Impact of Household Tobacco Smoke Exposure on Childhood Wheezing.

**Corresponding Author:**
Dr. Sukhbir Shahid,
Consultant Pediatrician, Pediatrics - India

**Submitting Author:**
Dr. Sukhbir Shahid,
Consultant Pediatrician, Pediatrics - India

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Author(s): Shahid S

Abstract

Objectives: Childhood wheezing is a multifactorial chronic airway disorder. It is more prevalent amongst those exposed to tobacco smoke at home. Does this passive tobacco smoke exposure prepond or increase severity of wheezing in children?

Study design: Prospective, descriptive study

Methods: In order to determine this, we carried out an analysis of wheezing children attending our clinic. The mean age of presentation and severity of wheeze in passive smokers was compared with those in non-passive smokers. Impact of type of tobacco unit smoked and its amount on age of first wheeze and wheeze severity were determined. Influence of tobacco smoke exposure on the above two parameters after controlling for family history of atopy was evaluated.

Results: 263 (43.8%) patients had confirmed history of exposure to household tobacco smoke. Age of presentation and severity of wheeze in passive smokers did not differ significantly from that in non-passive smokers. However, passive smokers from atopic families were not only significantly younger but also had more cases of moderate to severe wheeze compared to the wheezing non-exposed children from non-atopic families (1.5±0.22 vs. 2.25±0.30 years respectively, p<0.05); though their asthma severity was similar.

Conclusions: Genetically predisposed children had early onset and severe wheeze with household tobacco smoke exposure. Proper environmental control for such pollutants in early life would aid in curtailing the likelihood of development of wheeze in them.

Introduction

Childhood wheezing is a multi-causal chronic airway ailment. Its incidence is on the rise[1, 2]. Annual health care expenditure and loss of school days due to it is enormous, and it causes significant morbidity and mortality[1, 3]. Household indoor pollutants play a major role in its causation. Of these, household secondhand tobacco smoke exposure is a serious contributing factor, especially in young children[4-7]. Household secondhand tobacco smoke exposure, or passive smoking or involuntary smoking, is defined as exposure of a nonsmoking person to tobacco combustion products (exhaled or side-stream) from smoking by others[8]. Tobacco smoke comprises of around 4000 different chemical substances including a range of potent irritants, allergens, carcinogens, mutagens and toxins[9-13]. There is compelling evidence that passive smoking induces wheezing and asthma in children[14, 15]. Numerous large-scale studies and meta-analysis have documented significant relations between parental smoking and asthma development in children[16, 24]. Spirometric measurements were also found to be lower in passive smokers compared to non-passive smokers[25]. Childhood wheeze was found to be directly related to number of household smokers, number of cigarettes smoked per day, and duration of exposure [15, 16, 26, 27], with the strongest effect being detected in the youngest children [23, 28-30]. Maternal smoking seems to have a greater effect than other household members’ smoking[31, 32]. It forms a risk factor for wheezing throughout childhood[31, 33]. Gilliland FD et al found that prenatal passive smoking leads to physician-diagnosed asthma whereas postnatal passive smoke acts only as a trigger for wheeze with intercurrent infections, without inducing asthma[34]. In known asthmatics, passive smoking is known to precipitate attacks[9, 20]. It causes a significant reduction in FEV1/VC in them[35] and increases emergency room visits but not hospitalizations[36]. Studies of effect of smoking on severity and age of onset of wheeze have however shown variable results [23, 37-40]. We tried to analyze medical records of wheezing children following up with our clinic in order to determine impact of passive smoking on severity and age of onset of wheeze in these children.

