Respiratory Symptoms in School Children and the Role of Passive Smoking

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A study of respiratory symptoms in 1,987 school children aged 6-11 years was carried out in Suginami, Tokyo in 1976 and the response rate was 99.5%. The prevalence rate for symptoms was associated with their families' smoking habits and their residential conditions; the prevalence rate was the highest among heavy-smoker families living in the area along a main highway (within 50 meters), and the lowest in non-smoker families regardless of their residential areas. Furthermore, if they lived in an area well away from a main highway (over 100 meters), the prevalence rate was also the lowest regardless of the families' smoking habits. Relative risk in the highest group was 4.0.

Previous studies have already suggested that there was a relationship between symptoms and passive smoking while some have insisted that there was no relationship between them. This study may be useful in solving such contradictory problems and in establishing the role of passive smoking in respiratory symptoms.

(Key Words: Asthma, Passive Smoking, Air Pollution)

INTRODUCTION

The onset of unspecific respiratory diseases such as asthma in school children is considered to be caused or induced by a predisposition accompanied by all kinds of host, environmental and agent factors (3, 5, 6, 8, 13, 15, 16, 28, 29, 30).

Among these factors, when atmospheric air pollutants are examined in relation to asthmatic respiratory symptoms, sulfur dioxide, nitrogen dioxide and suspended particulates, the main action of which is stimulative, are considered to induce bronchial mucous membrane edema, bronchial muscle cramps, increased secretion and asthmatic respiratory symptoms. Therefore, such mechanisms are distinguished from those caused by antigen-antibody reactions of antigenic substances such as pollen. In addition (28, 29), actions other than stimulative action have been reported to damage the mucous membrane lining the airways so that antigenic substances might enter, to promote histamine or a modicum of other active substances to extricate from the airways directly without depending on an antigen-antibody reaction, to raise the histamine sensitivity of a child or to reduce the protective function against infection.

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All of this recent research (12) indicates that atmospheric substances such as sulfur dioxide and nitrogen dioxide, which are not able to become allergens themselves, not only stimulate the airways but also promote airway obstruction when an allergen exists, which consequently leads to an increase of asthmatic attacks. This shows that atmospheric air pollution promotes attacks in persons with latent predisposition through it's effect on airway sensitivity (27).

In other words (9, 17), SO₂ and NO₂ increase the possibility of an attack in a person loaded with hereditary factors or turn a latent patient into a conspicuous one. Kagawa (12) noted that when he looked into the problem of the frequent occurrence of asthma in Yokkaichi, Yokohama, and New Orleans which are notorious for air pollution, it could not be presumed that a person not having predisposing factors would have an attack of asthma.

Many epidemiologic investigations concerning the effects of air pollution on the human body by the BMRC method selecting SO₂ as an independent variable and symptoms of chronic bronchitis as a dependent variable have shown that prevalence rates rise with increasing levels of air pollution. Sulfur dioxide affects the comparatively upper part of the airways and chronic bronchitis is accompanied by spasms and paralysis of the ciliated epithelium, hypertrophy of the mucous gland and excessive secretion. In other words, because chronic bronchitis is caused by a lesion in the area where SO₂ is supposed to cause trouble, it is natural to expect some co-relation between both variables in order to measure the effect of atmospheric air pollution by SO₂ on the human body.

Although there have been few epidemiologic investigations concerning the effect of NO₂ on the human body, including the "Chattanooga Study" by Shy (20), two studies sponsored by the Japan Environment Agency (19, 21) and three studies reported by R. Yoshida (31), Tsunetoshi (26) and Tsubota (25), this results from the fact that there are no districts polluted only by NO₂. Since NO₂ affects the lower airways where there is neither ciliated epithelium nor mucous glands, Tsunetoshi (26) points out that, in those in the upper age brackets, selecting chronic bronchitis as a dependent variable might mean that the role of NO₂ is underestimated.

Accordingly, an epidemiological investigation concerning the actual effect of air pollution on the human body, including not only SO₂ and suspended particulates but also NO₂ requires the selection of a disorder based on a change in the peripheral airways on which NO₂ has an effect as a dependent variable.

