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Pulmonary Function Tests (PFTs) were performed twice (in 1996 and 1999) and analyzed in conjunction with air pollution estimates at the children's places of residence and several potential confounders - height, gender, parental education, passive smoking, housing density, length of residence in the study area and proximity to the main road.
Results: A significant negative association was found between changes in PFT results and individual exposure estimates to air pollution, controlled for socio-demographic characteristics of children and their living conditions.

Suggested Reviewers:

Opposed Reviewers:
Who is affected More by Air Pollution – Sick or Healthy? Some Evidence from a Health Survey of Schoolchildren Living in the Vicinity of a Coal-Fired Power Plant in Northern Israel

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Haifa -2009
Who is affected More by Air Pollution – Sick or Healthy? Some Evidence from a Health Survey of Schoolchildren Living in the Vicinity of a Coal-Fired Power Plant in Northern Israel

Abstract

Objective: To evaluate the effects of exposure to air pollution by NO$_x$ and SO$_2$ on the development of pulmonary function in children, characterized by different health status.

Methods: A cohort of 1181 schoolchildren from the 2$^{nd}$ - 5$^{th}$ grades, residing near a major coal-fired power plant in the Hadera district of Israel, was subdivided into three health status groups, according to the diagnosis given by a physician: a) healthy children; b) children experiencing chest symptoms, and c) children with asthma or spastic bronchitis. Pulmonary Function Tests (PFTs) were performed twice (in 1996 and 1999) and analyzed in conjunction with air pollution estimates at the children's places of residence and several potential confounders - height, gender, parental education, passive smoking, housing density, length of residence in the study area and proximity to the main road.

Results: A significant negative association was found between changes in PFT results and individual exposure estimates to air pollution, controlled for socio-demographic characteristics of children and their living conditions. A sensitivity test revealed a decrease in the Forced Expiratory Volume during the First Second (FEV$_1$) of about 21.5% for children with chest symptoms, 11.4% for healthy children, and approximately 8.7% for children diagnosed with asthma. Results of a sensitivity test for the Forced Vital Capacity (FVC) were found to be similar.

Conclusion: Exposure to air pollution appeared to have had the greatest effect on children with chest symptoms. This phenomenon may be explained by the fact that this untreated symptomatic group may experience the most severe insult on their respiratory system as a result of exposure to ambient air pollution, which is reflected by a considerable reduction in their FEV$_1$ and FVC. Since asthmatic children have lower baseline and slower growth rates, their PFT change may be affected less by exposure to air pollution, reflecting a well known relationship between pulmonary function change and height growth, according to which age
specific height is very similar for preadolescent children, but shifts upward with age during the growth spurt.

**Keywords:** children; air pollution; health status; pulmonary function test; individual exposure estimates.
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1. Introduction

According to several epidemiological studies, children are more susceptible to air pollution than adults due to increased respiratory rate and immature host defense mechanisms, which lead to the increased absorption of air pollutants (Bateson and Schwartz, 2008; Gauderman et al., 2000, 2002, 2004, 2007; Schwartz, 2004). Several studies indicated that gaseous pollutants and particulate matters tended to increase the risk of chronic chest symptoms and might have lifelong effects on children’s health (Bateson & Schwartz, 2008; Gauderman et al., 2004; Kajekar, 2007; Schwartz, 2004). Genetic factors, which regulate antioxidant defenses, may act together with environmental factors further elevating a potential risk of respiratory diseases in children (Gilliland et al., 1999, 2002; Kajekar, 2007; Peter and Felicity, 2008; Romieu et al., 2002; Schwartz, 2009).

The question whether sensitivity to air pollution, together with environmental factors and individual socio-demographic data, differs among children characterized by different health status (e.g., healthy children vs. asthmatic children) remains largely unanswered. Thus, for instance, Gauderman and colleagues (2004) did not find any significant effect of air pollution on asthmatic children, concluding that it may be influenced by a small sample size. Only a handful of studies looked into these differences, indicating that the long-term effects of air pollutants on pulmonary function (PF) growth may depend on children’s initial health status (see inter alia, Avol et al., 2001; Goren et al., 1997; Jedrychowski et al., 2002).

Several earlier studies demonstrated that the effects of air pollution on children’s PFTs differed between healthy children and asthmatics. In particular, such differences were detected for NO$_x$, SO$_2$, O$_3$ and PM exposures (see inter alia, Gauderman et al., 2004; Gilliland, 2009). Healthy preadolescents residing in neighborhoods with elevated levels of SO$_2$ and suspended particulate matters (SPM) were found to exhibit a delay in their pulmonary function growth (Jedrychowski et al., 2002), while pollutant initiated inflammation was found to worsen the pulmonary function (PF) performance among asthmatic children (Lee et al., 2002; Pikhart et al., 2000; Raizenne et al., 1996). Exposure to air pollution was also found to increase the number of asthmatic children residing in urban
areas (Delfino, 2002; Islam et al., 2007; Jerrett et al., 2008).

