



Trends in exposure to second hand smoke at home among children and nonsmoker adolescents☆



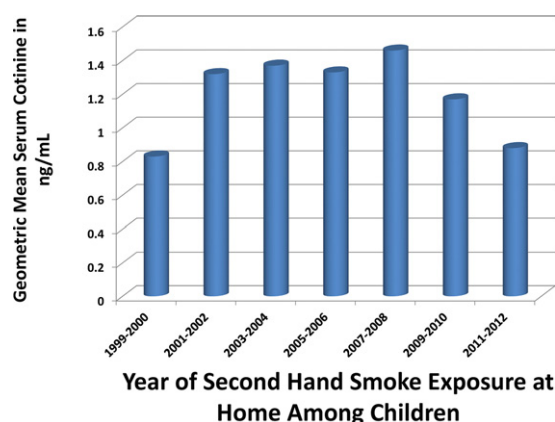
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HIGHLIGHTS

- Adjusted serum cotinine levels increased among children with SHS exposure at home.
- SHS exposure was inversely associated with poverty income ratio.
- Children had higher SHS exposure at home than nonsmoker adolescents.

GRAPHICAL ABSTRACT



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ABSTRACT

Data from National Health and Nutrition Examination Survey (NHANES) for the years 1999–2012 were used to evaluate trends in exposure to second hand smoke (SHS) at home among children aged 3–11 years and nonsmoker adolescents aged 12–19 years. A total of 12,815 children and 10,269 adolescents were included in the analyses. Serum cotinine was used as a biomarker for exposure to SHS at home. Regression models with log₁₀ transformed values of serum cotinine as dependent variables and age, race/ethnicity, NHANES survey year, and family poverty income ratio as a surrogate measure of socioeconomic status were used in models for those with and without exposure to SHS at home. In addition, for those with exposure to SHS at home, number of smokers smoking inside home and number of cigarettes smoked at home every day were also used as independent variables. There was a biennial increase of 1.05 ng/L in adjusted serum cotinine levels for children with exposure to SHS at home over the period of 1999–2012. Serum cotinine levels among nonsmoker adolescents with exposure to SHS at home did not change over time. When there was no exposure to SHS at home, there was a statistically significant downward trend for serum cotinine levels for both children and nonsmoker adolescents. Serum cotinine levels attributable to SHS exposure increased with age among nonsmoker adolescents ($p \leq 0.02$) but decreased with age among children ($p < 0.01$). For a unit decrease in family poverty income ratio, SHS exposure as measured by serum cotinine levels (Table 6) increased by 1.18 ng/L among

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children and by 1.30 ng/L among nonsmoker adolescents. In general, observed serum cotinine levels associated with SHS exposure at home were higher for children than they were for nonsmoker adolescents.

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1. Introduction

Association between exposure to second hand smoke (SHS) and increased morbidity and mortality has been comprehensively studied and reported. In 2004, worldwide, exposure to SHS was estimated to cause 603,000 deaths or about 1% worldwide mortality of which 28% deaths occurred in children (Oberge et al., 2011). Disability adjusted life years lost due to SHS exposure was 10.9 million of which 61% were among children (Oberge et al., 2011). In US, the annual cost of lost productivity due to exposure to SHS was estimated at \$6.6 billion (Max et al., 2012) or \$158,000 per death and SHS exposure was associated with 41,000 annual deaths among nonsmoker adults and 900 annual deaths among infants (Max et al., 2012).

Worldwide, 40% children were estimated to be exposed to SHS (Oberge et al., 2011). Based on data from National Health and Nutrition Examination Survey (NHANES) conducted in US, in 2011–2012, 40.6% children aged 3–11 years were exposed to SHS (Homa et al., 2015), a decrease of 37.4% from the 64.9% prevalence observed in 1999–2000. The prevalence of exposure to SHS among adolescents was 63.1% in 1999–2000 and 40.6% in 2011–2012 (Homa et al., 2015). More recently, in Portugal, Vitória et al. (2015a) reported SHS exposure at home among 62.9% children living with parents who smoke and 19.2% of children living with parents who do not smoke. SHS exposure in car was reported among 46.9% children with parents who smoke and among 8.6% children with parents who do not smoke (Vitória et al., 2015b). In a study done among 7th graders in Terrassa, Spain, 41.1% children were exposed to SHS at home, 40% at school, 53.9% in their leisure time, and 33.2% when using public or private transportation (Martín-Pujol et al., 2013). Using data from Center for Disease Control's Pregnancy Risk Assessment Monitoring System (<http://www.cdc.gov/prams/>) for the years 2000–2003, Hawkins and Berkman (2014) reported 9.9% of the infants being in the same room as someone smoking ≥ 1 h/day and in smoking households, infant with 1 and 2+ siblings were 25% and 59% more likely to be exposed to ≥ 1 h of SHS per day than infant with no siblings. There was 59% decline in exposure to SHS during 1996–2006 in England (Sims et al., 2010) and children from more deprived households were more exposed to SHS. In 2007, 45% Irish children were exposed to SHS at home (Kabir et al., 2010). About one in seven children aged 13–14 years was exposed to SHS in cars in Ireland (Kabir et al., 2009).

