Thirdhand smoke: when the danger is more than you can see or smell

Thirdhand smoke: quando o perigo vai além do que se vê ou sente

Thirdhand smoke: cuando el peligro va más allá de lo que se ve o se siente

Abstract

Recent discussion has focused on another form of exposure to tobacco – thirdhand smoke (THS) – consisting of residual pollutants from cigarette smoke that remain in environments. The main concern with THS is based on the presence and persistence of many toxic compounds, some specific nitrosamines from tobacco that have carcinogenic activity. Little is known about THS, and few people are aware of its existence and potential health repercussions, thus highlighting the need to shed light on the subject and incorporate it into the public health debate, as was done with passive smoking several years ago. THS is a form of passive smoking, together with secondary or involuntary exposure to cigarette smoke.

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Tobacco Smoke Pollution; Tobacco; Smoke
Introduction

The World Health Organization (WHO) estimates that one-third of the world’s adult population is exposed involuntarily to cigarette smoke 1. In Brazil, 10.7% of non-smokers are exposed to smoke in their homes and 13.5% in their workplaces 2. An estimated 600,000 non-smokers die in the world every year from exposure to passive smoking 3.

A meta-analysis concluded that elderly non-smokers living with smokers have 30% greater odds of suffering ischemic heart disease when compared to unexposed elders 4. Individuals exposed simultaneously at home and in the workplace show double the odds of acute myocardial infarction 5. Due to environmental tobacco smoke (ETS), 3,000 deaths occur per year from lung cancer, and the odds of neoplasia are 35% greater in passive smokers 6. Passive smoking is also associated with increased risk of type 2 diabetes mellitus, sinusitis, tuberculosis, and breast cancer 7,8,9,10. There is sufficient evidence that passive smoking causes diseases and premature deaths in children and adults and that there are no safe levels of exposure to cigarette smoke 11.

The harms to passive and involuntary smokers come from the substances released during the burning of tobacco. Cigarette smoke forms an aerosol of gases, vapors, and liquid particles that disperses homogeneously in the air, such that individuals are exposed to significant concentrations of these substances, regardless of distance from the source 12. Recent years have witnessed debate on the danger of contamination by tobacco’s harmful substances, even after the cigarette has been put out. This exposure has been called thirdhand smoke (THS) 13 (in Brazil, the term’s literal translation in Portuguese, fumo de terceira mão, is still not widely used). THS consists of a mixture of volatile compounds and particulate matter that can be deposited or adsorbed on surfaces, including clothing, furniture, and upholstery 14. The smoke that constitutes ETS changes both chemically and physically, especially over time 5,15,16, so THS can also be called residual tobacco smoke or aged tobacco smoke 17. As soon as the smoke begins and until several hours later, THS and smoke coexist, with the predominance of THS as the smoke dissipates and is removed by ventilation 16,17. The persistence of many toxic compounds, even if it is no longer possible to see or smell the smoke, is one of the main concerns over invisible exposure to THS 15,17.

Burton 18 described THS with three R’s: smoke pollutants from tobacco products that remain in the environment and are re-emitted into the air or react with oxidants and other compounds from the environment to form secondary pollutants. Residues of the smoke re-suspended in the air cause contamination by inhalation, and residues deposited on surfaces can be absorbed by the oral or dermal route 19. Passive smoking is no longer only involuntary exposure to cigarette smoke, but incorporates exposure to substances that remain or are formed in the environment after the smoke disperses 20.

In 2003, the Member States of the WHO, including Brazil, adopted the Framework Convention on Tobacco Control (FCTC), an international public health treaty that emphasizes the states’ responsibility to create and enforce effective tobacco control programs 21. The FCTC sets standards to protect present and future generations from the devastating health, social, environmental, and economic consequences of tobacco consumption and exposure to tobacco smoke; the Convention thus aims at continuous and substantial reduction in the prevalence of consumption and exposure to smoke 22.

