1–2.5; 2.6–3; 3.1–3.5; 3.6–4; 4.1–6; 6.1–9; 9.1–12; 12.1–15; 16–30; 31–45; 46–60 and >60 years. The mean age of each pool was calculated by taking the mean of all ages from the donors used in that pool.

Where possible, 1 mL of serum was used from each sample. Due to the small volume of blood obtained from infants and young children, less than 1 mL and fewer than 100 (2002/03 and 2004/05) and 30 samples (2006/07) were used in some pools. All samples in a given pool contributed the same volume. Disposable polyethylene pipettes (non sterile) were used to aliquot the serum, originally stored in polypropylene tubes, into a glass jar. After homogenization, subsamples were then aliquoted and stored at –20°C until analysis. Further details of sampling are available in the following papers (Harden et al., 2007; Allmyr et al., 2008; Toms et al., 2008, 2009b).

The original ethics approval for blood collection was granted on 20 September 2002 by The University of Queensland Medical Research Ethics Committee with an amendment to collect samples from the age brackets cited above, approved on 28 June 2006.

Analysis

Samples were analysed at the following laboratories and the analysis has been described in the references listed: dioxins and dioxin-like PCBs - Eurofins, Hamburg, Germany (Harden et al., 2007); OCPs and PBDEs - Center for Disease Control, Atlanta, USA (Toms et al., 2009a); PFCs - University of Orebro, Sweden and Center for Disease Control, Atlanta, USA (Karrman et al., 2006; Toms et al., 2009b); and triclosan - Department of Applied Environmental Science (ITM), Stockholm University, Sweden (Allmyr et al., 2008).
RESULTS AND DISCUSSION

The study allowed an evaluation of baseline concentrations of a broad range of environmental pollutants in the Australian human population. Below we discuss observable trends in the Australian population for a range of chemicals.

**Dioxins and dioxin-like PCBs**

A range of 2,3,7,8 chlorine substituted polychlorinated dibenzodioxins and dibenzofurans as well as dioxin-like PCBs were detectable consistently in most or all samples collected from the Australian population. 1,2,3,7,8 PeCDD is typically the chemical that contributes the highest to the overall toxicity equivalency concentration (approximately 35%). A relationship of increasing dioxin-like chemical concentrations with increasing age was observed in human serum pools (Harden et al., 2007) which is in good agreement with other studies (e.g. Päpke et al., 1996; Buckland et al., 2001). For a given age group, no differences were observed in concentrations between genders and/or between samples collected in different geographical regions of Australia.

**Persistent organochlorine pesticides**

Determination of OCPs in human milk showed no consistent regional trends and did not allow assessment of age trends based on the close age ranges of primipara mothers (Mueller et al., 2008). In human blood pools, concentrations increased with age and were similar between genders (data unpublished).

**PBDEs**

PBDE concentrations varied by age with concentrations increasing from birth to a peak between 2 and 5 years and then decreased until concentrations stabilized at around 30 years. BDE-47 was typically found at the highest concentrations. A trend of higher PBDE concentrations in males than in females was observable; however, this difference was only close to statistically significant for BDE-153 ($p = 0.05$). No regional differences were observed (Toms et al., 2009a).

**PFCs**

PFC concentrations appear to increase from birth with the maximum concentrations of all PFCs detected in children <15 years with the exception of PFOS. For all PFCs, the concentrations appear to be reasonably stable after 10 years of age with the exception of PFOS which increased steadily until >60 years. Concentrations of PFOA, PFOS, PFNA and PFHxS appeared to be higher in males than in females across all ages although the difference was only statistically significant for PFOS ($p = 0.002$) using ANOVA analysis (SPSS 16.0). No substantial differences were found between rural and urban regions (Toms et al., 2009b).
Triclosan

Determination of triclosan in human milk in Australia did not allow an assessment of age trends based on the close age range of first time mothers. However, an investigation of blood pools showed that age and region trends were not apparent, however, concentrations were higher in males than in females (Allmyr et al., 2008).

Reasons for age trends

The age trends for various chemicals are illustrated in Fig. 1. Concentrations of the OCPs - HCB, DDE and dioxins and dioxin-like PCBs increased with age. PFOS concentrations also showed a “positive” age trend (i.e. increase with age) although the trend is different with a rapid increase in the first 5 years. In contrast, the age trend for PBDEs was very different with a rapid increase in the first 1–2 years, with a plateau between 2–5 years and then a decrease with increasing age. For triclosan, no age trend was obvious. The reasons for differences in
Why are there Different Age Related Trends for Different Chemicals? 123

concentrations by age between the chemicals are related to history of exposure, half-lives, sources and exposure pathways:

• History of exposure. PBDE, PFC and triclosan exposure has been relatively recent (last ~30 years) with the older population receiving less over a lifetime than the younger population. In contrast, exposure to HCB, DDE and dioxins has decreased over time with the older populations receiving more than the younger populations.

• Half-lives. For PBDEs and PFCs these are substantially shorter in comparison to, for example, dioxins (Sjödin et al., 2003; Geyer et al., 2004; Olsen et al., 2007). Hence the body reaches a steady state for PBDEs and PFCs faster, the effect of past elevated PBDE and PFC exposure is observable for a shorter period and current PBDE and PFC sera concentrations are more likely to reflect a relatively recent exposure.

• Sources. The sources of “traditional” POPs such as dioxins and dioxins-like PCBs originate from the outdoor environment while the opposite is likely to be the case for PBDEs and PFCs. Products treated with PBDEs become sources in the indoor environment, where they are primarily used and PBDEs have been detected in indoor air and dust. Infants and young children are subjected to higher exposure indoors, in particular dust, as they are in close contact with the floor and exhibit high hand-to-mouth contact. Furthermore, age trends for PBDEs may be related to the use of PBDEs as an integral component of child specific items including car seats and bedding, hence potential sources for infants. Sources of PBDEs, PFCs and triclosan may be specific to an individual’s lifestyle rather than the broader community.

• Exposure pathways. Exposure to PBDEs and PFCs is still under debate but food, human milk and dust are all thought to be important whereas for “traditional” POPs, food is the major pathway. Triclosan exposure is related to personal preference of hygiène products which may or may not contain triclosan.

CONCLUSIONS

Overall, 10719 human samples were used for analysis of environmental pollutants in the Australian population including samples of blood collected from 2002–2003, 2004–2005 and 2006–2007. The results demonstrate differences in age trends between different pollutants which is explained by the persistence of the chemicals of interest and the history of exposure. The exposure source and pathway may also (indirectly) influence pollutant age trends since they may cause chemical specific inhomogeneity in exposure (i.e. higher exposure for example, of children through increased uptake of dust).

The work we present here has wider applications to the implication of pollutant monitoring strategies and the archiving of samples. While one needs to be aware of the limitations associated with the pooling of samples, there are a number of advantages. Among those is that the use of pooled samples allows cost-effective evaluation of population wide human body burden for chemicals that are bioaccumulative. Furthermore pooling samples provides a means to obtain larger sampling volumes which can be very beneficial for trace pollutant work where
some analytes/methods require larger sample volumes and where studies aim to retain relevant sample volumes for a sample archive and retrospective studies. Providing that studies are carefully designed and relevant QCQA steps are included (including archiving of reference material) archiving and retrospective analysis of archived samples is likely to provide an inexpensive vehicle to assess pollutant trends in a given population.

Acknowledgments—We thank the staff at Sullivan and Nicolaides Pathology. Entox is a partnership between Queensland Health and The University of Queensland.

REFERENCES


