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Secondhand smoke exposure at home: Assessment by biomarkers and airborne markers

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ABSTRACT

Objective: We assessed and characterized the relationship among biomarkers of secondhand smoke (SHS) exposure in non-smokers according to their exposure at home as measured by airborne markers.
Methods: We conducted an observational study on exposure to SHS at home using airborne markers (nicotine and benzene) and biomarkers from the non-smokers living in these homes. We selected 49 non-smoking volunteers from different homes: 25 non-smokers living with at least one smoker and 24 non-smokers living in smoke-free homes. We installed two passive devices to measure nicotine and benzene concentrations in the main room of the house (i.e., the living room). One week later, the researcher returned to the volunteer's home to collect the two devices, obtain saliva and urine samples, and administer a SHS questionnaire.
Results: Salivary and urinary cotinine concentrations highly correlated with air nicotine concentrations measured at the volunteers' homes ($r_{sp}=0.738$ and $r_{sp}=0.679$, respectively). The concentrations of airborne markers of SHS and biomarkers in non-smokers increased with increasing self-reported intensity and duration of SHS exposure at home during the previous week ($p<0.05$). The multivariable regression model showed a significant association with nicotine in air at home ($\beta=0.126$, $p=0.002$ for saliva and $\beta=0.115$, $p=0.010$ for urine).
Conclusions: Our findings suggest that, even in countries with comprehensive smoke-free legislation, exposure to SHS at home continues to be the main source of exposure for non-smokers who live in non-smoke-free homes. Therefore, public health policies should promote smoke-free homes.

1. Introduction

Secondhand smoke (SHS) has been classified as a type I carcinogen in humans by the International Agency for Research on Cancer (IARC) (IARC, 2004). SHS exposure also increases the risk of cardiovascular and respiratory diseases (IARC, 2004; US Department of Health and Human Services, 2006) and is responsible for approximately 630,000 deaths per year worldwide (Oberg et al., 2011).

Growing scientific evidence of the risk of diseases among non-smokers exposed to SHS has led several countries to implement smoke-free regulations in workplaces and public places, including hospitality venues (IARC Working Group, 2009). The implementation of smoke-free regulations has been accompanied by a decrease

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in SHS exposure among non-smokers in the general population (Haw and Gruer, 2007; Galan et al., 2007; Martinez-Sanchez et al., 2010) and specific groups (Allwright et al., 2005; Fernandez et al., 2009; Callinan et al., 2010). Furthermore, this decrease in SHS has reduced the incidence of diseases related to SHS exposure (IARC Working Group, 2009; Tan and Glantz, 2012). However, SHS exposure in private venues, particularly at home, continues to be a priority in tobacco control and public health research because this venue is not regulated by smoke-free legislation, and SHS exposure at home has an economic impact on healthcare and mortality (Max et al., 2012, 2014). In addition, private transportation (e.g., cars) and homes are the main settings of SHS exposure among children (Ashley and Ferrence, 1998; Jarvis et al., 2000; Longman and Passey, 2013; Nabi-Burza et al., 2012).

SHS is a complex mix of more than 4,000 substances, including toxic and irritant compounds and carcinogens (IARC, 2004). Among these substances, nicotine is usually used as an environmental marker of SHS in air (Lopez and Nebot, 2003). Cotinine, the main metabolite of nicotine, can be measured in body fluids, including serum, saliva, and urine, and is used as a biomarker of SHS exposure because its half-life is higher in these fluids than that of nicotine (Avila-Tang et al., 2013). Although some evidence indicates a correlation between airborne and biological markers of SHS exposure (Repace et al., 2006), few studies have used a combination of environmental and biological markers to assess SHS exposure in specific settings (Callinan et al., 2010). Further research is needed to describe how these markers are related (Kim et al., 2004; Butz et al., 2011; Henderson et al., 1989; Jones et al., 2014).

Therefore, the objectives of this study were to assess the relationship between airborne markers of exposure to SHS at home (air nicotine and benzene) and biomarkers of such exposure (cotinine in saliva and urine) among non-smokers who lived in these homes and to characterize salivary and urinary cotinine concentrations among non-smokers according to their exposure at home as measured by air nicotine and self-reported SHS exposure in different settings.

2. Material and methods

The fieldwork was conducted between November 2011 and February 2012. We selected 49 non-smoker volunteers from different homes: 25 non-smokers who lived with at least one smoker and 24 non-smokers who lived in smoke-free homes. The volunteers were recruited from among the personal contacts of the researchers. After the initial contact, a member of the research team went to the volunteer's home to explain the objective and procedure of the study, provide a presentation letter, and obtained written informed consent. During that visit, the researcher installed two passive devices to measure nicotine and benzene concentration in the main room of the house (usually the living room). One week later, the researcher returned to the volunteer's home to collect both devices, obtain saliva and urine samples, and administer a questionnaire. The research and ethics committee of Bellvitge University Hospital approved the study protocol, including the informed consent form.

2.1. Airborne markers of SHS exposure

We used passive sampling devices for the airborne nicotine measurements. Samplers contained a 37-mm diameter filter treated with sodium bisulfate. The sampling devices were installed following a standard protocol; they had to hang freely in air, not in an area where air does not circulate such as a corner, under a shelf, or buried in curtains, and they could not be placed within one meter of an area where someone regularly smokes.

