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# Sociodemographic disparities in thirdhand smoke exposure among children in the United States: a narrative review

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## Abstract

Thirdhand smoke (THS) is the toxic residue left behind by tobacco smoke that persists indoors on carpets, furniture, toys, and other items. Children are especially vulnerable to THS exposure due to their hand-to-mouth behaviors, activity near the floor, and developing organs. We provide a scoping narrative review of potential sociodemographic disparities in exposure to THS among children in the United States. Articles were obtained by conducting a search in the PubMed, SCOPUS, and CINAHL databases. Other relevant papers were also reviewed and integrated using narrative synthesis. Studies relevant to THS exposure among 0 to 17-year-olds and conducted in the United States were included. We identified lower socioeconomic status (SES), race, younger age (2-4 years), and living in multiunit housing (MUH) as potential contributors to higher THS exposure risk in children. Lower income was associated with greater THS exposure. THS exposure was higher in Black children, although the number of studies involving them was limited. Examining disparities in exposure and susceptibility to THS can inform future policies to reduce children's environmental exposure to toxic tobacco chemicals in THS. Housing type is likely a primary contributor to exposure disparities, as low-income children are more likely to live in multiunit, rented housing, where tobacco smoke can travel across housing units and where THS residue from former tenants

can persist. Policies limiting persistent THS residue indoors through comprehensive smoking bans and expanded outreach on approaches to reduce THS exposures among children are needed.

**Keywords:** Children, thirdhand smoke, health disparities, tobacco, environmental exposures

## INTRODUCTION

Thirdhand smoke (THS) is the toxic chemical residue originating from secondhand smoke (SHS) that remains in indoor environments[1]. THS can also be transported into nonsmoking environments by people and items exposed to tobacco smoke (e.g., clothing and furniture)[2]. THS residues are found on indoor surfaces, including carpets, furniture, toys, and bedding. THS contaminates house dust[3] and building materials (e.g., drywall), and it includes semi-volatile compounds that can be re-emitted into ambient air[1]. THS toxicants include nicotine, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK), and other tobacco-specific nitrosamines (TSNAs), heavy metals (lead, cadmium)[4,5], and polycyclic aromatic hydrocarbons[6-8]. Chemical reactions between THS constituents and environmental oxidants can yield secondary contaminants that are more toxic than the original compounds[7]. One example is the reaction of nicotine and indoor oxidants [e.g., nitrous acid (HONO)] to form NNK, a potent carcinogen[9].

THS is persistent and detected at least six months after residents have ceased smoking in or vacated their homes[3,10]. Matt *et al.* measured significant THS in multiunit housing (MUH) where there had been no smoking for ≥ eight years[11]. Consequently, when nonsmoking tenants and their children move into a home previously occupied by smokers, they are exposed to THS[3]. We use the term MUH to refer to separate housing units for residents that are within a single building. These units have shared walls and sometimes shared ventilation and hallways; examples include apartments in a complex and condominiums.

Methods to detect THS include collecting and testing indoor dust and surface wipe samples for nicotine[3,10-12], NNK[13], and other TSNAs[10]. In non-smoking homes, air nicotine serves as a marker for THS contamination in previously contaminated environments[3]. It should be noted that nicotine may also be detected where e-cigarettes containing nicotine have been used[14]. In SHS exposure studies, THS may be identified by nicotine found in house dust[15], on surfaces[16], and on hands or fingers, particularly among children[17-19]. Researchers have classified THS exposure among children as living with someone who smokes, even if not at home[20-22]. Biomarkers of exposure in nonsmoking environments or in studies of SHS and THS include serum cotinine[23], urinary cotinine[24-26], salivary cotinine[27-29], and urinary 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol (NNAL, the metabolite of NNK)[19,25,30]. There are no currently validated biomarkers specific to THS exposure, although compounds such as 4-(methylnitrosamino)-4-(3-pyridyl)butanal (NNA) have been reported as unique to THS[1,31].

