Spatial analysis of tobacco outlet density on secondhand smoke exposure and asthma health among children in Baltimore City

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ABSTRACT

Rationale Tobacco outlets are concentrated in low-income neighbourhoods; higher tobacco outlet density is associated with increased smoking prevalence. Secondhand smoke (SHS) exposure has significant detrimental effects on childhood asthma. We hypothesised there was an association between higher tobacco outlet density, indoor air pollution and worse childhood asthma.

Methods Baseline data from a home intervention study of 139 children (8–17 years) with asthma in Baltimore City included residential air nicotine monitoring, paired with serum cotinine and asthma control assessment. Participant addresses and tobacco outlets were geocoded and mapped. Multivariable regression modelling was used to describe the relationships between tobacco outlet density, SHS exposure and asthma control.

Results Within a 500 m radius of each participant home, there were on average six tobacco outlets. Each additional tobacco outlet in a 500 m radius was associated with a 12% increase in air nicotine (p<0.01) and an 8% increase in serum cotinine (p=0.01). For every 10-fold increase in air nicotine levels, there was a 0.25-point increase in Asthma Therapy Assessment Questionnaire (ATAQ) score (p=0.01), and for every 10-fold increase in serum cotinine levels, there was a 0.54-point increase in ATAQ score (p=0.05).

Conclusions Increased tobacco outlet density is associated with higher levels of bedroom air nicotine and serum cotinine. Increasing levels of SHS exposure (air nicotine and serum cotinine) are associated with less controlled childhood asthma. In Baltimore City, the health of children with asthma is adversely impacted in neighbourhoods where tobacco outlets are concentrated. The implications of our findings can inform community-level interventions to address these health disparities.

INTRODUCTION

Asthma affects over 5 million children in the USA and is the leading cause of health-related absences, accounting for nearly 14 million missed school days each year.1 The consequences of school absences can be profound and disproportionately impact inner-city communities with high rates of asthma. Asthma prevalence among black children (13.5%) is more than double that of white children, while the asthma mortality rate is nearly 10 times greater.1 Black children with asthma living in poor urban areas are at particularly high risk for disease-associated morbidity.2

In Baltimore City, 20% of children have asthma; nearly three times the national prevalence.3 In the USA, there is a higher prevalence of asthma among those living in poverty.4 5 Reflecting these national patterns, the asthma burden in Baltimore City disproportionately affects lower income neighbourhoods.6 This health disparity is a public health concern that requires greater focus on community-level strategies to improve asthma health.

Secondhand smoke (SHS) exposure is associated with more severe asthma and worse lung function among children with asthma.7–9 As children spend the majority of their time in the indoor environment,10 quantifying SHS exposure in this setting may be therefore particularly impactful. Community sources of ambient air pollution, which can include tobacco smoke, can permeate into indoor spaces and negatively impact indoor air quality.11 12 Community-level factors that influence indoor SHS prevalence and levels, as well as the consequences to children with asthma, are of high importance. Understanding the spatial relationship between community exposures, indoor air quality and asthma outcomes can provide significant insight into the health inequity that is adversely impacting the inner-city populations.

The socioeconomic status (SES) of a neighbour-hood has been shown to affect the health outcomes of individuals.13–15 Among the community factors influenced by neighbourhood SES is tobacco outlet density. Driven by low rental costs, tobacco outlets are concentrated in low-income neighbourhoods, creating a competitive market for the sale of tobacco products.16 17 In Baltimore City, tobacco outlet density has been shown to be associated with maternal smoking during pregnancy and higher infant mortality rates.18

The goals of this study were to investigate the relationship between tobacco outlet density, a community-level factor, and individual SHS exposure, and subsequently to quantify the relationship between SHS exposure and asthma outcomes. Neighbourhoods with higher tobacco outlet density have higher rates of smoking prevalence,19 20 and this can increase indoor air pollution from household members smoking, or alternatively from smoking behaviours among neighbours indoors or outdoors. We had two hypotheses. The first was that higher tobacco outlet density was associated...
with higher SHS exposure, measured by indoor air pollution and serum cotinine levels. The second was higher levels of SHS exposure were associated with more severe and less controlled asthma.