Even before signing the FCTC, Brazil took a pioneering role by establishing its National Anti-Smoking Program, implementing important measures. Brazil’s tobacco control policy has produced some of the greatest effects over the years. The proportion of Brazilian smokers dropped significantly, from 32% in 1989 to 15% in 2013 23. This reduction was due to numerous interventions, including a ban on cigarette advertising in the mass media and sponsorship of events by tobacco companies, tax hikes on tobacco products, health warnings on cigarette packs, awareness-raising campaigns, and greater availability of behavioral and pharmacological smoking cessation treatment in the public healthcare system, among others 24. Federal Law 12,546/2011, regulated in 2014, with amendments to Law 9,294/1996, bans the use of tobacco products in all closed collective premises, both public and private 25,26. One of the beneficial results of the “antismoking law” was a reduction in exposure of non-smokers to cigarette smoke in workplaces with closed environments, from 22.8% in 2008 to 13.5% in 2013 2,27. Indirectly, all the laws and measures to reduce active and passive smoking have also resulted in reduced exposure to THS.

With the enforcement of the protective measures recommended by the FCTC, there was an increase in “100% tobacco-free environments”, but such areas are located in the public domain or collective-use establishments. The convention fails to include places where people are most vulnerable: in their own homes or other places in which the legal instruments are unable to exercise strict control, as in private cars 28.
The first study on THS was published in 2009 and concentrated on the perception of such smoke’s existence and the enforcement of rules to restrict smoking in smokers’ homes in the United States 13. Almost at the same time, three more studies proposed to publish original findings on the topic, highlighting nicotine’s persistence in the environment and the formation of carcinogenic substances 14,15,19. In the following two years, more research was published in the form of editorials, letters to editors, or expert commentary 18,20,29,30,31,32. The first publication in Portuguese was in 2015 as a letter to the editor 33. More studies were developed, adding new evidence over the years 34,35, but thus far scarcely more than 50 articles are retrieved in PubMed with the search term “thirdhand smoke”. Little is known about THS, and few people know of its existence and repercussions on health. This emphasizes the need to focus attention on THS and include it in scientific and public health discussions, as was done with passive smoking several years ago, among other reasons because passive smoking includes THS.

Retention of THS

The first publication showing that nicotine persists in the indoor dust of smokers’ homes dates to more than 20 years ago 36. Ten years ago, Matt et al. 37 reported that even in environments in which smokers refrained from smoking, as in children’s bedrooms, nicotine was detected in the house dust at levels five to seven times higher than in non-smokers’ homes.

A single cigarette smoked on one day in a given environment can expose many people to the smoke’s toxic compounds for days and even months 38. Exposure to THS has been proven by several studies in recent years. In a study in California (USA) published in 2011, even two months after homes previously inhabited by smokers were vacated and a month after the new non-smoking residents moved in, nicotine was detected in the air, dust, and surfaces of the furniture in the bedroom and living room (Figure 1 – for comparison, the figure presents nicotine levels in houses formerly occupied by non-smokers and the limits used in the study to discriminate between smoking and non-smoking or between environments exposed versus unexposed to cigarette smoke). There was a considerable decrease in the amount of nicotine, but the reduction did not reach the levels found in homes formerly occupied by non-smokers. The study proved not only the persistence of THS, but also the exposure to new residents: nicotine on the fingertips of children who moved into homes formerly occupied by smokers was seven times higher and urinary cotinine (the principal metabolite of nicotine) was three times higher than when the former residents were non-smokers 14.

Nicotine also accumulates in vehicles. In a truck’s cab, the front lid to the glove compartment accumulated 0.6μg/cm² of nicotine and a strip of paper exposed for three days adsorbed 1.4μg/cm² 15. In rental cars in California, even in those designated as non-smoking, nicotine was detected in the air, dust, and inside surfaces of the vehicles, clearly demonstrating the rental companies’ failure to protect non-smokers from passive smoking. Table 1 shows the amount of nicotine found in the study, published in 2013 39. For comparison, information is also shown on nicotine concentration in non-smokers’ private cars 40. Various jurisdictions have laws against smoking in private vehicles with children onboard, including England, California, Australia, and Canada, among others 41. However, since nicotine and possibly other compounds remain inside the vehicle, this measure fails to completely protect occupants from THS exposure. Although Brazil has no law that prohibits smoking inside vehicles, article 252 of the Brazilian Traffic Code specifies that it is a violation to drive with only one hand, except in specific situations like changing gears, making hand signals, or operating the vehicle’s instruments and accessories 42. Smoking while driving can also be interpreted as inattention to essential safety measures. Thus, the tacit understanding is that drivers should not smoke while driving, but the situation is not seen routinely. Besides, Brazil has no rule that applies to passengers, so there is currently no way to prevent contamination of the inside of vehicles with cigarette smoke.