Both *in vitro* and animal studies suggest that THS exposure may result in adverse human health consequences. *In vitro* exposure to THS extracts resulted in genotoxic effects[32] and metabolic alterations[33] in cell systems as well as damage to lung cells[34]. Studies with mice models report alterations in multiple organ systems, including an increased risk of liver disease[35-38] and lung cancer[39]. THS exposure among mice has been linked to delayed wound healing[38], insulin resistance[40], alterations in the immune response[41], and behavioral changes such as hyperactivity[38].

There are limited human studies on the adverse health effects of THS. However, among THS-exposed children, studies demonstrate alterations in gut microbiomes[42,43], increased gastrointestinal problems[44], respiratory symptoms[45], bacterial illnesses[24], oral health problems[46], inadequate sleep patterns[47,48], and

exposure to NNK[25,30,49-51]. Dermal THS exposure among nonsmoking adults has resulted in harmful inflammatory effects similar to actual cigarette smoking, with alterations in the human plasma proteome and elevations in urinary oxidative stress biomarkers[52]. Further research is needed to quantify and better understand the direct toxicity of THS on human health.

In this paper, we review current evidence on THS contamination and exposure among children in the United States. This review differs from other reviews of THS[1,7,8,53-57] in that we focus on potential disparities in children's exposure related to sociodemographic factors. Other reviews have focused on chemical composition, toxicity, exposure, health effects, and policy implications. Our narrative scoping review examines the available literature on disparities and highlights the need for additional studies and approaches to reduce THS exposure in vulnerable populations.

## METHODS

We searched the PubMed, SCOPUS, and CINAHL databases on October 7, 2024, using the following terms: (thirdhand OR "third hand") AND (smoke OR cigarette OR tobacco) AND (child OR children OR children's OR infant), for papers on THS exposure among children. The search yielded 376 results across the three databases. We used Covidence, a web-based management software, to eliminate duplicates, to allow for dual reviewers, and to track the screening process of each reviewer. The search results (e.g., reference and abstract) were imported into Covidence, and once duplicates were removed, there were 183 citations remaining [Supplementary Figure 1]. Two reviewers (LH and PJEQ) evaluated the 183 abstracts independently based on criteria developed by the research team (described below). Once all abstracts were screened individually by both reviewers, they met to discuss and resolve any disagreements. Studies were included if they were relevant to THS exposure among 0-17-year-old children and conducted in the United States. We limited the review to studies from the United States as most THS research using objective field measurements (e.g., THS residue in dust, on surfaces, and from hand wipes) has been conducted in the United States. Twenty-eight publications from the search met the inclusion criteria. Other papers were excluded for not being original research ($n$ = 64), laboratory or chamber studies ($n$ = 14), studies not including THS measures and not conducted in the United States ($n$ = 29), studies including THS objective measures but not conducted in the United States ($n$ = 4), field studies but not involving children ($n$ = 33), or primarily focusing on SHS or not distinguishing THS ($n$ = 11). The Thirdhand Smoke Research Collection was also reviewed for publications relevant to THS exposure disparities among children[58]. One additional paper was identified and included, a seminal paper published before the term THS was used[59]. A list of all 29 studies is available in the supplemental materials [Supplementary Table 1]. Although these 29 studies met the criteria for focusing on THS exposure among children in the United States, they did not all assess disparities in exposure. We reviewed all papers to extract information that could be relevant to disparities (e.g., income, housing type, *etc.*) and compiled them to narratively identify potential disparities in exposure. The papers that did not include information related to disparities were still integrated in other sections of this report (e.g., background on THS and adverse health among children).

## CHILDREN ARE VULNERABLE TO THS EXPOSURE

Children are susceptible to indoor dust exposure, both from surfaces and airborne particles, due to their behaviors and physiology[60]. Children spend more time than adults on or near the floor, where they can touch or resuspend and inhale house dust, and their hand-to-mouth and exploratory behaviors amplify THS exposure[60]. Due to their smaller size, children inhale and ingest more toxicants per kg than adults in the same environment. Their developing organs, limited ability to clear toxicants, and inability to remove themselves from THS exposure places them at greater risk for potential adverse health effects associated with such exposure[49,61,62]. A seminal THS study identified multiple sources of exposure for children[59].