An investigation utilizing the so-called asthma symptoms, which include regular bronchial asthma and asthmatic bronchitis, or recurrent lower respiratory diseases as a variable should be carried out and investigations using only the symptoms of chronic bronchitis as an index should not be relied on. Especially when observing the effect on air pollution on school children, it is preferable to restrict the investigation to the prevalence rate of asthmatic respiratory symptoms because chronic bronchitis and pulmonary emphysema are comparatively uncommon among such subjects.

When the prevalence rate in a group of school children is observed
regionally, it is assumed that allergens which bring about an attack or induce an endocrine disorder, a mental disorder, fatigue, a change of weather and other factors are distributed almost uniformly in each district, and it is possible to regard the prevalence rate which gives an overall reflection of such factors as the basic value for the district.

If specific factors are added to a specific small area and sub-group of that district, a new prevalence rate which exceeds the basic value can be expected to appear for such specific small areas and sub-groups. Of course when the factors added are air pollutants such as SO₂, it goes without saying that as long as the level in those small areas and sub-groups remain below the threshold value, the prevalence rate naturally does not differ from the district's own basic value.

The district investigated in this work is a typical residential area in the Tokyo Metropolis except for partial commercial zone, where there are no fixed sources with any great effect on the air pollution and generally speaking the values of SO₂, NO₂ and CO (7, 10, 11) are far below the environmental standards in the U.S.A. but the value of NO₂ is over the relieved new environmental standard in Japan which was revised in 1978. Moreover, there is no possibility of an inhalant antigen of pollen or mites and mold in house-dust, a dietary antigen or a medical antigen being added in high concentrations to any specific small areas or sub-groups in the district investigated. With respect to air pollutants, most of the SO₂ is based on environmental atmospheric SO₂. Even though there is SO₂ added from diesel automobile exhaust and smoke from smoking, the quantities can be assumed to be extremely small. However, the proportion of NO₂ from automobile exhaust added to the areas along the main highways and from smoking in families is assumed to be rather large, when compared with the NO₂ concentrations in the environmental atmospheric air as a background.

This investigation was concerned with the relation between air pollutants and the prevalence rate primarily in school children, taking automobile exhaust in the areas along the main highways as a factor added to a specific small area and so-called passive smoking in families, use of non-ventilated type stoves, ventilation deficiencies due to the house structure and the history of inhabitation as additional factors in a specific sub-group.

MATERIALS AND METHODS

This investigation was carried out on 1,937 school children aged 6 - 11 years attending two schools in Suginami Ward, Tokyo Metropolis. (916 children in A school and 1,021 children in B school, as of May, 1975)

The first screening was carried out in May, 1957, and the second was performed in October of the same year.

The first screening by questionnaire:

Health examination papers concerning asthmatic respiratory symptoms were distributed through the school to all children's families, asking for completion by their parents. The response rate was 99.5%.

Questions in the health questionnaire were designed to screen asthmatic symptoms by certain criteria including a combination of wheezing, breathlessness and their recurrences, and a history of asthma. They were
based on questions devised by Yoshida (23, 24) of Chiba University to survey the prevalence rate of bronchial asthma in school children.

Health Questionnaire,
Please answer the following questions:
1. Have you ever complained of difficulty in breathing because of wheezing during the last two years?
   a. Yes  b. No
2. Have you ever complained of difficulty in breathing because of wheezing during the last two years?
   a. Yes  b. No
3. Have you ever had trouble sleeping because of wheezing and orthopnea at night during the last two years?
   a. Yes  b. No
4. How many times did you have such symptoms (wheezing or difficulty in breathing) as mentioned in the above three questions?
   a. No time  b. one time  c. two times  d. more than three times
5. Have you ever been diagnosed to be asthmatic by a physician?
   a. Yes  b. No
6. Those who have been diagnosed to be asthmatic are to answer the following questions.
   (if you answered "No" to question No. 5, you may omit answering them)
   a. How long have you been asthmatic?
      From about _______ years and _______ months old
      To about _______ years and _______ months old
      Present age _______ years and _______ months old
   b. What were the symptoms?
      (a) wheezing.
      (b) wheezing and difficulty in breathing.
      (c) having trouble sleeping because of wheezing, difficulty in breathing and panting.
      (d) please add any other symptoms you had.