A study in hotels in California in 2014 (Table 2) showed that merely restricting cigarette smoke to some environments does not protect people. Non-smoking rooms in hotels without a complete restriction on smoking showed higher nicotine levels in the air on the bedhead, and on the outside of the door when compared to hotels that were 100% cigarette-free. Guests in these rooms also showed higher levels of nicotine on their fingertips and cotinine in their urine 43. As a collective use environment, smoking in hotels, even in partially closed environments, is banned in Brazil, so a study in such an environment serves as a warning that THS is ubiquitous, since THS is not confined to the space in which the cigarette was smoked 44, but moves easily through the ventilation system, corridors, air ducts, windows, and doors. This underscores the inefficacy of designated smoking areas (banned...
Results reported by Matt et al.\textsuperscript{14} in rental homes previously occupied by smokers and rental homes previously occupied by non-smokers and later occupied by non-smokers.

<table>
<thead>
<tr>
<th>Former residents</th>
<th>New residents</th>
<th>Non-smokers</th>
<th>Cut-off *</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Smokers</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nicotine in air</td>
<td>Living room: 1.86μg/m(^3)</td>
<td>Living room: 0.2μg/m(^3)</td>
<td>Nicotine in air 0.1μg/m(^3)</td>
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<td></td>
<td>Bedroom: 1.44μg/m(^3)</td>
<td>Bedroom: 0.12μg/m(^3)</td>
<td>Nicotine on surfaces 5.0μg/m(^2)</td>
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<tr>
<td>Nicotine on surfaces</td>
<td>Living room: 98.7μg/m(^2)</td>
<td>Living room: 10.9μg/m(^2)</td>
<td>Nicotine in dust 5.0μg/g</td>
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<tr>
<td></td>
<td>Bedroom: 50.1μg/m(^2)</td>
<td>Bedroom: 7.5μg/m(^2)</td>
<td>Nicotine on fingertips: 5.19ng</td>
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<tr>
<td>Nicotine in dust</td>
<td>Living room: 39.6μg/g</td>
<td>Living room: 10.9μg/g</td>
<td>Urinary cotinine: 0.45ng/mL</td>
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<td>Nicotine on fingertips</td>
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<td>Nicotine on fingertips: 0.75ng</td>
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<tr>
<td>Uninary cotinine</td>
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<td>Uninary cotinine: 0.13ng/mL</td>
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<tr>
<td><strong>Non-smokers</strong></td>
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<tr>
<td>Nicotine in air</td>
<td>Living room: 0.02μg/m(^3)</td>
<td>Nicotine in air: 0.14μg/m(^3)</td>
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</tr>
<tr>
<td></td>
<td>Bedroom: 1.6μg/m(^2)</td>
<td>Nicotine on surfaces: 1.5μg/m(^2)</td>
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</tr>
<tr>
<td>Nicotine in dust</td>
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<tr>
<td>Nicotine on fingertips</td>
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<td>Nicotine on fingertips 50.0ng</td>
</tr>
<tr>
<td>Uninary cotinine</td>
<td>0.15ng/mL</td>
<td>Uninary cotinine: 0.13ng/mL</td>
<td>Urinary cotinine 0.30ng/mL</td>
</tr>
</tbody>
</table>

* Used to discriminate between smoking and non-smoking or between environments exposed versus unexposed to cigarette smoke.

Note: indoor measurements in homes first occupied by non-smokers were taken in the living room.

Table 1

Thirdhand smoke in rental and private cars.