**METHODS**

**Study design**

This study is an analysis of participants enrolled in AIRWEIGHS, a home intervention study of children with asthma. This study used a convenience sample that was baseline data from the AIRWEIGHS trial, prior to randomisation to an air cleaner intervention. Enrollment started in September 2016; participants were recruited through March 2020.

**Study population**

The study population included 139 children with asthma living in Baltimore City. Participants were recruited from prior asthma studies, the emergency department and paediatric pulmonology clinics. Enrolment criteria were: (1) ages 8–17 years, (2) symptomatic asthma, (3) non-smoker (validated with urine cotinine), (4) spending ≥4 nights/week at home, and (5) no plans to move during the study. Participants were excluded on the basis of significant pulmonary or cardiac disease, home not appropriate for indoor air monitoring due to disrepair, enrolment in an environmental asthma trial in the previous 12 months and/ or pregnancy. One hundred and ninety-eight participants were consented, baseline data were collected for 164 participants. Indoor air nicotine levels were available for 160 participants, and of those 139 lived within city limits of Baltimore. Since cross-sectional baseline data were used for this study, there was no loss to follow-up.

**Participant characterisation**

Participant caretakers provided demographic data including participant age, race, sex, caretaker education, insurance status and income, as well as information about the home environment including the number of individuals in the home and presence of smokers in the home. Data regarding the type of dwelling were collected by field staff as part of a home inspection.

Participant caretakers filled out questionnaires regarding asthma severity and control. Asthma severity was determined based on the National Asthma Education and Prevention Program (NAEPP) classification guidelines, which included number of symptomatic days and nights, albuterol use and forced expiratory volume in 1 s. Asthma control was evaluated using the Pediatric/Adolescent Asthma Therapy Assessment Questionnaire (ATAQ), with higher scores indicating less controlled asthma (range 0–7).

**Assessment of SHS exposure**

Air monitoring equipment was deployed by field staff and placed in the participant’s bedroom prior to the air purifier intervention. The average indoor air nicotine level was monitored using a passive badge that was deployed over a 7-day monitoring period and analysed with gas chromatography according to standard methods providing exposure assessment. Airborne nicotine levels below the level of detection (0.036 μg/m³) were assigned a value of half the level of detection (0.018 μg/m³). Serum samples were obtained from a subset of 44 participants (dual enrolled in another study) by venipuncture during a clinic visit and sent for cotinine analysis. Serum cotinine levels below the level of detection (0.1 ng/mL) were assigned the value of half the level of detection (0.05 ng/mL).

**Spatial mapping**

ArcMap in ArcGIS V10.7 software (Environmental Systems Research Institute, Redlands, California) was used to geocode participant addresses and tobacco outlets using the ArcGIS World Geocoding Service as the address locator. Using the Spatial Join tool in ArcGIS, a count of tobacco outlets within a 500 m radius of each participant’s home was obtained. This distance has been used in tobacco outlet density and accessibility studies. The Near tool in ArcGIS was used to measure the distance from each participant’s home to the closest tobacco outlet. A comprehensive list of tobacco outlets in Baltimore City was provided by the Office of the Comptroller of Maryland (last updated 13 January 2020). A tobacco outlet was defined as an establishment with a licence to sell tobacco products. A tobacco product was defined by the Maryland Department of Health as cigarettes, cigars, pipe tobacco, chewing tobacco, snuff and snus, electronic smoking devices and filters, rolling papers, pipes, liquids used in electronic smoking devices (regardless of nicotine content), as well as accessories and components of the listed products. Baltimore City organises neighbourhoods into Community Statistical Areas (CSAs), clusters of communities developed by the Baltimore City Department of Health. In ArcGIS, a CSA map was overlaid on a map of Baltimore City. Using the Spatial Join tool in ArcGIS, the corresponding CSA for each participant address was identified. For mapping, R software (R V4.0.0 and RStudio V1.3.959) was used to jitter participant addresses in order to protect participant privacy. Original participant addresses were used for the purposes of analysis.