Here, a case is defined as follows:
1. One who answered, "Yes, I have been diagnosed to suffer from asthma by a doctor. "to question (5) and had attacks during the past two years.
2. One who answered, "Yes, I had wheezes and whoops." to question (1) and had recurrences more than three times during the past two years, even if he had no doctor's diagnosis.
3. One who answered, "Yes, I had both wheezes and difficulties in breathing." to question (2) and had recurrences more than two times.
4. One who answered, "Yes, I had wheezes, difficulties in breathing and orthopnea." to question (3).

As it is clear from these questions, in addition to the usual bronchial asthma, cases in this investigation may suffer from asthmatic bronchitis such as recurrent bronchitis and allergic bronchitis, or recurrent upper respiratory infections as the preceding stage of asthma.
The second screening by interview;

The second screening of a total of 204 children, 98 from A school and 106 from B school, selected as cases in the first screening was performed by selecting the cases again, ascertaining their histories of inhabitation, family and allergic diseases and confirming conditions of wheezing, breathlessness and recurrences by interviews with a parent, about ninety of whom were mothers. However, the method of interviewing a parent sometimes made it impossible to distinguish between allergic bronchitis and asthmatic bronchitis which are accompanied by a slight breathlessness and repeated recurrences from bronchial asthma.

Accordingly, the prevalences here indicated patients suffering from recurrent lower airway diseases which include some asthmatic bronchitis in addition to bronchial asthma. However, no chronic bronchitis or pulmonary emphysema could be found.

The report by Yoshida (23, 24) gives classification I for bronchial asthma and II for recurrent bronchitis and allergic bronchitis. Therefore, our cases correspond to I plus II. Incidentally, according to the results obtained by Yoshida (24) (1975), the prevalence rate of II in eight schools in the Tokyo Metropolis was around 1% on the average. The lung function test, measurement of physical fitness, biochemical blood tests, X-ray examination and H.I. tests against various infectious diseases were also carried out on the cases found by the first screening, but the results are omitted in this report.

Classification of districts and school children’s families:

The district used in this investigation is a typical residential area, except for a partial commercial zone, and no specific establishments causing air pollution are located there. However, three main highways each with a traffic volume of 40,000 – 80,000 cars a day, pass through the district, so that the air pollutional effects of automobile exhaust can not be ignored in the areas along these main highways. Therefore, the district was divided into three parts: one was the area within 50 meters of the main highway, the second was within 100 meters and the third more than 100 meters away.

The basic level of atomospheric air pollution in the district was estimated on the basis of the results of monitoring stations set up in A school and Kugayama (10, 11). The local level of air pollution in the area within 50 meters of the main highway was estimated based on the results of some special measurements by the local government (Suginami Ward) (10, 11).

The former was estimated as NO₂ (0.02 – 0.04 ppm per year) and SO₂ (0.01 – 0.02 ppm), while the latter was NO₂ (0.04 – 0.07 ppm) and SO₂ (0.02 – 0.03 ppm). In the area more than 100 meters away, the levels decreased according to the distance from a highway to the value of the usual background. In other words, the average value of NO₂ in the area within 50 meters of a main highway was twice as much as that in the area over 100 meters away and the SO₂ was one and half times. Therefore, the investigated district except for the areas along main highways belongs to the least air-polluted districts, in the Tokyo Metropolis (7).

The families of school children were divided into three classes according to the conditions of smoking in the family. One was as family of non-
smokers, the second was a family with a light smoker who smoked within 20 cigarettes a day and the third was a family with a heavy smoker who smoked more than 21 cigarettes a day. When a family had more than two smokers, it was classified by the total number of cigarettes. Smokers of pipe tobacco and cigars were classified in terms of cigarettes.