<table>
<thead>
<tr>
<th></th>
<th>Non-smoking</th>
<th>Smoking</th>
<th>Private cars</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rental cars</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nicotine in air (μg/m(^3))</td>
<td>0.024 (0.008-0.063)</td>
<td>0.050 (0.018-0.086)</td>
<td>0.010 (0.000-0.010)</td>
</tr>
<tr>
<td>Nicotine on surfaces (μg/m(^2))</td>
<td>0.200 (0.000-1.200)</td>
<td>0.090 (0.300-2.900)</td>
<td>0.000 (0.000-0.070)</td>
</tr>
<tr>
<td>Nicotine in dust (μg/g)</td>
<td>8.100 (4.900-28.600)</td>
<td>33.200 (16.800-52.100)</td>
<td>3.100 (1.500-6.000)</td>
</tr>
</tbody>
</table>

Note: data presented as medians (25th percentile – 75th percentile). For smoking cars, the older the car and the greater the mileage, the higher the nicotine concentration in the dust and on surfaces.

in Brazil) in protecting non-smokers. Even the homes of smokers who do not allow indoor smoking show considerable levels of nicotine on surfaces\textsuperscript{45}. Rooms in a neonatal intensive care unit in which the infant’s caregiver was a smoker showed significant nicotine levels on surfaces of the furnishings reaching 34.2μg/cm\(^2\). These infants also presented detectable levels of nicotine metabolites in their urine\textsuperscript{46}. Persons themselves can be a vehicle for such pollutants, adsorbed on their clothing, skin, and hair and later deposited on the surfaces of other environments and causing contamination of other individuals who have not even had any contact with the cigarette smoke. This characteristic of THS has been called the “grasshopper effect”, alluding to the components’ capacity to “hop” from one environment to another\textsuperscript{47,48}. 
Non-smokers’ homes showed quantifiable levels of nicotine in the dust (median = 2.3μg/g), but ten times lower than in smokers’ homes. Smokers’ homes, even when no one smoked indoors, showed a correlation between nicotine concentration and the number of cigarettes smoked by the residents. This emphasizes that cigarette smoke’s components are released into the environment by routes other than the mainstream (smoke exhaled by smokers during puffs) and sidestream (smoke emanating from the smoldering cigarette tip). Any tobacco byproduct that generates smoke can contribute to the formation of THS. Bedrooms of children in homes where hookahs were the only form of tobacco use showed higher nicotine levels in the air and on surfaces when compared to non-smokers’ homes, even when the hookah was only smoked a few times per week or month.

Nicotine’s persistence has also been analyzed experimentally. Cotton and polyester fabrics were exposed continuously to cigarette smoke for a year and eight months, respectively, and analyzed after 11, 16, and 19 months. Considerable amounts of nicotine were only detected in the cotton fabric (105.8μg/g, 112.9μg/g, and 69.9μg/g of fabric, respectively). Even electronic cigarettes using nicotine refills increased the amount of the substance deposited on surfaces. On floor tiles, the place that accumulated the most nicotine, the amount increased from practically zero before the use of electronic cigarettes to 205μg/m² after 100 puffs. Studies are needed on electronic cigarettes, since they have been viewed by the general population and sold as less harmful and as a strategy to achieve cessation, but there is no evidence that these devices require regulation as to areas where their use is allowed, since their residues can persist in the environment and act as a source of exposure to non-smokers.

**THS composition**

Other compounds besides nicotine have been identified in THS. In rental cars and hotel rooms where smoking was permitted, levels of 3-ethynylpyridine (3-EP) in the air were higher than in cars and rooms in which smoking was prohibited. Meanwhile, levels of the substance deposited on furniture surfaces did not differ between smoking and non-smoking rooms in the hotels. The only semi-volatile amine found in the gaseous phase two hours after burning the cigarette was 3-EP, but it later disappeared (at 18 hours), probably due to the high adsorption to surfaces.