**Statistical analysis**

Summary statistics were generated for baseline demographic characteristics, home environment, indoor air nicotine, serum cotinine, tobacco outlet density, asthma severity and control. Income data were missing for 14% of participants, imputation strategies were not used and analysis was run with available data. Serum cotinine measurements were available for 44 participants. Subset analysis of participant characteristics performed using Fisher’s exact and rank-sum tests demonstrated a greater proportion of moderate and severe asthma in this cohort, no other significant differences. Outcome measures included indoor air nicotine levels, serum cotinine levels, asthma control (ATAQ score) and asthma severity (NAEPP category). Values of air nicotine and serum cotinine were log transformed for statistical analysis. Univariable linear regression models of the outcome measures were fit for the following demographic and home environment characteristics: age, race, gender, caregiver education, insurance type, caregiver income, household occupancy, type of dwelling, presence of smokers in the home and tobacco outlet density (online supplemental table 1). Variables with p values <0.05 in univariate models were selected for inclusion in the multivariate models.

Tobit regression models of SHS exposure (air nicotine, serum cotinine) were fit for tobacco outlet density, adjusting for clustering by CSA and possible confounders: age, caregiver education, health insurance, caregiver income, presence of smokers in the home, household occupancy and type of dwelling. Multivariable linear regression models of asthma control (ATAQ score) were fit for air nicotine and serum cotinine, adjusting for possible confounders: age, caregiver education, health insurance, caregiver income, household occupancy and type of dwelling. Multivariable logistic regression was used in an adjusted model to generate ORs for asthma severity (NAEPP category) based on air nicotine and serum cotinine. In these models with direct
measures of SHS exposure (air nicotine, serum cotinine) as the explanatory variable, there was no adjustment for the presence of smokers in the home or clustering. Analyses were performed with Stata V.14.0 (StataCorp, College Station, Texas). Statistical significance was defined as \( p < 0.05 \).

**RESULTS**

The median age of participants was 11 years; majority were black and from low-income households. About 70% reported annual income less than $40,000, the median household occupancy was four individuals and 85% of individuals lived in an apartment or row home. Sixty percent of participants had moderate or severe asthma based on NAEPP guidelines, and the median ATAQ score was 2, indicating that asthma was not well controlled (table 1).

We identified 1331 tobacco outlets in Baltimore City, which included grocery/convenience stores (44%), bars/restaurants (19%) and liquor stores (15%). On average, participants were living in the vicinity of six tobacco outlets in a 500 m radius (figure 1). The median distance from residence to the nearest tobacco outlet was 182 m (IQR 109–293 m). Forty-six per cent of participants reported smokers in the home, air nicotine was detected in 48% of homes. In homes with detectable air nicotine, the average level was 1.45 \( \mu g/m^3 \).

Although there was no association between distance to the nearest tobacco outlet and indoor air nicotine levels, greater tobacco outlet density was associated with higher air nicotine concentrations. Tobit regression modelling demonstrated that each additional tobacco outlet in a 500 m radius was associated with a 12% increase in indoor air nicotine levels (\( p < 0.01 \)) and an 8% increase in serum cotinine levels (\( p = 0.01 \)), adjusting for clustering by CSA and possible confounders: age, caregiver education, health insurance, caregiver income, presence of smokers in the home, household occupancy and type of dwelling (table 2, figure 2). In adjusted models, for every 10-fold increase in air nicotine levels, there was a 0.25-point increase in ATAQ score (\( p = 0.01 \)), and for every 10-fold increase in serum cotinine levels, there was a 0.54-point increase in ATAQ score (\( p = 0.046 \)) (table 3).

**DISCUSSION**

These findings demonstrate that among an inner-city population, higher density of tobacco outlets is associated with higher levels of indoor air nicotine and serum cotinine, which are both associated with less controlled childhood asthma. Prior studies have demonstrated that neighbourhoods with higher tobacco outlet density also have higher rates of smoking prevalence.\(^{18,19}\) SHS exposure is known to have detrimental health effects on children with asthma,\(^{7–9}\) and these results suggest that regulations on establishments selling tobacco could improve the health of children with asthma from disadvantaged backgrounds.