Subjects and methods

We studied wheezing children 0-12 years of age attending our clinic. Questionnaire-based assessment was done to determine tobacco smoke exposure at home. The socio-economic status, type of tobacco...
smoke exposure (bidi and/or cigarettes), number of family members smoking at home and average number of tobacco units smoked per day was noted down. Coincident tuberculosis, if any, was looked into. Presence of atopy in near family members was inquired into. The patients were grouped into those with history of exposure to tobacco smoke at home, and those which denied such an exposure. The severity of wheezing illness and age of onset of wheeze in these children were evaluated and compared in both groups for statistical significance, if any. Wheeze severity was graded as per Global Initiative on asthma (GINA) guidelines. Patients were either mild intermittent, mild persistent, moderate persistent or severe persistent wheezer[41]. Impact of quality and quantity of passive smoking on age of onset and severity of wheeze was determined. Severity of wheeze in tobacco-exposed wheezing children from atopic families was compared with that in non-tobacco-exposed children from non-atopic families, as well as with, tobacco-exposed children from non-atopic families. The mean age of onset of wheeze in these three subgroups was also compared and tested for statistical significance, if any.

**Data analysis:** All data are expressed as means ± SEM. Comparison of demographic data was done by Chi-square test. Continuous data of two subgroups were tested for significant difference by unpaired Student's t-test for normally distributed data. Significance was considered when p < 0.05. Odds ratio was calculated with 95% confidence interval[42].

**Results**

600 wheezing children formed our study group. The mean age of these children was 4.84 years ± 0.14 (range 4 months to 12 years of age). 257 (42.8%) were females; male to female ratio being 1.3:1. All these children belonged to lower and lower middle socio-economic strata of society. The mean age of onset of wheeze was 2.09 years (±0.10). Three hundred and twenty four (54%) of these wheezing children had their first wheeze before their first birthday, 86% wheezed before age 5. Eight (1.3%) were mild intermittent wheezers, and 91 (15.17%) were mild persistent wheezers. Moderate and severe persistent wheezers comprised 55.67% (n=334) and 27.83% (n=167) respectively of these wheezing population. 263 (43.8%) of the children reported household tobacco smoke exposure. The active smoker was the father in 248 (94.30%) of the cases, and uncle and/or grandfather in 51 (19.39%) of the cases. 36 (13.69%) had 2 or more relatives smoking at home. There was no case of exposure to maternal smoking. 114 (43.35%), 98 (37.26%) and 51(19.39%) of these tobacco-exposed wheezing children had relative/s who smoked bidi or beedi or biri, conventional cigarette or both at home, respectively. Bidi or beedi or biri is a leaf- and hand- rolled cigarette made of low-grade, coarse and uncurbed tobacco popular among the rural folk and urban poor of South Asia [43]. The tobacco units consumption was <10/day by one or more active smokers at home in 239 (90.87%) of these children. Simultaneous tuberculosis was present in 53(15.73%) of non-passive smokers and 51(19.39%) of passive smokers respectively (p>0.05, odds ratio=0.78, 95% confidence interval of 0.51 to 1.19). Past or current TB was seen in 74 (21.96%) and 71 (26.99%) of non-tobacco-exposed and passive smokers respectively. (p>0.05, odds ratio of 0.76, 95% confidence interval of 0.52 to 1.11). The mean age of onset of wheeze in those who reported exposure to household tobacco was 2.05 ±(0.16) years, whereas it was 2.13 ±(0.14) years in those who had no history of such exposure; p value =0.70. Moderate to severe wheezers were not significantly different in the non-exposed and exposed groups (238 vs. 186 respectively, p=0.98, Odds ratio=1.00; 95% confidence interval of 0.70 to 1.43). The mean age of onset and severity of wheeze did not differ significantly between non-tobacco-exposed group, and each of the subgroup exposed to bidi, cigarette or both. Children exposed to more than 10 tobacco units per day were neither significantly younger nor had more severe wheeze than the non-tobacco-exposed children with wheeze. However, passive smokers with positive family history of atopy were younger and had more severe wheeze compared to wheezing children from non-smoking, non-atopic homes (Table 1); with odds ratio of having severe wheeze in passive smokers from atopic families being 1.91, 95% confidence interval of 1.00 to 3.64. In children who were tobacco-exposed, type of tobacco unit exposed to did not affect the age of onset or severity of wheezing illness (Table 2). Neither was age of onset or severity of wheeze affected by the number of tobacco units exposed to per day. The severity did not vary in passive smokers from atopic homes from that in passive smokers from non-atopic homes (p>0.05, Odds ratio 1.57, 95% confidence interval of 0.80-3.06). Age of onset of wheeze was however significantly less in passive smokers with positive family history of atopy compared to passive smokers with no such history (1.50±0.22 vs. 2.25±0.30 years respectively, p=0.03).
Discussion