Nicotine was detected in the air, on surfaces, and in settled house dust, regardless of whether mothers smoked inside or only outside the home. Additionally, infants may be exposed to nicotine via the hands of caregivers, as nicotine was detected on the index fingers of maternal smokers[59].

Hand nicotine measurement among children is used as a marker of THS exposure. In a study of 104 children of smokers, 2-4-year-olds had the highest hand nicotine levels compared to younger and older children[63]. In another study of 193 children < 12 years old, it was reported that 2-4-year-olds had the highest levels of hand nicotine[17]. Mahabee-Gittens *et al.* reported nonlinear relationships between age and hand nicotine in children who lived with a smoker and were tested during emergency room visits; children aged 2-4 years had the highest hand nicotine levels compared to other age groups[24]. Although biomarkers cannot distinguish between SHS and THS exposure, negative correlations between age and both urinary cotinine and NNAL have been reported among children who lived with a smoker[25].

Surprisingly, Neonatal Intensive Care Units (NICUs) can also provide environmental THS exposure to newborn infants, despite strict no-smoking hospital policies[26]. Northrup *et al.* (2021) reported that over 90% of NICU bedside furniture was contaminated with nicotine[26], which would subject infants to ongoing exposure to nicotine and potentially other THS pollutants for the duration of their time in the NICU. Another study by Northrup *et al.* also demonstrated that nicotine could be transported into the NICU by visitors and staff[18]. Approximately 80% of NICU staff had detectable levels of nicotine on their fingers[18], suggesting that handwashing and hand sanitizing may not fully remove nicotine residues[64]. A longitudinal study of discharged NICU patients found that although homes with indoor smoking bans had lower levels of THS residues compared to those without bans, smoking bans did not eliminate THS inside the homes[29].

## SOCIODEMOGRAPHIC STATUS AND THS EXPOSURE

Disparities in SHS exposure by race, income, and education level are well-documented, particularly among nonsmokers[65]. MUH residents in California were more likely than single-family housing residents to be smokers[66]. Racial/ethnic minorities and individuals with low incomes were also more likely to live in MUH[66]. These disparities in SHS exposure are relevant for THS exposure because THS results from SHS in indoor environments[1]. An estimated three million children < 6 years old are exposed to SHS at least four days a week in the United States[31,67]. Although there has been an overall decline in SHS exposure in the United States since 1999, there remains disproportionately higher SHS exposure among children from low socioeconomic status (SES) families[15,17,20,21,23,28]. A study using serum cotinine to assess exposure reported that SES factors such as lower income and maternal education were significant predictors of increased SHS exposure in children[28]. Residential instability, defined as the number of times a family moves homes, also increases the odds of exposure for children[28]. SHS exposure disparities are relevant to THS exposure in the following ways: SHS exposure at home also exposes children to THS residues that contribute to their total toxicant exposure. In addition, SHS exposure in low-income communities increases the likelihood of children's tobacco toxicant exposure in THS-contaminated homes, as THS persists even after smokers move out[3].

Family income may correlate inversely with THS exposure in children, suggesting that it may be an important factor to consider for disparities in exposure. Among 504 children who were not exposed to SHS (i.e., no parental smoking and home smoking bans), those with family incomes of ≤ $15,000 had 5.7 times higher levels of hand nicotine contamination compared to children from families with incomes of ≥ $30,000[17]. Merianos *et al.* (2023) assessed surface levels of the carcinogen NNK in the homes of 84 children who lived with a smoker and found higher levels in homes with household incomes of ≤ $15,000 compared to > $15,000[68]. Child-occupied households with low parental income had significantly higher

concentrations of nicotine in household dust compared to those with higher incomes, despite no smoking among either group of parents[15].

THS-exposed children in one study also faced structural barriers to achieving better health. Children aged 6-11 years exposed to THS (defined as living with a smoker who did not smoke inside the home) had a significantly lower number of total neighborhood amenities such as sidewalks and parks compared to non-exposed children[20]. Those with THS exposure were also more likely to report moving more often and having mold in the home[69]. Their odds of food and financial insecurity were also higher compared to children with no THS exposure[21].