Use of non-ventilated stoves employing petroleum, natural gas or propane gas and the structure of the house, classified as wooden or reinforced concrete, were included in the home environment questionnaire.

Supplementary investigation (11):

To assess the above mentioned study, a supplementary study by questionnaire was carried out on 1,928 school children (A and B schools in Suginami Ward) and 3,262 (C and D schools in Shimizu City, Shizuoka Prefecture) in May – July 1977.

The A and B schools were the same as those investigated in 1976. The C and D schools were selected because the annual atmospheric level of NO₂ and SO₂, and other demographic factors were very similar to those in the district of the A and B schools but the traffic volume of a highway passing through the area was about a half as that of one in the former district. Prevalence rates from the first screening of school children were compared between Suginami and Shimizu. The second screening was carried out in Suginami only.

RESULTS

The prevalence rates for symptoms in the first and second screenings were 10.8% and 7.7% respectively.

This report took up five elements for a specific small area or a subgroup within the investigated district as additional factors which raise the basic prevalence rate in school children locally which is reflected by the common combined factors of the investigated district. The factors were as follows: area according to distances from a main highway, smoking habits of the family, stove of the non-ventiled type, structure of the house and history of inhabitation. When a prevalence rate was examined for each factor, as shown in Table 1 and the Fig. 1, no significant differences were recognized for any of the factors. In other words, it was recognized that the nearer to a main highway an area is located, or the more heavy smokers in a family, the higher the prevalence rate tended to be but there were no significant differences.

When prevalence rates were analyzed by a combination of area according to distances from a main highway and smoking habits of the family as shown in Table 2 and the Fig. 2 the important points were as follows: The first problem was that all prevalence rates in school children in non-smoker families were almost the same over the whole district independent of the distance from a main highway; and in all families living more than 100 meters away, prevalence rates were almost the same independent of the family's smoking habits.

The second problem was that in the area within 100 meters of a main highway, the prevalence rate rose steeply as the distance decreased and showed a dose-response relationship in the sequence of non-smoker, light
smoker and heavy smoker families.

Relative risks in heavy smoker families by area with respect to the prevalence rate in non-smoker families were 4.0 in areas within 50 meters of a highway and about 3.0 in areas within 100 meters. The proportion of attributable risk to prevalence rate was 75 percent in heavy smoker families in areas within 50 meters of a highway.

From the above results, it is assumed that the basic atmospheric air pollution which is common over the whole investigated district can not raise the prevalence rate of symptoms by itself since it is at a level under the threshold value, even in areas along a main highway. Only when the effect of passive smoking is added, can the pollutitional level exceed the threshold value in areas within 100 meters of a highway.

Therefore the prevalence rate may change according to the distance from a highway and the degree of passive smoking. Thus, in an area more than 100 meters away from a highway, even if passive smoking is added, the prevalence rate still remains at a level under the threshold value because there is no effect of automobile exhaust. Of course, it is assumed that the pollution level in the investigated district is quite low for a huge city and the traffic volume of the main highway is from forty thousand to eighty thousand cars a day. If the annual average of background NO₂ is over 0.05 ppm and the traffic volume is over one hundred thousand cars, the prevalence rate will rise remarkably without the addition of passive smoking. No significant relationship with a combination of other factors could be found.

Table 1. Each Factor's Effect on the Prevalence Rate
(Suginami, 1976)