The aim of this study was to evaluate the impact of environmental tobacco smoke exposure on severity and age of onset of childhood wheezing. We found that genetically predisposed children had earlier onset of and more severe wheeze compared to non-exposed wheezers from non-atopic families. This is the first study of its kind reported from Mumbai, India. We used medical records of wheezing children attending our clinic to study impact of passive smoking on childhood wheezing. 600 wheezing children formed our study group. We based our study on historical details and clinical examination. Measurement of cotinine, the major metabolite of nicotine of tobacco, in body fluids such as serum, urine, and saliva has been shown to be more reliable, accurate, sensitive and objective method of assessing tobacco smoke exposure [19, 44-46]. The cotinine levels in body fluids have been found to be high in asthmatics and passive smokers, irrespective whether they have wheeze or not[46, 47]. The levels correlate well with the quantity of reported exposure and also with respiratory function[48]. We could not use the biomarker cotinine in our study due to the cost factor and lack of availability of this test in our setup. The detailed questionnaires used in our study have also been shown to give valuable information with less cost[49]. Besides, cotinine levels with half-life of 16 h indicate recent, but not remote exposure and may not be valid when long-term exposure is to be ascertained[40]. It also gives us no idea about the duration of exposure or about inhalation of components of tobacco smoke which may be more important than nicotine[50]. We were unable to evaluate effect of maternal smoking on childhood wheezing in our study as we had no case of maternal smoking. In our study, a significant proportion of children were exposed to bidi smoke. Bidi is a traditional, inexpensive method of tobacco use popular in south Asia and parts of Middle East [43, 51]. It is also called as ‘cigarette with training wheels’ or ‘poor man’s cigarette’. It consists of sun-dried and processed tobacco rolled manually in tendu leaf (Diospyros elanoxylon) or temburni leaf (Diospyrus ebenum), and held together by a cotton thread[52]. The relatively low combustibility and non-porous nature of the tendu leaves requires more frequent and deeper puffs by the smoker to keep bidis lit, and is therefore harder on the smoker’s lungs than cigarettes rolled in paper[53]. Bidi produces 3 times more nicotine and carbon mono-oxide and 5 times more tar compared to cigarettes[54]. There is no published account of worsening of wheeze with bidi secondhand smoke, but considering the biological plausibility these effects are likely with bidi similar to that with cigarette secondhand smoke exposure[52]. There has also been no study to date which evaluated the clinical difference between bidi and cigarette secondhand smoke exposure. In our study, we have tried to assess the difference in age of first wheeze and wheeze severity in patients exposed involuntarily to cigarette and bidi smoke, and found no difference in them in both groups. Increased prevalence of tuberculosis has been noted in active adult smokers, particularly with bidi use [52, 55, 56]. But impact of secondhand smoke exposure on incidence of tuberculosis (TB) in children had not been studied to date. We tried to correlate secondhand smoke exposure with clinical TB and found no increase in TB cases amongst those exposed to tobacco smoke. We used GINA guidelines to grade wheeze severity. This is primarily a symptom-derived severity. Other authors have employed other methods, including objective parameters such as lung function tests [57, 58]. We did not have pulmonary function tests in our patients. We also did not have any serum immunoglobulin E levels, skin prick or allergen test reports in our patients and hence could not determine the influence of patient’s atopy with passive smoke on age of first wheeze and its severity in these patients. Wheezing and asthma has been known to have a slight male predominance[59]. Similar findings were seen in our study. In our study, 54% of the children had their first wheeze before the age of 1 year. 86.00% wheezed before their 5th birthday. Wafula EM et al also found that more than 50% of their wheezing children were less than 12 months at the time of first wheeze[38]. Mild to moderate wheezers comprised majority of our study group. Similar results were reported by other authors [38, 60, 61]. We found that 43.8% of our wheezing patients were exposed to tobacco smoke. Population-based studies however have pegged this figure as being more than 50% [25, 62]. The lower percentage in our study could be due to regional differences, or due to awareness-induced quitting of smoking by some parents of wheezing children, or due to deliberate hiding of facts by some parents fearing rebuke by physicians as regards their smoking habits. Maternal smoking is a rare phenomenon in India. Hence we could not find any child with history of maternal smoking in our study. This was unlike the European study where exposure to maternal smoking is a common phenomenon. Our study could find no effect of passive smoking on presentation of first wheeze or severity of wheeze in children. A number of studies have studied effect of parental smoking on