## MUH AS A SOURCE OF THS EXPOSURE

Housing type, specifically MUH, contributes to THS exposure disparities. Established THS detection methods (e.g., surface nicotine levels measured via wipes, TSNA levels measured via dust samples) suggest that THS can travel to other non-smoking homes and persist in homes where previous tenants smoked, despite cleaning efforts[3,10,11]. In homes occupied by smokers who later quit, Matt *et al.* found that THS contaminants, such as nicotine and TSNAs, can remain in homes even six months after smoking cessation among occupants[10]. In homes of former smokers that underwent standard deep cleaning in preparation for new nonsmoking tenants, nicotine levels decreased, but after six months, these homes still had significantly higher THS contamination compared to homes of nonsmokers[3]. THS-contaminated MUH low-income homes achieved lower levels of dust nicotine after a standard deep cleaning, but levels rebounded after three months, indicating hard-to-remove persistent reservoirs in MUH[70,71]. These findings suggest that THS contamination in homes is not eliminated by smoking cessation or with standard cleaning methods. More intensive cleaning methods are needed to fully remove THS and thus address potential disparities in exposure and possible health effects for lower SES MUH occupants.

A cross-sectional study that assessed THS levels in 220 low-income MUH homes in California confirmed the presence of THS in all homes, including nonsmoking homes[11]. Although surface nicotine levels were typically higher in homes where occupants or visitors smoked indoors (Median = 297 $g/m^2$), compared to the overall level in all homes (Median = 1.47 $g/m^2$), the homes with the two highest surface nicotine concentrations were nonsmoking homes[11]. Follow-up with occupants of the unit with the second highest nicotine concentration (2,586 $g/m^2$) revealed that a previous resident had smoked in the home until stopping three years prior to sample collection[11]. In a study of surface nicotine concentrations in subsidized private MUH housing in Ohio, mean nicotine surface levels in non-smoking homes (11.4 $g/m^2$) were higher than those in the California study, and mean nicotine surface levels in smokers' homes were 90.9 $g/m^2$[16]. In addition to greater THS exposure among nonsmoking MUH residents due to higher housing turnover rates, SHS from neighbors can also drift into adjacent spaces in MUH.

Recent studies have reported disparities in SHS and THS exposure among children according to housing type. Merianos *et al.* found that serum cotinine levels among children declined from 1999 to 2014; however, exposure was more than two times more likely among children living in homes that were rented, and a majority of these were in MUH[23]. In addition, low-income children living in MUH had significantly higher salivary cotinine measurements compared to children residing in single-family homes[27].

## RACE/ETHNICITY AND THS EXPOSURE

Few publications have focused on race/ethnicity with regard to THS exposure among children[17,21]. SHS exposure is known to be associated with race/ethnicity[23,72]. Findings that Black children were disproportionately exposed to SHS also have implications for THS exposure[72-74]. Based on a national

sample, Black children were more likely than White children to be exposed to SHS/THS (measured by serum cotinine), and Hispanic children were less likely than White children to be exposed[23]. Matt *et al.* reported significantly higher levels of nicotine on the hands of children of Black parents compared to children of White parents[17]. In their analysis of a national survey on children's health, Mahabee-Gittens *et al.* found that non-Hispanic White children had the highest prevalence rates of THS exposure (58.2%)[21]. THS exposure was defined as living with a smoker who did not smoke in the home. The prevalence of THS exposure was 22.2% among Hispanic children compared with 9.5% among Black children; however, SHS + THS exposure was higher among Black compared to Hispanic children (23.0% *vs.* 6.1% respectively).

In some regions of the United States, a significant proportion of Hispanic families live in MUH, which could increase the risk of THS exposure among their children. In California, 45% of the Hispanic population lives in MUH[75]. Through focus group discussions with Hispanic parents living in MUH, Rendón *et al.* found that participants were generally aware of the potentially adverse health impacts of SHS/THS exposure[75]. However, they expressed reluctance to negotiate with neighbors who smoked nearby, citing language and fear of retaliation as major barriers.