<table>
<thead>
<tr>
<th>Number of school children</th>
<th>Prevalence in the 1st screening</th>
<th>Prevalence in the 2nd screening</th>
</tr>
</thead>
<tbody>
<tr>
<td>Area according of distances from a main highway</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0~50 m.</td>
<td>111</td>
<td>19</td>
</tr>
<tr>
<td>0~100 m.</td>
<td>200</td>
<td>30</td>
</tr>
<tr>
<td>100 m.~</td>
<td>1696</td>
<td>174</td>
</tr>
<tr>
<td>Total</td>
<td>1896</td>
<td>204</td>
</tr>
<tr>
<td>Structure of house</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wooden</td>
<td>1347</td>
<td>146</td>
</tr>
<tr>
<td>Reinforced concrete</td>
<td>517</td>
<td>56</td>
</tr>
<tr>
<td>Total</td>
<td>1864</td>
<td>202</td>
</tr>
<tr>
<td>Omission</td>
<td>32</td>
<td>2</td>
</tr>
<tr>
<td>History of inhabitation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>0~three yrs</td>
<td>432</td>
<td>37</td>
</tr>
<tr>
<td>Three yrs~</td>
<td>1424</td>
<td>166</td>
</tr>
<tr>
<td>Total</td>
<td>1856</td>
<td>203</td>
</tr>
<tr>
<td>Omission</td>
<td>40</td>
<td>1</td>
</tr>
<tr>
<td>Stove of non-ventilated type</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Use</td>
<td>789</td>
<td>86</td>
</tr>
<tr>
<td>Non-use</td>
<td>1107</td>
<td>118</td>
</tr>
<tr>
<td>Total</td>
<td>1896</td>
<td>204</td>
</tr>
<tr>
<td>Smoking habits in the family</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-smokers</td>
<td>533</td>
<td>52</td>
</tr>
<tr>
<td>Light-smokers</td>
<td>1052</td>
<td>109</td>
</tr>
<tr>
<td>Heavy-smokers</td>
<td>311</td>
<td>43</td>
</tr>
<tr>
<td>Total</td>
<td>1896</td>
<td>204</td>
</tr>
</tbody>
</table>
Fig. 1  Effect of each factor on the prevalence rate.

- prevalence rate in the 1st screening
- prevalence rate in the 2nd screening

Distances from a highway.

Structures of house.

History of inhabitation.

Stove of non-ventilated type.

Smoking habits in the family.
### Table 2. Prevalence Rate according to Smoking Habits in the Family and Area. (Suginami, 1976)

<table>
<thead>
<tr>
<th></th>
<th>Non-smoker family</th>
<th>Light-smoker family</th>
<th>Heavy-smoker family</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>A</td>
</tr>
<tr>
<td>1.0~50m.</td>
<td></td>
<td></td>
<td></td>
<td>32</td>
</tr>
<tr>
<td>%</td>
<td></td>
<td></td>
<td></td>
<td>9.4</td>
</tr>
<tr>
<td>2.50m.~</td>
<td>501</td>
<td>49</td>
<td>37</td>
<td>989</td>
</tr>
<tr>
<td>%</td>
<td>9.8</td>
<td>7.4</td>
<td>10.0</td>
<td>7.0</td>
</tr>
<tr>
<td>3.0~100m</td>
<td>53</td>
<td>5</td>
<td>3</td>
<td>113</td>
</tr>
<tr>
<td>%</td>
<td>9.4</td>
<td>5.7</td>
<td>14.2</td>
<td>8.8</td>
</tr>
<tr>
<td>4.100m.~</td>
<td>480</td>
<td>47</td>
<td>36</td>
<td>939</td>
</tr>
<tr>
<td>%</td>
<td>9.8</td>
<td>7.5</td>
<td>9.9</td>
<td>6.8</td>
</tr>
<tr>
<td>Total</td>
<td>533</td>
<td>52</td>
<td>39</td>
<td>1053</td>
</tr>
<tr>
<td>%</td>
<td>9.8</td>
<td>7.3</td>
<td>10.4</td>
<td>7.0</td>
</tr>
</tbody>
</table>

A: Number of schoolchildren  
B: Prevalence in the 1st screening  
C: Prevalence in the 2nd screening

(1-3-4)  

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heavy-smokers</td>
<td>01≤p&lt;025</td>
<td>05≤p&lt;1</td>
</tr>
<tr>
<td>Light-smokers</td>
<td>1≤p&lt;25</td>
<td>5≤p</td>
</tr>
<tr>
<td>Non-smokers</td>
<td>n.s</td>
<td>n.s</td>
</tr>
</tbody>
</table>

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**Fig. 2** Prevalence rate according to smoking habits in the family and area (Suginami, 1976).  
Area according to distances from a main highway  
I - 50 m.  
II - 100 m.  
III 100 m.  
- prevalence rate in the 1st screening  
- prevalence rate in the 2nd screening
The supplementary investigation showed the following:

To simplify these data, the prevalence rate (in the first screening) in an area within 50 m from a highway was compared with one in an area over 100 m away, as shown in Fig. 4.