severity of childhood wheeze [36, 57, 63-73]. But different approaches have been employed to gauge wheeze severity. Hence formal meta-analysis is not possible[23]. Wafula EM et al have shown that age of onset of wheeze is earlier in passive smokers and also passive smoking possibly augments severity of wheeze in those children[38]. Similar results were reported by other authors [50, 69]. Cook et al have shown that severity of wheeze was increased in children with exposure to household passive smoke [16]. Mannino DM et al also found that involuntary tobacco smoke exposure increases wheeze severity and worsens lung function [40]. The variation in our study could be due to deliberate or non-deliberate under-reporting of tobacco smoke exposure by some parents[74]. Hence exposed children might have been falsely classified under the non-exposed group[75]. Also children with past and not current tobacco exposure might have been misclassified as non-exposed. Besides, children in the 'unexposed' category can have exposures from non-parental sources or in places other than the home [40, 50, 76, 77]. It could also be due to children from non-exposed homes being exposed to tobacco smoke from neighboring households given to understand the congested conditions in which the patients resided. Crombie et al used health service contacts as proxy for wheeze morbidity and found a weak U-shaped relationship between health service contacts and salivary cotinine levels with moderate cotinine level being associated with fewer health service contacts [78]. This was attributed to lack of awareness of asthma symptoms among heavy smokers or a reluctance to visit the general practitioner. We could not corroborate our historical data of exposure with objective biological markers of passive smoking such as cotinine levels in body fluids due to cost factor and non-feasibility of test in our setup. Considering the fact that bidi is a crude form of tobacco, exposure to its smoke should have been associated with more severe and early wheeze. But we could not find this in our study. Various studies have revealed a dose-related effect of passive smoking on childhood wheeze [16, 32, 50, 79]. In our study, we found no significant difference in age of onset or severity of wheeze in children exposed to < or > 10 tobacco units per day. We found that passive smokers from atopic homes had earlier and more severe wheeze compared to non-passive smokers from non-atopic homes. Similar findings have been reported by Strachan DP et al who found that asthma was more severe in passive smokers from atopic homes[23]. This implies that genetically susceptible children have a tendency to early and severe wheeze. Hence environmental control for inducing/provoking factors should be aimed in order to avoid the likelihood of developing asthma in them. Recent studies have revealed that tobacco smoke generates more endotoxin [80, 81]. Exposure to this endotoxin could possibly incite formation of immunoglobulin E and thus affect the likelihood of development of subsequent atopy [14]. Several studies have shown that serum IgE is more with passive smoking [82, 83], especially in females [84]. However, Janson C et al found no significant correlation between passive smoking and total serum IgE[62]. Magnusson also found that maternal smoking only appeared to lead to a transitory rise in IgE in infants but this was not associated with atopy or disease at 7 years of age [85]. Meta-analysis by Strachan DP et al also revealed that parental smoking is unlikely to lead to allergic sensitization[86]. We did not measure immunoglobulin E levels in our patients due to non-availability and cost of this test in our setup. Hence correlation between their levels and wheeze parameters and passive smoking could not be done. Interleukin-10, an anti-inflammatory protein which seemingly protects against asthma and allergies, is low in infants exposed to tobacco smoke [87]. We were unable to perform this assay in our study and hence could not correlate it with passive smoking. This study is the first of its kind in Mumbai, India. It has its potential limitations, but it has shown beyond doubt that genetically predisposed children are prone to early and stronger wheeze. Environmental tobacco smoke not only induces wheeze but also has a causal role in poor overall control of symptoms. Genetically susceptible children should avoid contact with involuntary smoke in order to avert development and worsening of wheezing symptoms.