These findings underscore the need for further research on racial disparities and the intersection of race, ethnicity, SES, poverty, and living in MUH as risks for THS exposure and potential adverse health effects.

## PROTECTIVE FACTORS AGAINST THS EXPOSURE

Given the limited research on THS exposure among children, as well as the ubiquitous nature of THS, few effective protective factors have been identified. Living in single-family homes and having higher incomes are associated with lower THS exposure among children[17,27]. Home smoking bans can also help lower THS exposure. Children who lived in homes with smoking bans had significantly lower levels of nicotine on their hands and lower urinary NNAL[19]. In Quintana *et al.*'s study (2024), NNK was detected on pillows deployed in smoking homes with no bans and not on pillows from the other homes[76]. A longitudinal study by Matt *et al.* (2023) examined hand nicotine levels among children of smokers who presented to the emergency department or urgent care and again six weeks later at a home visit. Those with home smoking bans had lower levels of hand nicotine six weeks later compared to their initial measurement, while children without home smoking bans had higher hand nicotine levels six weeks later compared to their initial measurement[77].

Parental beliefs can affect home smoking bans and, thus, potential exposure to THS. A pilot study tested an educational intervention on THS among smoking caregivers of children presenting to urgent care; among 12 people who received the intervention, 25% changed their home and car smoking policies[78]. Although this was a pilot study with a small sample, educational interventions on THS may have the potential to promote home smoking bans[78]. Among a national sample of 1,478 participants, 93% of respondents believed SHS is harmful to children, but only 61% believed THS is harmful to children[79]. Having the belief that THS is harmful to children's health was associated with having strict nonsmoking rules in the home[79]. Increasing levels of education have also been positively associated with implementing smoking bans: college graduates were over four times more likely to have household smoking bans compared to those who did not complete high school[79]. Record *et al.* (2024) reported that among 363 parents in California, those with higher education attainment were more likely to have endorsed THS-protective behaviors[80]. This indicates that parental educational status needs to be considered when planning information campaigns related to THS. Gatzke-Kopp *et al.* identified time spent in a daycare center as a protective factor. This suggests that measures to make spaces free of THS contamination are protective[28].

## DISCUSSION AND CONCLUSION

We identified the following factors that may create or exacerbate disparities in THS exposure among children: lower SES, Black race, younger age (2-4 years), and living in MUH. Although Black children were reported to have greater THS exposure, studies of minority populations are limited, highlighting the need for additional studies. THS can further exacerbate health disparities among low SES populations who live in MUH. Low-income housing has a high risk of THS contamination and should be considered a major contributor to THS exposure disparities as children in these settings face a combination of risk factors: low household income and living in MUH which may have THS contamination from previous tenants or neighbors. Additionally, there is no nationwide policy that bans indoor smoking in all low-income housing. In settings with THS protective policies (e.g., public housing[81]), those living there may not necessarily benefit from these policies due to a lack of implementation and enforcement. It should also be highlighted that the COVID-19 pandemic resulted in more time spent at home for children living in MUH during school closures, which may have increased THS exposure[82] and exacerbated already existing disparities in exposure.

Limitations of this review include a lack of information in published studies allowing for comparisons of THS exposure by race/ethnicity and household income of participants. In addition, most studies were conducted across only a few geographic locations within the United States, thus limiting generalizability. Aside from a third of the studies that analyzed national survey data (e.g., National Health and Nutrition Examination Survey, National Survey of Children's Health), most of the remaining studies were based in California or Ohio. The limited regional representation across studies can increase the risk of bias. For example, California has strict anti-smoking laws, which is not the case across all other states; therefore, exposure disparities may be greater elsewhere. Other considerations for bias in studies include participant selection that tends to capture those from low-income backgrounds (e.g., recruitment through Women, Infants and Children Supplemental Nutrition Program sites). Lastly, medical mistrust may have underestimated exposures reported as some studies took place in medical settings. To our knowledge, this is the first review to focus on disparities in THS exposure among children. Because THS research is still an emerging topic area, studies were quite limited overall, and thus, a systematic review is not possible at this time. However, collaborative research partnerships have been formed to advance the field (e.g., California Consortium on Thirdhand Smoke)[1,83].