1. In the Suginami study (1976), the above epidemiological pattern was observed. A significant difference was found in the heavy-smoker families \((p < .025)\) and the same tendency was viewed but there was no significance in the light-smoker families.

2. In the Suginami study (1977), a significant difference was given \((p < .01)\) in the heavy-smoker families but the pattern disappeared in the light-smoker families.

3. In the Shimizu study (1977), it vanished completely, even in the heavy-smoker families.

### Table 3. Prevalence Rate according to Smoking Habits in the Family and Area (Suginami, 1977)

<table>
<thead>
<tr>
<th>Area</th>
<th>Non-smoker family</th>
<th>Light-smoker family</th>
<th>Heavy-smoker family</th>
<th>Total</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>%</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>0~50 m.</td>
<td>49</td>
<td>4</td>
<td>8.2</td>
<td>66</td>
<td>5</td>
</tr>
<tr>
<td>0~100 m.</td>
<td>83</td>
<td>7</td>
<td>8.4</td>
<td>118</td>
<td>8</td>
</tr>
<tr>
<td>100 m.~</td>
<td>484</td>
<td>43</td>
<td>8.9</td>
<td>680</td>
<td>62</td>
</tr>
<tr>
<td>Total</td>
<td>567</td>
<td>50</td>
<td>8.8</td>
<td>798</td>
<td>70</td>
</tr>
</tbody>
</table>

A: Number of school-children
B: Prevalence rate in the 1st screening.
H-S: \(p<.01\)
L-S: n.s.

**Fig. 3** Prevalence rate according to smoking habits in the family and area (Suginami, 1977). Area according to distances from a main highway:

- I: 0~50 m.
- II: 0~100 m.
- III: 100 m.~

- \(\square\) prevalence rate in the 1st screening
- \(\blacksquare\) prevalence rate in the 2nd screening
This disappearance of the pattern may be attributed to a decrease of NO₂ and other pollutants which mainly originated from the differences in traffic volume between the two districts and the chronological effects of strict regulations on automobile exhaust by the Environment Agency since 1973.

Therefore, this detectable change of a dependent variable may be considered to be an important epidemiological cutpoint which suggests intervention of some independent variables.

In conclusion, our study on the appearance or disappearance of this cutpoint will be useful in estimating the threshold value of NO₂.

Table 4. Prevalence Rate according to Smoking Habits in the Family and Area (Shimizu, 1977)

<table>
<thead>
<tr>
<th></th>
<th>Non-smoker family</th>
<th>Light-smoker family</th>
<th>Heavy-smoker family</th>
<th>Total</th>
<th>Unknown</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>A</td>
<td>B</td>
<td>%</td>
<td>A</td>
<td>B</td>
</tr>
<tr>
<td>O~50 m.</td>
<td>13</td>
<td>1</td>
<td>7.7</td>
<td>10</td>
<td>0</td>
</tr>
<tr>
<td>0~100 m</td>
<td>30</td>
<td>1</td>
<td>3.3</td>
<td>28</td>
<td>1</td>
</tr>
<tr>
<td>&gt;100 m</td>
<td>961</td>
<td>78</td>
<td>8.1</td>
<td>1467</td>
<td>104</td>
</tr>
<tr>
<td>Total</td>
<td>991</td>
<td>79</td>
<td>8.0</td>
<td>1495</td>
<td>105</td>
</tr>
</tbody>
</table>

H-S; L-S; n.s.

Fig. 4 Appearance and disappearance of the cutpoint.