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Competing interests: None

Ethical approval: None to declare because it was an observational study with no active interventions done other than that recommended.

References

Chest 1987; 91 (suppl): 65S-74S


56. Kolappan C, Gopi PG. Tobacco smoking and...
pulmonary tuberculosis. Thorax. 2002; 57: 964-966
57. Murray AB, Morrison BJ. Passive smoking by asthmatics: its greater effect on boys than on girls and on older than on younger children. Pediatrics 1989; 84: 451–459
70. Murray AB, Morrison BJ. The decrease in severity of asthma in children of parents who smoke since the parents have been exposing them to less cigarette smoke. J Allergy Clin Immunol 1993; 91: 102–110
75. Benowitz NL. Cotinine as a biomarker of environmental tobacco smoke exposure. Epidemiol Rev 1996; 18: 188-204
78. Crombie IA, Wright A, Irvine L, Clark RA, Slanec PW. Does passive smoking increase the frequency of health service contacts in children with asthma? Thorax 2001; 56: 9-12
84. Oryszczyn MP, Annesi-Maesano I, Charpin D, Paty E, Maccario J, Kauffmann F. Relationships of active
and passive smoking to total IgE in adults of the Epidemiological Study of the Genetics and Environment of Asthma, Bronchial hyperresponsiveness, and Atopy (EGEA). Am J Respir Crit Care Med 2000; 161: 1241-1246
85. Magnusson CGM. Maternal smoking influences cord serum IgE and IgD levels and increases the risk of subsequent infant allergy. J Allergy Clin Immunol 1986; 78: 898–904
Illustrations

Illustration 1

Table 1

Table 1: Impact of type of tobacco exposure and family atopy on mean age of onset and severity of wheeze in passive smokers

<table>
<thead>
<tr>
<th>Groups (n)</th>
<th>Mean age of wheeze onset (y)</th>
<th>p value</th>
<th>moderate-severe wheezers (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>-Non-passive smokers (337)</td>
<td>2.13 ± 0.14</td>
<td>0.70</td>
<td>70.72</td>
<td>0.98</td>
</tr>
<tr>
<td>-Passive smokers (263)</td>
<td>2.05 ± 0.16</td>
<td></td>
<td>70.92</td>
<td></td>
</tr>
<tr>
<td>-Non-passive smokers (337)</td>
<td>2.13 ± 0.14</td>
<td>0.59</td>
<td>70.72</td>
<td>0.42</td>
</tr>
<tr>
<td>-Bidi-exposed passive smokers (114)</td>
<td>2.28 ± 0.24</td>
<td></td>
<td>74.56</td>
<td></td>
</tr>
<tr>
<td>-Non-exposed from non-atopic families (238)</td>
<td>2.28 ± 0.18</td>
<td>0.025</td>
<td>68.07</td>
<td>0.04</td>
</tr>
<tr>
<td>-Passive smokers from atopic homes (71)</td>
<td>1.5 ± 0.22</td>
<td></td>
<td>80.28</td>
<td></td>
</tr>
</tbody>
</table>
Table 2: Influence of type and daily amount of tobacco unit exposed to on mean age of onset and severity of wheeze:

<table>
<thead>
<tr>
<th>Parameter(n)</th>
<th>Mean age of wheeze onset (y)</th>
<th>p value</th>
<th>moderate-severe wheezers (%)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco type:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-Bidi(114)</td>
<td>2.28±0.24</td>
<td>&gt;0.05</td>
<td>85(74.56)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>-Cigarette(98)</td>
<td>1.90±0.26</td>
<td></td>
<td>70(71.43)</td>
<td></td>
</tr>
<tr>
<td>-Both(51)</td>
<td>1.80±0.32</td>
<td></td>
<td>32(62.75)</td>
<td></td>
</tr>
<tr>
<td>Daily amount of tobacco units exposed to:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-&lt;10(239)</td>
<td>1.92±0.24</td>
<td>&gt;0.05</td>
<td>165(69.04)</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>-&gt;10(24)</td>
<td>2.10±0.16</td>
<td></td>
<td>17 (70.83)</td>
<td></td>
</tr>
</tbody>
</table>
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