Prior to the pandemic, children from inner-city neighbourhoods spent more than 90% of their time indoors.\(^{10}\) During the pandemic, families have spent more time in the home environment, potentially increasing exposure to indoor air pollutants. Understanding how community-level variables associate with

**Table 1** Demographic and home characteristics from 139 participants from AIRWEIGHS study

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Values</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years; median (range))</td>
<td>11 (8–17)</td>
<td>139</td>
</tr>
<tr>
<td>Race (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>88</td>
<td>139</td>
</tr>
<tr>
<td>White/other</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Sex (% male)</td>
<td>59</td>
<td>139</td>
</tr>
<tr>
<td>Caregiver education (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Less than college</td>
<td>59</td>
<td></td>
</tr>
<tr>
<td>Some college/beyond</td>
<td>41</td>
<td></td>
</tr>
<tr>
<td>Health insurance (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Private</td>
<td>16</td>
<td></td>
</tr>
<tr>
<td>Public</td>
<td>84</td>
<td></td>
</tr>
<tr>
<td>Caregiver income (%)</td>
<td></td>
<td>139</td>
</tr>
<tr>
<td>&lt;$20,000</td>
<td>42</td>
<td></td>
</tr>
<tr>
<td>$20,000–$39,999</td>
<td>26</td>
<td>26</td>
</tr>
<tr>
<td>&gt;$40,000</td>
<td>19</td>
<td>19</td>
</tr>
<tr>
<td>Unknown</td>
<td>14</td>
<td>14</td>
</tr>
<tr>
<td>Household occupancy (median (IQR))</td>
<td>4 (2–9)</td>
<td>138</td>
</tr>
<tr>
<td>Type of dwelling (%)</td>
<td></td>
<td>139</td>
</tr>
<tr>
<td>Detached/semidetached</td>
<td>15</td>
<td></td>
</tr>
<tr>
<td>Row home/town home/apartment</td>
<td>85</td>
<td></td>
</tr>
<tr>
<td>Smokers in home (%)</td>
<td>46</td>
<td>138</td>
</tr>
<tr>
<td>Indoor air nicotine (median (IQR))</td>
<td>0.02 (0.02–5.17)</td>
<td>139</td>
</tr>
<tr>
<td>Serum cotinine (median (IQR))</td>
<td>0.20 (0.05–2.7)</td>
<td>44</td>
</tr>
<tr>
<td>Tobacco outlet density (median (IQR))</td>
<td>6 (0–37)</td>
<td>138</td>
</tr>
<tr>
<td>NAEPP asthma severity (%)</td>
<td></td>
<td>139</td>
</tr>
<tr>
<td>Intermittent</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Mild persistent</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>Moderate persistent</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Severe persistent</td>
<td>17</td>
<td></td>
</tr>
<tr>
<td>ATAQ score (mean (range))</td>
<td>2.5 (0–7)</td>
<td>137</td>
</tr>
</tbody>
</table>

ATAQ, Asthma Therapy Assessment Questionnaire; NAEPP, National Asthma Education and Prevention Program.
Original research

Table 2  Association between tobacco outlet density and SHS exposure (airborne nicotine and serum cotinine), adjusting for confounders and clustering

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Air nicotine (n=108)</th>
<th>P value</th>
<th>Serum cotinine (n=36)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>β (95% CI)</td>
<td></td>
<td>β (95% CI)</td>
<td></td>
</tr>
<tr>
<td>Tobacco outlet density</td>
<td>0.05 (0.02 to 0.08)</td>
<td>&lt;0.01</td>
<td>0.03 (−0.01 to 0.06)</td>
<td>0.01</td>
</tr>
<tr>
<td>Age</td>
<td>−0.06 (−0.23 to 0.12)</td>
<td>0.53</td>
<td>−0.11 (−0.03 to 0.25)</td>
<td>0.11</td>
</tr>
<tr>
<td>Caregiver college education</td>
<td>−0.22 (−1.11 to 0.66)</td>
<td>0.62</td>
<td>−0.43 (−1.28 to 0.42)</td>
<td>0.32</td>
</tr>
<tr>
<td>Public insurance</td>
<td>−0.12 (−1.78 to 1.53)</td>
<td>0.88</td>
<td>−4.71 (−6.31 to 3.10)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Caregiver income</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;$20 000</td>
<td>−2.04 (−1.17 to 0.69)</td>
<td>0.62</td>
<td>0.01 (−0.80 to 0.83)</td>
<td>0.98</td>
</tr>
<tr>
<td>$20 000–$39 999</td>
<td>−1.40 (−3.22 to 0.42)</td>
<td>0.13</td>
<td>1.77 (0.69 to 2.85)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Public insurance</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smokers present</td>
<td>2.07 (1.16 to 2.99)</td>
<td>&lt;0.01</td>
<td>1.77 (0.69 to 2.85)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Household occupancy</td>
<td>0.17 (−0.03 to 0.36)</td>
<td>0.10</td>
<td>−0.06 (−0.49 to 0.36)</td>
<td>0.77</td>
</tr>
<tr>
<td>Row hometown/home/apartment</td>
<td>0.26 (−0.69 to 1.21)</td>
<td>0.59</td>
<td>−0.38 (−1.79 to 1.04)</td>
<td>0.60</td>
</tr>
</tbody>
</table>

Adjusted tobit regression model used.