There is a need to standardize THS contamination and exposure measurements for children in order to document disparities and link these disparities to adverse health outcomes. Validated measures to distinguish THS from SHS exposures are lacking. In the studies reviewed here, THS was assessed through questionnaires[20,21,44,46,48,69,84], surface wipes[11,16,68], dust[15], children's hand wipes[17,24,63,76,77,85], pillows[76], and/or biomarkers[23,25,27,28,30,76]. Most biomarker measures were for nicotine[3,10,11,17,24,63,74] or its metabolite cotinine[23,25,27,28,73,76]. Relatively few studies measured other markers such as NNK or NNAL[25,30,51,74,76]. The most accurate measurements are through direct THS sampling on surfaces, dust, or hands. Mahabee-Gittens *et al.* reported that hand nicotine may be a reliable source of data on THS exposure, given the associations found between hand nicotine levels and the presence of home smoking bans[19,85]. A limitation of using nicotine on surfaces as a marker for THS contamination is that the sources of nicotine contamination could be due to cigarettes and/or electronic nicotine delivery systems (e.g., e-cigarettes)[14,86], causing THS contamination from tobacco smoke to be overestimated when vaping has taken place. It is important, however, to note that nicotine on surfaces from thirdhand vape also poses risks, as nicotine has been shown to be converted by common indoor oxidants to the potent carcinogen NNK and other TSNAs[9]. Surface samples collected from vape shops had a higher ratio of NNK to nicotine than seen in thirdhand tobacco smoke surface samples[68,87,88]. Therefore, though nicotine on surfaces may indicate both

THS and thirdhand vape, risks to children may occur under both scenarios. However, thirdhand vape is less likely to contain high levels of other toxicants such as polycyclic aromatic hydrocarbons (PAHs) and heavy metals measured in THS, and better markers for THS from tobacco smoke are needed.

Reducing THS exposure among children necessitates better detection methods for THS. Although methods to detect THS contamination in homes are improving, there are difficulties in establishing public standards for "safe" exposure levels[31]. One approach might be to model THS testing requirements on those for lead and radon testing (e.g., when selling a home)[89]. The US Environmental Protection Agency recently proposed a strict rule to eliminate lead contamination in homes[90]. Since indoor smoking is a source of lead in house dust[5], smoking bans and THS remediation may be considered a part of interventions to reduce lead exposure. Furthermore, following the precautionary principle[91], policies to prevent smoking in MUH complexes (indoors and outdoors within the complex) would reduce THS exposure among vulnerable children[31]. The US Department of Housing and Urban Development proposed a rule in 2017 mandating that all public housing agencies implement smoke-free policies[81]; these must be more effectively implemented and enforced[17]. Indoor smoking leads to pervasive THS-contaminated housing, which affects children living with smokers, future tenants, and neighbors. Information about reducing THS exposure among children should be included in caregiver education and outreach that targets other forms of environmental contamination, such as house dust in the home[92]. Dust reduction will also reduce children's exposure to toxic chemicals in THS.

This review summarizes current information regarding disparities in THS exposure among children, and these results should be considered in efforts to equitably address the health consequences of smoking, not only for people who smoke but for the most vulnerable people who do not smoke.

## DECLARATIONS

### Authors' contributions

Conceptualization, search strategy, study screening, manuscript writing - original draft, manuscript writing - review and editing: Hamzai L
Conceptualization, search strategy, manuscript writing - feedback, review, and editing: Quintana PJE
Conceptualization, manuscript writing - feedback, review, and editing: Matt GE, Novotny TE
Manuscript writing - feedback, review, and editing: Mahabee-Gittens EM, Merianos AL, Dodder NG, Lopez-Galvez N, Hoh E

### Availability of data and materials

The data supporting this article have been included as part of the Supplementary Materials.

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### Conflicts of interest

All authors declared that there are no conflicts of interest.

### Ethical approval and consent to participate

Not applicable.

### Consent for publication

Not applicable.



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