Nicotine desorbed from surfaces and reemitted into the ambient air may form secondary pollutants by reacting with atmospheric oxygen and nitrogen species. Nicotine undergoes a nitrosation from nitrous acid (HONO) to form the tobacco-specific nitrosamines 4-(methyl nitrosamine)-1-(3-pyridil)-1-butanone (NNK), N’-nitrosonornicotine (NNN), N-nitrosopyrrolidine (NPYR), and 4-(methyl-nitrosamino)-4-(3-pyridyl)-butanal (NNA). HONO is present in high concentrations in indoor air, since it results from direct emissions from combustion equipment without ventilation, cigarette smoke, and conversion of NO₂ and NO. The ready availability of agents that promote the formation of these nitrosamines is important, since NNK and NNN have carcinogenic activity. NNA is absent from recently emitted smoke, due to its high instability at high temperatures; its mutagenic activity is similar to that of NNN.
Many nitrosamines are natural components of cigarette smoke, and when they are present in THS, it is difficult to determine their precise origin, whether already existing in the cigarette smoke or formed through reactions with nicotine. Approximately 80% of nitrosamines are deposited on the surfaces of environments and are not removed by normal ventilation. NNK has been found on the surfaces of nearly all homes and cars of smokers, but is found only occasionally in nonsmokers’ homes. Ramirez et al. found that the concentration of five tobacco-specific nitrosamines (including NNN, NNK, and its metabolite 4-(methyl-nitrosamino)-1-(3-pyridyl)-1-butanol – NNAL) and eight other volatile nitrosamines was some eight times higher in the indoor dust of smokers’ as compared to non-smokers’ homes. A subsequent study detected other carcinogenic aromatic amines for the first time, with the greatest abundance of 2-aminonaphthalene and 4-aminobiphenyl in smokers’ homes. NNAL was detected in the urine of mice exposed only to THS. A study on the effect of time on the concentration of nitrosamines deposited on cotton fabric found that NNK levels increased up to 16 months and decreased three months later. The increase also occurred in the concentration of NNN, and NNA dropped from 16 to 19 months. The increase was explained by the de novo formation of nitrosamines from nicotine deposited on the material before reaching a limit and beginning to decay due to chemical transformations. De novo formation of these nitrosamines was also proven in a longitudinal experiment by the increase in concentration during months in which there was no exposure to cigarette smoke.

Other byproducts are formed by nicotine’s reaction with the ozone (O₃) from air contamination with outdoor air pollutants and the use of air purifiers. Nicotine’s reaction with ozone forms cotinine, formaldehyde, n-methylformamide, and nicotinaldehyde. In a controlled setting that used a chamber to simulate a real environment, the presence of O₃ did not lead to the reemission of nicotine to the gaseous phase, but to products of nicotine’s oxidation (N-methylformamide and miosmine). The chamber walls yielded nicotine, nicotine-1-oxide, and two organophosphates that are absent from cigarette smoke but are commonly used in flame retardant formulations.

Sleiman et al. suggested that chlorinated compounds and chloride ions could react and form chloromethane and dichloromethane, two substances that are not usually present in ETS. Pesticide residues in tobacco and in the bleaching agents used in cigarette paper are the probable sources of these chlorinated compounds.

Cigarette smoke is a significant source of exposure to polycyclic aromatic hydrocarbons (PAHs), a group of substances with carcinogenic potential. PAHs result from the incomplete combustion or pyrolysis of organic materials; there are few studies on their persistence in THS. The amount of PAHs in smokers’ hands was nearly three times that on non-smokers’ hands, and PAH levels in house dust in smokers’ homes were double those in non-smokers’ homes. There was also a positive correlation between PAH levels in the living room (where the smoker smoked) and the bedroom of a non-smoking resident, showing that these substances can be transported to environments in which smoking is not permitted. Approximately 60% of the PAHs present in cigarette smoke are deposited on surfaces in the environments.

