Environmental Research 142 (2015) 387–406

Contents lists available at ScienceDirect

Environmental Research

journal homepage: www.elsevier.com/locate/envres

Tooth matrix analysis for biomonitoring of organic chemical exposure: Current status, challenges, and opportunities

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ARTICLE INFO

Article history:
Received 18 March 2015
Received in revised form
21 June 2015
Accepted 25 June 2015
Available online 25 July 2015

Keywords:
Biomarker
Chromatography
Dentine
Mass spectrometry
Organics
Prenatal
Teeth

ABSTRACT

Epidemiological evidence supports associations between prenatal exposure to environmental organic chemicals and childhood health impairments. Unlike the common choice of biological matrices such as urine and blood that can be limited by short half-lives for some chemicals, teeth provide a stable repository for chemicals with half-life in the order of decades. Given the potential of the tooth bio-matrix to study long-term exposures to environmental organic chemicals in human biomonitoring programs, it is important to be aware of possible pitfalls and potential opportunities to improve on the current analytical method for tooth organics analysis. We critically review previous results of studies of this topic. The major drawbacks and challenges in currently practiced concepts and analytical methods in utilizing tooth bio-matrix are (i) no consideration of external (from outer surface) or internal contamination (from micro-odontoblast processes), (ii) the misleading assumption that whole ground teeth represent prenatal exposures (latest formed dentine is lipid rich and therefore would absorb and accumulate more organic chemicals), (iii) reverse causality in exposure assessment due to whole ground teeth, and (iv) teeth are a precious bio-matrix and grinding them raises ethical concerns about appropriate use of a very limited resource in exposure biology and epidemiology studies. These can be overcome by addressing the important limitations and possible improvements with the analytical approach associated at each of the following steps: (i) tooth sample preparation to retain exposure timing, (ii) organics extraction and pre-concentration to detect ultra-trace levels of analytes, (iii) chromatography separation, (iv) mass spectrometric detection to detect multi-class organics simultaneously, and (v) method validation, especially to exclude chance findings. To highlight the proposed improvements we present findings from a pilot study that utilizes tooth matrix biomarkers to obtain trimester-specific exposure information for a range of organic chemicals.

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1. Introduction: Tooth analysis for the assessment of prenatal exposure to environmental chemicals

Perinatal exposures to environmental chemicals have been linked to a multitude of health outcomes, including impaired neurodevelopment (Bellinger, 2013) and metabolic syndrome (Behl et al., 2013; La Merrill and Birnbaum, 2011; Wang et al., 2014) in children. Exposure to environmental chemicals in utero has gained much attention because of the heightened susceptibility of developing organs, particularly the brain (Adams et al., 2000; Grandjean and Landrigan, 2006, 2014; Rice and Barone, 2000). The concept of 'critical windows of susceptibility' supports the rationale that disruption of developmental processes during early life would change the trajectory of long-term health status (Cory-Slechta et al., 2008; Grandjean and Landrigan, 2006, 2014). The fetal organs are highly susceptible to environmental chemical exposures because of (i) greater absorption of chemicals due to immature chemical transport mechanisms, (ii) underdeveloped biological mechanisms to detoxify chemicals, (iii) greater exposure due to greater intake (pound for pound) compared to adults, and (iv) heightened susceptibility to even small amounts of chemicals which alters subsequent growth and development (Grandjean and Landrigan, 2006, 2014; Landrigan et al., 2004).

This review will focus on risk associated with early life exposure to organic toxicants and we direct the reader to several other reviews for information on risks associated with metal

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http://dx.doi.org/10.1016/j.envres.2015.06.032
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exposures (e.g. arsenic, cadmium, and manganese (Rodriguez-Barranco et al., 2013); lead (Guilarte et al., 2012); mercury (Gundacker et al., 2010; Yoshimasu et al., 2014)). The risks from prenatal and early childhood exposure are particularly significant for certain organic toxicants including polybrominated diphenyl ethers (PBDEs), environmental tobacco smoke, and organophosphate (OP) and organochlorine (OC) pesticides. PBDEs can be transferred to the developing fetus across the placenta and to children via breast milk and fat-containing food (Lorber, 2008). A prior U.S. study has shown almost identical PBDE concentrations in maternal blood collected at delivery and in cord blood, suggesting that the placental barrier offers limited protection to the fetus (Mazdai et al., 2003). Neurodevelopment outcomes in children from prenatal exposures to environmental organics are a widely studied research topic. Example studies are for (i) PBDE: (Chen et al., 2014; Eskenazi et al., 2013; Gascon et al., 2011; Herbstman et al., 2010); (ii) bisphenol A: (Braun et al., 2009; Harley et al., 2013; Perera et al., 2012; Yolton et al., 2011); and (iii) phthalates: (Kobrosly et al., 2014; Polanska et al., 2014; Tellez-Rojo et al., 2013; Yolton et al., 2011).