In a review article, Treyster and Gitterman (2011) found children under the age of 18 years with SHS exposure to have increased risk of SIDS, asthma, altered respiratory function, infection, cardiovascular effects, behavioral problems, sleep difficulties, cancer, and higher likelihood of smoking initiation. Exposure to SHS, among children, has been reported to be associated with increased risk of ear infections (Hawkins and Berkman, 2011), hearing loss (Talaat et al., 2014), and lower and upper respiratory tract infections (Yilmaz et al., 2012). SHS is also associated with increased risk of abortion, low birth weight (LBW), and prematurity (Joya et al., 2014). Among Polish children, between 13% and 27% cases of SIDS and between 3% and 16% cases of LBW were attributable to SHS exposure (Jarosińska et al., 2014).

Based on a review of five case–control and three cross-sectional studies, Jafta et al. (2015) reported odds of childhood tuberculosis disease to be 2.8 and tuberculosis infection to be 1.9 among those exposed to SHS as compared with those not exposed to SHS. Using serum cotinine as a biomarker of SHS exposure, Evlampidou et al. (2015) reported a negative association between SHS exposure among nonsmoker pregnant females and their children's gross motor function at 18 months of age. Relative risk of developing carotid plaque in adulthood among

children with prenatal exposure to SHS was reported to be 1.7 when compared with children with no exposure to SHS (West et al., 2015). Based on a review of 77 studies from 39 countries, SHS exposure was associated with increased risk of mortality from acute lower respiratory infections among children under five years of age (Sonego et al., 2015). Reduced lung function was associated with children's exposure to SHS (Brunst et al., 2012). In a population based cohort of 7 year old children in Toronto, SHS exposure during pregnancy was associated with increased risk of childhood asthma (Simons et al., 2014). Odds of persistent asthma among children aged 1–12 years were more than 17 times higher when father smoked >5 cigarettes/day indoors (Awasthi et al., 2012). Based on an article (Chen et al., 2013) which reviewed 15 studies, prenatal exposure to SHS was found to be negatively associated with neurodevelopmental outcomes among young children; postnatal SHS exposure was associated with poor academic achievement and neurocognitive performance among older children and adolescents; and SHS exposure was associated with increased risk of neurodevelopmental delay. Based on a review of 18 studies, Murray et al. (2012) concluded that SHS exposure at home doubled the risk of meningococcal disease. SHS exposure during pregnancy was associated with statistically significantly higher risk of externalizing behavioral problems among offsprings of the exposed mothers (Liu et al., 2013). SHS exposure was found to be associated with chronic rhinitis among children (Higgins and Reh, 2012). Maternal exposure to SHS was associated with autism in a case–control study conducted among 190 Han children in China (Zhang et al., 2010).

Odds of being exposed to SHS were more than four times higher among children from socioeconomically deprived neighborhoods of Maoris as compared with those living in socioeconomically well-to-do neighborhoods of European New Zealanders (Kelly et al., 2013). SHS exposure at home was associated with low socioeconomic status, large household size, and single-parent households in Australia (Longman and Passey, 2013). SHS exposure was found to be associated with low socioeconomic status including lower household income, lower caregiver education, and public versus private insurance (Collaco et al., 2014).

There are at least two distinct ways that have been used to define exposure to SHS. First, the observed levels of cotinine in serum have been used to define exposure to SHS. For example, Pirkle et al. (2006) defined nonsmokers as having serum cotinine levels <10 ng/mL and all nonsmokers thus defined were considered to be exposed to SHS. This definition practically does not distinguish between nonsmokers and those exposed to SHS and as such, leaves no room for a category called not exposed to SHS. Homa et al. (2015) defined SHS as having serum cotinine levels between 0.05 and 10 ng/mL. This implies those with serum cotinine levels below 0.05 ng/mL could be considered as not being exposed to SHS. Second, exposure to SHS has been defined based on the responses to certain questions asked in surveys and other study questionnaires. If the presence of one or more smokers smoking inside home is reported, all persons including the respondents in that home are considered to be exposed to SHS at home. The extent and intensity of SHS exposure can then be determined based on observed cotinine levels in serum or other matrices like urine, hair etc. This approach seems to have been used by Vitória et al. (2015a); Collaco et al. (2015); Simons et al. (2014); Kelly et al. (2013); Talaat et al. (2014), and Longman and Passey (2013) among others. Jain (2015) defined SHS exposure at home based on the response to the question “Does anybody smokes inside home?” asked in family smoking questionnaires used in NHANES. An affirmative response to this question resulted in being classified as being exposed to SHS at home and a negative response resulted in

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