Different types of fabric impregnated with cigarette smoke desorbed many volatile organic compounds while staying 30 minutes in a smoke-free environment. The concentration released was lower than levels typically found in secondary smoke, and compounds with greater volatility desorbed more rapidly from the fabrics (10 minutes). Like particulate matter, the substances benzene, 2,5-dimethyl-furane (2,5-DMF), and toluene are found in the air exhaled by smokers up to ten minutes after they stop smoking. Many other compounds not usually reported in ETS have been detected in laboratory studies using a machine to release cigarette smoke in a chamber, such as metacrolein, methyl-vinylketone, 2-methylfurane, 1,3-pentadiene, 1-butene, and propanonitriile. The concentration of volatile compounds taper off over time, and nicotine was only detected in the air up to 20 minutes after the flame was extinguished, and was inextinct after two hours. Meanwhile, the levels of some compounds increased, indicating reemission or reaction and de novo formation of the compounds. Eight hours after the last cigarette was smoked in the house, some pollutants were found exclusively or predominantly indoors. Therefore, acetonitrile, 2,5-DMF; 2-methylfurane, p-xylene, acrolein, benzaldehyde, metacrolein, heptane, undecane, 1-nonenone, and isoprene may be useful markers of THS.

Effects of THS on health

The risk of individuals’ exposure to THS components is still not totally clear, since there are few studies on its effects. However, the presence and persistence of toxic compounds with harmful health effects mean that the danger THS cannot be ignored. Half of the nicotine deposited on
clothing and dissolved by sweat can penetrate the body through the skin and be absorbed 71. There is no clear linear relationship between the exposure dose to THS and harmful effects 72,73.

Children are more susceptible to THS, since they spend more time indoors and breathe closer to the floor, where dust accumulates, in addition to moving objects from hand to mouth after touching contaminated surfaces 13. Children are also more vulnerable because they have a higher respiratory rate in relation to their body size, more immature respiratory and immune systems, and lower metabolic capacity 44. Previous cited studies proved the exposure of children and the environments in which they live to THS even if they are not exposed to the smoke during its emission 14,45,46,50. However, there are still few studies on the effects of THS on children’s health. According to a study in South Korea with 31,584 children 6 to 11 years of age, dry cough, nighttime cough, and coughing fits were associated with exposure to THS 74. The irritating products generated by oxidation of cigarette smoke’s components may be the principal factors responsible for these respiratory symptoms 63.

Ramírez et al. 49 estimated the risk of cancer from exposure to carcinogens in THS. The greatest risk was for children one to six years of age exposed to nicotine and others substances deposited in the dust in smokers’ homes (9.6 additional cancer cases per 100,000 children exposed). In non-smokers’ homes, the risk was one-third as high. The maximum risk of exposure to all nitrosamines from THS in smokers’ homes was one extra case of cancer per thousand persons exposed.

In a bacterial model to mimic the action of N2 and O3 on a surface impregnated with nicotine, no mutagenic effect was observed in different strains of Salmonella typhimurium (Ames test) 19. Meanwhile the genotoxic effect was observed in human hepatocellular carcinoma cells (HepG-2) exposed to THS, resulting in increased DNA strand breaks using the comet assay 72. Murine neural stem cells (mNSC) and human dermal fibroblasts (hDF) were exposed to a culture medium prepared with fabric material present inside automobiles from which THS was collected and evaluated with the comet assay. Cells treated with this material showed a higher percentage with migration of chromosomal DNA and longer tails 62. Chronic exposure to THS resulted in oxidative DNA damage in genes involved in nucleotide metabolism and repair mechanism in human bronchial epithelial cells (BEAS-2B) 72. In addition, together with NNK, NNA can induce base mutation in DNA and result in uncontrolled cell growth and thus tumor formation 35.

THS cytotoxicity was verified by altered mitochondrial function in mNSC, but the potential for damage decreased as the sample aged 62,75,76. There was even a decrease in the total number of cells, caused by inhibition of cell proliferation. THS also led to decreased growth, lower cell motility, and greater occurrence of cellular alterations, such as vacuoles, fragmentation, and cytoplasm loss 62,75. In addition, decreased cell viability, as assessed by the amount of protein, was found in fibroblasts of mice (L929) exposed to THS 71.

Even at concentrations incapable of causing cytotoxic effects, two murine male reproductive cell lines (GC-2 and TM-4), when exposed to THS, presented altered metabolite levels and gene expression of enzymes related to metabolism and oxidative stress 76. The study warned of THS action on systems that are not the prime target of cigarette smoke, but which can have their function compromised by continuous exposure. Exposure to THS also led to shorter dendritic length and altered heartbeat in zebrafish embryos 71.