The greatest uncertainty of epidemiological studies is often associated with the estimation of exposure levels. A commonly used approach assumes that each person in a concerned region has the same exposure level, since the pollution often originates from the same pollution source (Elliott and Watenberg, 2000, 2004; Nuckols et al., 2004; Thurston et al., 2009). Exposure level assigned to each individual are often obtained from a few air quality monitors and reflect the mean concentrations in the entire urban area or community (Nuckols et al., 2004; Portnov et al., 2007). This approach may underestimate the individual exposure to air pollutants in the most polluted areas and overestimate it in the least polluted neighborhoods, thus leading to a measurement error known as exposure misclassification (Elliott and Savitz 2008; Nuckols et al., 2004; Portnov et al., 2007). Furthermore, the use of mean air pollution levels “smoothes” air pollution “spikes” (or so called “air pollution events”), and may thus result in unreliable estimates of health effects, which maybe especially harmful for children.

Several epidemiological surveys carried out in Israel compared the prevalence of respiratory symptoms, related to asthma and bronchitis, among subgroups of children residing in highly polluted areas with the prevalence of these symptoms among children residing in less polluted areas. Most of these studies detected the attenuation of PF development and an increase in the prevalence of respiratory morbidity in polluted areas (Goren et al., 1990, 1991, 1997; Peled et al., 2005). However, these studies were rather inconclusive, at least in part, in regard to the differential effect of air pollution on children, characterized by different health status.

Following our previous studies (Dubnov et al., 2007; Portnov et al., 2007), in the present analysis, we attempt to determine whether the previously detected effect of air pollution on children’s PF development vary across subgroups of children characterized by different health status, i.e., healthy children vs. asthmatics, etc.

The study is based on a cohort of 1181 schoolchildren residing in the Hadera district of Israel (Fig. 1), and subdivided into three health status groups - a) healthy children (children without known respiratory diseases and symptoms); b) children who experienced chest symptoms during one year preceding the pulmonary
function test, and c) children diagnosed by a physician as suffering from either asthma or spastic bronchitis.

2. Methods

2.1. Study area

The Hadera district of Israel, which forms our study area, is situated on the Mediterranean shore, some 50 km north of Tel Aviv. Its total population amounts to approx. 350,000 residents (ICBS, 2008). The study area hosts two townships (Hadera and Pardes-Hana-Karkur), two small suburbs (Beit Eliezer and Givat Olga), and a major coal-fired power plant with production capacity of about 2,590 MWatt (see Fig. 1). Sources of ambient air pollution in the area originate mainly from the power plant’s smokestacks, as well as from several small industrial plants scattered throughout the study area, and from motor vehicles.

2.2. Air pollution estimates

Air quality data for our study were obtained from the network of 12 air quality monitoring stations (AQMSs), maintained by the Hadera Association of Towns for the Environmental Protection (ATEP) and providing continuous measurements of air pollution by SO$_2$ and NO$_x$. In the present study, we used the "event" approach, used in several previous studies and found to be an effective and sensitive measure of local air pollution levels (Dubnov et al., 2007; Portnov et al., 2007). According to this approach, half-an-hour concentrations of NOx and SO$_2$ that simultaneously exceeded some predefined air pollution levels (i.e., 0.125 ppm for NOx and 0.070 ppm for SO$_2$) were multiplied by the average concentrations during the "air pollution events" and then summed up over the entire study period (for more detail on this approach, see Dubnov et al., 2007). These NOx*SO$_2$ "event" concentrations, observed at the location of AQMSs were then interpolated using the kriging interpolation method (Beers and Kleijnen, 2003; Dubnov et al., 2007; Sun-Young, et al., 2009), which enabled us to obtain a continuous surface of NOx*SO$_2$ air pollution estimates covering the entire study area (see Fig. 1).

To illustrate differences in the children’s PF performances across communities with distinctively different levels of air pollution, we also estimated
the average NOx*SO$_2$ "event" concentrations for 20 Small Census Areas (SCAs), into which the study area is divided, and then grouped these SCAs into three clusters, with distinctively different air pollution levels: low pollution (NOx*SO$_2$<312 ppm); medium pollution (312<NOx*SO$_2$<2640 ppm), and high pollution (NOx*SO$_2$>2640 ppm). The classification was performed using the Jenks’ “natural breaks” method in the ArcGIS9.x™ software. This method determines the best arrangement of values into classes by comparing the sum of squared differences of values from the means of their classes and thus identifies "natural break points" in the data distribution by picking the class breaks that best group similar values and maximize differences between classes (Minami & ESRI, 2000).