Similar to PBDEs, OC pesticides such as dichlorodiphenyl trichloroethane (DDT) and its metabolites are higher in fetal circulation than maternal matrices. For example, a study of 90 mother/infant pairs from Mexico with no known occupational exposure found that in most cases the concentrations of the pesticide residues in cord blood were higher than maternal blood. Of the OCs detected, p,p′- dichlorodiphenyl dichloroethylene was the most concentrated (lipid-adjusted means, 4.4 ppm maternal; 4.7 ppm cord blood), followed by p,p′-DDT (1.8 ppm maternal, 2.8 ppm cord blood), and o,p′-DDT (0.30 ppm maternal, 0.35 ppm cord blood) (Waliszewski et al., 2001). Studies have shown that OP pesticides readily cross the placental barrier to reach the fetus (Rauh et al., 2006; Whyatt et al., 2009) and dialkyl phosphate metabolites have been detected in amniotic fluid (Bradman et al., 2003). Emerging evidence suggests that prenatal, but not postnatal OP exposure is associated with Attention Deficit Hyperactivity Disorder (Eskenazi et al., 2007; Marks et al., 2010) and impaired cognitive development (Bouchard et al., 2011; Harari et al., 2010; Rauh et al., 2006). Impacts on neurodevelopment in children from prenatal exposures to pesticides are widely recognized and reviewed elsewhere (Ding and Bao, 2014; Muñoz-Quezada et al., 2013; Polanska et al., 2013). However, direct fetal measurements of the intensity and timing of intrauterine exposure have not been undertaken for OP and OC pesticides, primarily due to the absence of a direct fetal biomarker of environmental chemical exposure.

Tobacco smoke contains many toxicants and one recent study found higher levels of tobacco smoke markers (cotinine and nicotine) in the first urine of newborns of actively smoking mothers than offspring of non-smokers (Florek et al., 2011). Potential health impacts in infants and children from their mothers smoking during pregnancy are multifarious (a critical review by Bruin et al., 2010) and examples are (i) low birth weight (Ricketts et al., 2005) or childhood overweight (Moller et al., 2014), (ii) stillbirth risk and congenital malformation (meta-analysis report by Leonardi-Bee et al., 2011), (iii) wheezing and asthma incidence (meta-analysis study by Burke et al., 2012) and (iv) neurodevelopment impairment (reviewed by Herrmann et al., 2008).

Prenatal exposure assessment is one of the greatest challenges in developmental epidemiologic studies. This is limited by (i) the lack of biomarkers that directly measure fetal (vs. maternal) exposure at specific intrauterine developmental periods; (ii) the inability to objectively reconstruct past exposure at specific life stages outside the use of questionnaires; and (iii) the expense and time needed to conduct prospective studies of prenatal exposure and health. No contemporary biomarker such as chemicals measured in cord blood, placenta, and maternal markers during pregnancy, or infant samples can provide a direct measure of exposure *timing* at different times of intrauterine development in large epidemiologic studies. To address these issues studies have attempted to use primary teeth to provide a direct measure of the timing and intensity of exposure to metals from approximately the 14th gestational week to birth and into early childhood (e.g. lead (Arora et al., 2014; Gulson et al., 1997; Needleman and Shapiro, 1974; Needleman et al., 1974; Rabinowitz et al., 1976; Uryu et al., 2003), manganese (Arora et al., 2011, 2012; Ericson et al., 2001; Gunier et al., 2013), and barium and strontium (Austin et al., 2013; Humphrey et al., 2008).

Teeth are uniquely positioned to capture childhood chemical exposures in continuum starting from early (second trimester in utero) to progressive life stages (until deciduous teeth are shed around 6–12 year's age). Unlike the common choice of bio-matrices such as urine and blood that can be limited by short half-lives for some chemicals, exposure signals in teeth remain stable enabling a longer detection window (Gulson et al., 1997). Although hair also provides longer detection windows, it is limited to a monthly time scale (Gareri and Koren, 2010; Hinners et al., 2012). The human tooth as a bio-matrix of past exposures to metals was detailed in a recent review, which also discussed tooth components and tooth development process (Arora and Austin, 2013). In brief, enamel and dentine are the primary mineralized components of a tooth crown that differ significantly in their mineralization pattern. This characteristic determines their usefulness in capturing and measuring chemical exposures.

Previous applications of tooth-chemical concentrations in epidemiologic studies did not provide detailed temporal (i.e. weekly, monthly or trimester-specific) information on prenatal and early childhood exposure to metals. In early studies, whole teeth were digested and metal toxicants concentrations reported on a whole-tooth basis (reviewed by Arora and Austin, 2013). Although teeth have been used for exposure assessment to inorganic chemicals since the 1960s (Altshuller et al., 1962; Arora and Austin, 2013; Needleman et al., 1974; Needleman and Shapiro, 1974), the use of teeth for assessing exposures to organic chemicals is in the early 2000s (Table 1). Moreover, the use of tooth chemical biomarkers for detailed assessment of exposure timing is a relatively recent development, unlike the use of other conventional biomarkers in blood or urine that have a long tradition. These studies cannot give exposure information on a finely resolved temporal scale for organic chemicals, which prevent the study of critical windows of vulnerability. Grinding whole teeth or utilizing large fragments of teeth ignores the complex developmental physiology and microstructure of teeth and can lead to gross exposure misclassification, as has been shown in an important study by Rabinowitz et al. (1993), which will be discussed in Section 3.The goal of this review is to present a comprehensive background on the current state-of-the-art of tooth organics analysis, its limitations, challenges and future opportunities. We discuss the different parameters affecting each of the following steps: (i) tooth collection, (ii) decontamination and pre-treatment, (iii) analytes extraction, clean-up and pre-concentration, and (iv) separation, detection and quantification using chromatography and mass spectrometry tools. We summarize the novel application of tooth bio-matrix analysis for reconstructing prenatal exposures to environmental chemicals and the practical applications. The review concludes with emphasizing the need to understand which chemical classes may be affecting specific critical windows of development related to childhood health disorders so that the source of exposure may be identified and thereby provide a means of intervention to reduce exposure and the consequent health effects.

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