Martins-Green et al. 61 and Karim et al. 73 investigated the effects attributed only to THS. Mice exposed to THS presented altered levels of triglycerides, fasting glucose, lipids, and fatty liver (steatosis), collagen production, alveolar thickness and structure including cellular infiltration, and pro-inflammatory cytokines 61, as well as increased platelet aggregation and secretion, resulting in shorter bleeding time and increased risk of thrombosis 73. Alterations in liver metabolism show potential risk for diabetes, myocardial infarction, and other cardiovascular diseases, with thrombosis as the principal mechanism in mortality from cardiovascular disease associated with smoking 18. Meanwhile, elevated interstitial collagen level, thickened walls, and presence of macrophages in the alveoli and elevated levels of pro-inflammatory cytokines confer elevated risk of developing pulmonary fibrosis after prolonged exposure to THS 63. In addition, using the nitrosamines NNK and NNA as surrogates for THS in rat lung explants led to interruption of homeostasis signaling, altering the levels of proteins that affect lung development, such as peroxisome proliferator-activated receptor gamma (PPAR-γ), fibronectin, and calponin 77.

As additional evidence of the impact of THS on health, fine particles contribute to more than 90% of the total damage caused by involuntary smoking and THS. A non-smoker living 50 years with a smoker can lose 0.3 to 7 years of life from inhaling toxic compounds from THS. Even if both individuals spend most of their time away from home, non-smokers can lose 0.3 to 4.1 years of life due to this exposure. Depending on the time
transpired until the transition to THS, 5% to 60% of the total damage caused by exposure to cigarette smoke pollutants in the environment can be attributed exclusively to THS.

Behavioral disorders are more common in children exposed involuntarily to cigarette smoke, while the impact of THS on behavior has received little research. Animals exposed to THS displayed hyperactivity-type behavior, running longer and faster in the open field test than unexposed animals. In addition, children exposed to THS in the home which reacted positively to cigarette smell from objects or persons (in other words, children that felt pleasure or euphoria or simply liked the smell) were more prone to smoke a cigarette if a friend offered one. Thus, reactions to THS and the tendency to smoke can be socially influenced and act as a risk factor for smoking initiation.

Making the danger visible

Although viewed with skepticism by some, a national telephone survey in the United States published in 2009 found that 61% of nearly 1,500 adults acknowledged THS as harmful to children, while 22% had no opinion. Another study in private pediatric offices in various counties in the United States from 2009 to 2011 found that 91% of parents who smoked believed that THS could harm their children. In low-income communities in Georgia (USA), more individuals were worried about THS harming their children than involuntary smoking causing heart diseases. Hispanics living in residential buildings in California were unfamiliar with the term THS, while admitting that although they banned smoking in their own homes, they were unable to effectively avoid exposure to cigarette smoke coming from their neighbors. The problem of contamination of multiunit dwellings like apartment buildings, condos, and row houses is a challenge for any tobacco control policy.

Smokers who believed that THS could be harmful to children's health were significantly more prone to adopt measures to restrict smoking in their homes and vehicles. Discussion with smokers on the effects of THS can provide a further reason for encouraging smoking cessation or adopting procedures to prevent exposure of non-smokers to cigarette smoke and THS. Concern for one's own health is generally reported as the main reason for smokers to try quitting, but a review that included studies spanning more than 30 years found that smoking's effect on family members was a frequent reason for attempting to quit.

Concern over the effects of THS on children's health is particularly important. Children have age-specific physiological characteristics, as discussed above, and limited autonomy in relation to their immediate surroundings (when family members smoke in their presence they have no way to avoid exposure to the smoke). They are thus the most defenseless population vis-à-vis the effects of cigarette smoke and thirdhand smoke. One of the Sustainable Development Goals (SDGs) of the United Nations Development Program (UNDP) is to ensure children's quality of health, which includes decreasing infant mortality. Reducing involuntary exposure to cigarette smoke helps meet this goal, since strong evidence shows that exposure to smoke increases infant mortality. Strengthening implementation of the FCTC is also one of the SDGs approved by the UNDP, and the two goals' convergence emphasizes the importance of on-going research on THS in children to generate evidence allowing Brazil and other countries to further strengthen the struggle against the tobacco epidemic and thus achieve healthier living.