- area within 50 m. of a highway
- area over 100 m. from a highway
DISCUSSION

To explain the reasons for the higher prevalence of coughing in a child whose parents are smokers, Colley (2) first proved that the prevalence of coughing in children whose parents have respiratory symptoms is high, and referring to the common knowledge that the prevalence of unspecific respiratory symptoms in smokers is high, concluded that the passive smoking effect on children and the sharing of genetic susceptibility to respiratory symptoms need not be taken into account, and airway cross-infections in a family are more causative.

Taylor and Dickinson (18) reported the relationship between the prevalence rate in children and their parents' smoking habits, but they did not mention the reason for it.

Cameron (1) and Harlap (4) admitted the relation between the two and considered passive smoking by parents as the reason.

On the other hand, Shy (20) denies the relation between them in his "Chattanooga Study" concerning the environmental criteria of NO₂. Thus, some scholars admit the relationship between the prevalence rate in children and their parents' smoking, while others deny it. In addition, some of scholars who admit the relation estimate the role of passive smoking very highly, while other scholars give little importance to it.

The results of this investigation show that in areas more than 100 meters away from a main highway, the prevalence rate in heavy smoker families is equal to that in non-smoker and light smoker families and the theory of Colley is clearly denied. If the prevalence rate is higher when children's parents are smokers and airway cross infections from parents to children raise the prevalence rate in children as Colley stated, the higher prevalence rate must occur in heavy smoker families independently of the distance from a main highway. Similarly, the sharing of genetic susceptibility to the respiratory symptoms will be denied, while the role of passive smoking is strongly supported.

Although Shy's report is contradictory to the report by Cameron, Harlap and Taylor with respect to the relationship between parental smoking and children's respiratory diseases, this is due to a difference between pollution levels in the environment of children during their investigation and such a contradiction is not unusual. Therefore if the basic pollution level of the environment in the district investigated by Shy is assumed to be comparatively low, the district which he investigated was Chattanooga where combined pollution by NO₂ and SO₂ does not exist but there is single pollution by NO₂. The prevalence rate will remain lower than the threshold value even with the addition of pollution by passive smoking, as well as in the area investigated more than 100 meters away from a highway. If environmental pollution in Cameron's district is close to or above the threshold value, as in our area within 50 meters of a highway, the prevalence rate should rise according to the dose-response of the added exposure to smoke from parents' smoking.

In conclusion, from the aforementioned results, the basic value of the prevalence rate among children of a certain district is assumed to be decided
by an allergic predisposition and a combination of host, environmental and agent factors including air pollution. Moreover, it became clear that the addition of NO\textsubscript{2} and suspended particulates caused locally by automobile exhaust and passive smoking raises the prevalence rate among children in the area along a main highway and in the heavy smoker families. We must leave this for future study because the effects of suspended particulates contained in smoke from smoking and sulfuric acid mist from automobile exhaust have not been explained epidemiologically.

(The summary of this study was reported by H. Kasuga, on the 8th International scientific meeting of the IEA, Puerto Rico, 1977.)

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REFERENCES

6) Ishizaki T, Kabe J, Nakagawa K; The effects of air pollution against asthma attack. J Jpn Society Air Poll, 7(1), 7-12, 1972.
13) Macriss RA, Elkins RH; Control of the level of NO\textsubscript{x} in the indoor environment. 4th International Clean Air Congress, Materials 4, 510-514, 1977.
17) Norman-Taylor W, Dickinson VA; Dangers for children in smoking families. Com-
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22) Task Group (Japan Environment Agency); Study on the effects of air pollution for human body (Observation of the level of NOx in the indoor environment). 1977, Jpn Pub Health Ass, Tokyo.
23) Task Group (Public Health Bureau, Tokyo Metropolis); The report on the prevalence of asthma in school children. 1975, Public Health Bureau, Tokyo Metropolis.
25) Tsubota N, Masuda H; Epidemiologic study on chronic respiratory symptoms in Okayama Prefecture. (Application of multiple regression analysis concerning air pollution and respiratory symptoms.) Record of lectures on the 18th Jpn Society of Air Poll. 247, 1977.
26) Tsunetoshi Y, Yamaguchi Y; Correlation of air pollution and chronic respiratory symptoms, JPH, 24(4), 293-300, 1977.