SHS, secondhand smoke.

indoor air pollution and asthma health has become increasingly relevant. Our findings demonstrate that among children with asthma in Baltimore City, the number of tobacco retailers in a three-block radius is nearly six times greater than the tobacco outlet density reported using the same buffer in Houston, Texas.23

There is growing evidence to support that the SES of a neighbourhood can impact health outcomes. Predominantly black neighbourhoods28 and low-income areas have been found to have a higher concentration of tobacco outlets,15 29 30 likely driven by low rental costs in these neighbourhoods. Prior studies have demonstrated that independent of SES, predominantly black neighbourhoods have greater tobacco outlet availability and access.28 Simulation models have suggested that the cost of tobacco products decreases by up to 20% in neighbourhoods where tobacco outlets are more highly concentrated.31 32 Subsequently, the residents of these disadvantaged areas have greater access to tobacco products at a lower cost. The downstream effect seen is a higher prevalence of smoking in low-income neighbourhoods with increased tobacco outlet density.18 19 33

We recognise there are multiple pathways that may contribute to the association between tobacco outlet density and indoor air nicotine. About 25% of caretakers reported that they smoked in the home, a total of 46% reported the presence of smokers in the home and air nicotine was detected in about half of the homes. It is unlikely that the participants (children with asthma) contributed to airborne nicotine, because study criteria required that all participants be non-smokers, and this was confirmed with urine cotinine testing. However, household smoking data were provided by self-report which may underestimate the true

Figure 2  The association between tobacco outlet density and indoor air nicotine levels. Outlier removed for graph only (tobacco outlet density of 9, indoor air nicotine level of 21.7 µg/m³).
prevalence. The detectable levels of indoor air nicotine could alternatively be from individuals loitering and smoking around tobacco outlets, visitors or neighbours smoking directly outside the home or due to cross-contamination from adjacent homes with shared ventilation. Though the source of airborne tobacco may vary, tobacco outlet density demonstrated a measurable association with indoor air pollution which had a negative association with childhood asthma control.

Tobacco outlet density has been previously described in the literature as a neighbourhood characteristic that is associated with poor health outcomes. These results show that the number of tobacco outlets within a three-block radius of the home is associated with higher levels of detectable indoor air nicotine and serum cotinine, independent of the presence of smokers in the home. Our finding of higher levels of SHS exposure among participants with higher tobacco outlet density is consistent with previous reports of higher rates of smoking prevalence in low-income neighbourhoods with high tobacco outlet density. However, air nicotine levels and serum cotinine levels were elevated even in non-smoking homes, and a plausible explanation would be that the presence of tobacco outlets is leading to more smoking of tobacco products within the vicinity of tobacco outlets, which is polluting the nearby indoor environment. Prior studies of cue reactivity have demonstrated that cravings among tobacco users are increased when exposed to drug-related stimuli. While smoking behaviours around tobacco outlets were not directly assessed in the scope of this study, future studies could investigate this postulation as a potential cause of higher indoor airborne nicotine in homes with high tobacco outlet density.

Indoor air nicotine can leave an imprint in the home. Literature suggests that persistent SHS can accumulate on surfaces, clothing and dust, lending to third-hand smoke (THS) exposure. Some THS pollutants can react with ambient toxins and create novel compounds with carcinogenic potential. Children can be exposed to THS toxins by touching surfaces in the home, breathing indoor air and ingesting residue that settles on their hands. Research shows that young children are often exposed to higher levels of THS because they spend a significant amount of time crawling on floors and touching other contaminated surfaces. Studies have shown that children from low-income backgrounds have higher levels of exposure to environmental smoke.