Since Brazil has no law against smoking in people's own homes, raising smokers' awareness of the risks of THS to their families may be an effective strategy for smokers to refrain from smoking in areas shared by their non-smoking family members. In fact, among light smokers there was an important decline in passive smoking from 2008 to 2013, suggesting that smokers who continue to smoke are considering social disapproval of smoking as a reason for quitting. It is unlikely or even impossible (for ethical reasons involving individual freedom) that specific legislation will be created to ban the use of tobacco products in private residences; therefore, education and awareness-raising on the effects of THS may be useful strategies. The impact of THS on health, and especially that of children, could be the theme for campaigns to reach individuals concerned about their family's welfare.

Final remarks

Thirdhand smoke is not harmless. The few studies on its effects serve as a warning that contaminated fabric and surfaces have the potential to cause adverse health effects, even when there is no immediate contact with the smoke. The risks of THS are still unknown to the population at large, so mobilization of public opinion to better understand its harmful effects should be part of tobacco control programs.

Even ventilation and air cleaning systems are ineffective in preventing exposure to the com-
ponents of cigarette smoke, so the only way to eliminate non-smokers’ exposure to passive smoking is to totally prevent active smoking in closed spaces. Despite progress in decreasing the proportion of smokers and behavior changes among the remaining smokers in Brazil, which has resulted in a reduction in passive smoking, it is still a challenge for tobacco control policies to protect all non-smokers from involuntary exposure to smoke and THS. Fulltime 100% tobacco-free environments are thus needed, adopting restrictions on smoking to protect non-smokers in private residences and environments. Meanwhile, many of the components of THS are carried from one place to another by smokers themselves, which may make it impossible to guarantee complete absence of exposure to non-smokers, so awareness-raising on the harms caused by THS could be used clinically to encourage smoking cessation.

In short, while there is scientific evidence on the harms of involuntary smoking for individuals’ health, and there is no way to deny the existence of THS, little is known about this pollutant’s mechanisms of action, as a whole or for its components. Not all of the components have been identified. The potential effects of THS on individual and public health highlight the need for more research to prove or disprove such risks and encourage an evidence-based debate.

Contributors

L. R. Figueiró, A. L. Ziulkoski and D. C. M. Dantas contributed substantially to the paper’s conception and planning and critical revision of the content, and revised and approved the manuscript’s final version.

References


Resumo

Recentemente, passou a ser discutida mais uma forma de exposição ao tabaco – thirdhand smoke (THS) – que consiste nos poluentes residuais da fumaça de cigarro que permanecem nos ambientes. A principal preocupação com o THS é embasada na presença e longa persistência de muitos compostos tóxicos, algumas nitrosaminas específicas do tabaco que têm atividade carcinogênica. Além de se saber pouco sobre o THS, poucos sabem de sua existência e preocupante repercussão na saúde. Coloca-se em destaque a necessidade de trazê-lo à luz e incluí-lo nas discussões, assim como foi feito com o tabagismo passivo alguns anos atrás, até mesmo porque o THS se caracteriza como uma forma de tabagismo passivo junto à exposição secundária ou involuntária da fumaça de cigarro.

Contaminación por Humo de Tabaco; Tabaco; Fumaça

Resumen

Recientemente comenzó a ser discutida otra forma de exposición al tabaco – thirdhand smoke (THS) – que consta de contaminantes residuales de humo de cigarrillo que permanecen en el medio ambiente. La principal preocupación con la THS se basa en la presencia y larga persistencia de muchos compuestos tóxicos, como algunas nitrosaminas específicas que tienen actividad cancerígena. Pocos saben de la existencia de los THS y su impacto preocupante en la salud. Se plantea la necesidad de sacarlo a la luz e incluirlo en las discusiones, como se hizo con el tabaquismo pasivo hace unos años, incluso porque el THS se caracteriza como una forma de tabaquismo pasivo por la exposición secundaria o involuntaria de humo de cigarrillo.

Contaminación por Humo de Tabaco; Tabaco; Humo