Nicotine was extracted from the filter and analyzed by gas chromatography with detection by mass spectrometry (GC/MS) at the Laboratory of the Public Health Agency of Barcelona. The limit of quantification for nicotine was 5 ng, equivalent to 0.02 $\mu g/m^3$ per exposure time of one week as in previous studies (Fernandez et al., 2008; Sureda et al., 2012; Nebot et al., 2009). The nicotine concentration was determined by dividing the amount of nicotine collected by the filter by the flow rate (24×10^{-6} m^3/min) and allowing for the time the filter had been exposed over the last week. Samples with nicotine concentrations below the quantification limit were assigned a value of 0.01 $\mu g/m^3$ (half the limit of quantification).

We also used a diffusive passive sampler (Radiello®) to measure airborne benzene exposure. This sampling system is made up of a cylindrical adsorbing cartridge housed coaxially inside a cylindrical diffusive body of polycarbonate and microporous polyethylene. Benzene was extracted from the cylinder and analyzed by GC/MS at the Laboratory of the Public Health Agency of Barcelona with a limit of quantification of 50 ng, equivalent to 0.18 $\mu g/m^3$ per week. The benzene concentration was determined by dividing the amount of benzene collected by the cylinder by the sampling rate (27.8×10^{-6} m^3/min) and allowing for the time the filter had been exposed over the last week.

2.2. Biomarkers of SHS exposure

We obtained saliva and urine samples for cotinine analysis. Participants collected roughly 20 ml of urine the same day as the second visit using a container provided by the researcher at the first visit. For the saliva sample, participants were asked to rinse their mouths and then suck a lemon candy (Smint®) to stimulate saliva production. First, they were asked to spit out a small amount of saliva, and then they were asked to provide roughly 9 ml of saliva by spitting it into a funnel placed in a test tube. Later, the saliva sample was separated into 3 ml aliquots in the laboratory in case further analyses were required. The saliva and urine samples were frozen at -80 °C for storage (Fernandez et al., 2009; Martinez-Sanchez et al., 2009a).

The frozen samples were sent to the Bioanalysis Research Group of the IMIM (Hospital del Mar Medical Research Institute) in Barcelona. Cotinine was measured by liquid chromatography coupled to tandem mass spectrometry with multiple reaction monitoring (LC/MS/MS). The limit of quantification was 0.10 ng/ml and the limit of detection was 0.03 ng/ml (quantification error $< 15\%$) for both salivary and urinary cotinine.

2.3. Self-reported exposure to SHS

We used a face-to-face questionnaire (Martinez-Sanchez et al., 2009a) to obtain participants'self-reported exposure to SHS during the week the devices were installed in their home. Dichotomous questions (yes/no) were used for the following settings: home, work/education venues, leisure time, and transportation. We also obtained information on the intensity and duration of the exposure at home, work, education venues, and during leisure time.

To measure SHS exposure intensity and duration at home, we asked three questions: “During the past week, how many persons per day usually smoked inside your home?” (recoded as 0, 1, or ≥ 2 persons per day); “During the past week, how many cigarettes per day have been smoked in your presence in the room where the devices were installed?” (recoded as 0, 1, 2–6, or ≥ 7 cigarettes per day); and “During the past week, how many hours per day have you been exposed to tobacco smoke in the room where the devices were installed?” (recoded as 0, < 2, or ≥ 2 h per day). The last two questions were asked for both a typical working day and a typical non-working day.

SHS exposure intensity at work was obtained using two questions: “During the past week, has anybody smoked in close proximity to you at work?” (recoded as 0, 1, or ≥ 2 persons per day); and “During the last week, how many hours per day do you think you have been exposed to tobacco smoke at your work?” (recoded as 0, < 1, or ≥ 1 h per day). The duration of SHS exposure in education venues was obtained using one question: “During the last week, how many hours per day do you think you have been exposed to tobacco smoke at your educational venue?” (recoded as 0, < 1, or ≥ 1 h per day). Finally, the duration of SHS exposure during leisure time was obtained using one question: “During the last week, how much time per day did you spend in a place with tobacco smoke during your leisure time (not at home or at work)?” (recoded as 0 h, < 2 h, or ≥ 2 h per day). The leisure time question was asked for both a typical working day and a typical non-working day.

2.4. Data analysis

We described the concentrations of airborne markers (nicotine and benzene) and biomarkers (cotinine in saliva and urine) using medians and interquartile ranges (IQRs). We described the concentration of airborne markers according to the self-reported intensity and duration of exposure at home and the characteristics of the house and room where the devices were installed (size, number of rooms, and ventilation of the room – opening the windows after or before smoking). We also described the concentration of biomarkers according to the self-reported SHS exposure in different settings, intensity and duration of SHS exposure, and socio-demographic characteristics (sex, age, and body mass index). We compared the concentrations of airborne markers and biomarkers using the Mann Whitney U-test for two independent samples and the Kruskal–Wallis test for more than two groups. We evaluated the linear trend with logarithmic transformation of the concentrations of the airborne markers and biomarkers. We used Spearman's rank correlation coefficient (r_{sp}) to assess the association between airborne markers and