SHS exposure has a multitude of health effects, and in this study we focused on the health impact on childhood asthma. We found that children with asthma exposed to higher levels of SHS had a significant reduction in asthma control as measured by the ATAQ score. Both tobacco outlet density and the presence of smokers in the home were strongly associated with higher indoor air nicotine levels and could contribute to the downstream effect of poor asthma control. Quantifying this relationship between SHS exposure and asthma control with granular measurements of airborne nicotine and serum cotinine further enriches the literature in support of the detrimental health effects of SHS on asthma.

On a national scale, paediatric asthma healthcare expenses amount to nearly $6 billion annually. The Maryland Health Services Cost Review Commission (HSCRC) reports that the average cost of an emergency room visit for paediatric asthma is approximately $1000, and in 2019 there were more than 11 500 emergency room visits for asthma. The cost is even greater for children hospitalised for asthma; the HSCRC reports over 960 paediatric asthma hospitalisations averaging $9000 each. The cost of asthma care is a large burden to the healthcare system,
and efforts to reduce SHS exposure can have significant impact on addressing this public health crisis.

We acknowledge that our study focused on low-income urban neighbourhoods, which may limit the generalisability of findings. These neighbourhoods in Baltimore do have many multiunit row homes with shared ventilation systems, which can allow indoor air pollution to spread among neighbouring homes. Although our analysis did adjust for the type of dwelling, we recognise that there are other potential confounders of SHS exposure. Specifically, smoking policies of residential buildings and observed smoking behaviour around/outside the home were not accounted for. While we did collect air nicotine data for all participants, serum cotinine measurements were only available for a subset, and future studies with a larger number of participants may be able to expand on these findings.

Tobacco outlets are rapidly evolving, with an uptake in the sale of electronic cigarettes and vaping products in the recent years. In Baltimore City, a tobacco licence permits sale of any type of tobacco product, and the city does not provide records of the types of tobacco products sold at each outlet. For this reason, our study does not specifically look at the effect of electronic cigarette and/or vaping products on indoor air nicotine levels, but this is a topic to consider for future study if information regarding sales of these products at specific outlets were to become available.

Tobacco outlets are concentrated in urban areas and neighbourhoods with poverty, and there is increasing evidence to support how this is associated with SHS exposure and health outcomes. There are several elements of the retail-focused tobacco control strategies which include federal regulation of advertisements, addressing inequities of the tobacco outlet environment and retail strategies controlling licensing, pricing and product availability.

On a local level, approaches to decrease tobacco outlet density could include measures that limit the number of tobacco licences in a given jurisdiction, prohibiting tobacco outlets within a given distance of schools or setting a standard for the minimum distance between two tobacco outlets. Current legislation in Baltimore City does not prevent tobacco outlets from opening in residential neighbourhoods, nor does it limit the number of tobacco outlets that can exist in a given neighbourhood. Like the sale of tobacco products, alcohol sales were previously unregulated in Baltimore City. After studies demonstrated a strong correlation between presence of liquor outlets in Baltimore City neighbourhoods and poor public health, in 2016 Baltimore City implemented a liquor zoning law prohibiting liquor outlets from operating in residential neighbourhoods. Zoning laws restricting tobacco outlets in residential neighbourhoods in Baltimore City will advance the goal of reducing tobacco-related health consequences.

This paradigm likely exists beyond just the Baltimore City. In addition to what is known about the effect of tobacco outlet density and proximity on smoking behaviour, our findings bring to the forefront the association between tobacco outlet density, indoor air quality and the health of children with asthma, which can inform policies restricting the sales of tobacco, particularly in disadvantaged neighbourhoods.

What this paper adds

What is already known on this subject
⇒ Tobacco outlets are concentrated in low-income neighbourhoods. Tobacco outlet density is associated with higher prevalence of smoking. Children with asthma are susceptible to effects of tobacco, and secondhand smoke (SHS) exposure is associated with more severe asthma and worse lung function among children with asthma.

What important gaps in knowledge exist on this topic
⇒ The association between tobacco outlet density and SHS exposure among children in the community is unknown and the potential association with health outcomes among children with asthma, who represent a high-risk group, is of particular relevance.

What this paper adds
⇒ Living in a home with higher density of tobacco outlets in the surrounding neighbourhood is associated with higher levels of indoor air nicotine and serum cotinine, independent of the presence of smokers in the home. Using direct measurements of airborne nicotine in the home, we found as indoor SHS exposure increases, there was a significant reduction in asthma control as measured by the Asthma Therapy Assessment Questionnaire score.

What is already known on this subject
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