2.3. Study population

The health, demographic and pulmonary test data used in the present analysis are described in detail in Dubnov et al. (2007). In brief, the same cohort of 1181 schoolchildren performed pulmonary function tests (PFT) (FVC and FEV$_1$) in 1996 and, again, in 1999. The changes in pulmonary functions tests ($\Delta$PFTs) between the years were calculated as the percent difference between the observed and expected lung function volumes (Hankinson J. et al. 1999). For each child in the sample, health and socioeconomic data were obtained from individual questionnaires (Dubnov et al., 2007; Feris, 1978), filled out by the children's parents. The questionnaires included questions on housing density; exposure to passive/active tobacco smoking; parental education and duration of living in the study area.

2.4. Health status classification

The classification of the children into health status subgroups was based on the health questionnaire described in Dubnov et al. (2007) and being similar to those used elsewhere (Leonardi et al., 2002). In particular, the following three "health status" categories (subgroups) were distinguished:

1. Healthy subgroup: The child has been never diagnosed by a physician with either spastic bronchitis or asthma and have not had any chest symptoms
during 12 months prior to PFT.

2. **Chest symptoms subgroup**: The child has had at least one of the following symptoms during 12 months preceding PFT: wheezing with or without upper respiratory tract infection (URTI); coughing with or without URTI; coughing with phlegm or wheezing with dyspnea.

3. **Asthma/spastic bronchitis subgroup**: The child was diagnosed by a physician with either asthma or spastic bronchitis and exhibited at least one of the above respiratory symptoms, as *idem* in (2).

2.5. Individual exposure estimates

In the present study, we used an indirect approach to estimating individual air pollution exposure (Dubnov et al., 2007; Jerrett et al., 2008; Sun-Young, et al., 2009). According to this approach, the homes of the children participating in the survey were positioned on a map and then superimposed with the levels of NOx*SO$_2$ air pollution at the corresponding locations (see Section 2.2 and Fig. 1). Next, the levels of NOx and SO$_2$ air pollution to which each child was exposed were calculated. The task was performed in the ArcGIS 9$^\text{TM}$ software, using its "spatial join" tool, which makes it possible to match between different geographic layers (maps) based the spatial relationship between them (Minami & ESRI, 2000).

2.6. Statistical analysis

The analysis was carried out in four phases. During the *first phase*, changes in the proportional shares of different health status groups in the total population between 1996 and 1999 was examined separately for the sub-regions with low, medium and high air pollution (see Section 3.1). During the *second phase* of the analysis, interaction between $\Delta$PFT observed across different "health status" groups of children (see Section 3.4) and air pollution levels (Section 3.2) was investigated using the General Liner Model (GLM) technique, a statistical tool which helps to reveal intra-group interactions (McCullough & Nelder, 1989). During the *third phase* of the analysis, the multiple regression analysis was run to indentify and measure the effect of air pollution on 1996-99 changes in the children's PFT (both $\Delta$FEV$_1$ and $\Delta$FVC), adjusted for several potential confounders,
such as the duration of residence in the study area, parental smoking, housing density, father’s education, and proximity to the main road. The analysis was performed separately for each health status subgroup under study (see Section 3.3). During the analysis, the normality and the heterogeneity of variance assumptions were verified and the results were found to be satisfactory. Lastly, during the fourth phase of the analysis, a sensitivity test was run to determine the magnitude of changes in the children's $\Delta$FEV$_1$ and $\Delta$FVC, attributed to plausible changes in ambient air pollution levels.

3. Results

3.1. Cross-group comparison

Table 1 illustrates changes in the numbers of children in each health status group in different air pollution regions, which occurred between 1996 and 1999. As the table shows, the percent of children, diagnosed as healthy, decreased between 1996 and 1999 in all air pollution regions, but most substantially in the “high air pollution” zone. For example, the number of healthy children in the least polluted area (“low air pollution” region) dropped during the study period by some 13.4%, from 268 children diagnosed as healthy in 1996 to 232 such children in 1999. Concurrently, the corresponding decrease in the number of healthy children in the “high air pollution” zone reached 25%, from 40 children diagnosed as healthy in 1996 to 30 such children in 1999.

The opposite trend is, however, found in the “chest symptoms” group. The number of children in this group increased in all air pollution zones, but, again, most substantially, in the “high air pollution” region. In particular, the number of children exhibiting chest symptoms in this region increased between 1996 and 1999 by nearly twofold, from 20 children diagnosed with chest symptoms in 1996 to 37 such children in 1999, or by 85%.

Characteristically, the direction of changes appear to be less consistent in the “pulmonary diseases” subgroup, which may be explained by a relatively small numbers of children in this group overall, which may not be sufficient for generalization.

<<< Table 1 about here >>>
3.2. Inter-group interaction

Table 2 shows the results of GLM analysis, which illustrates the strength of interaction between air pollution levels and observed $\Delta FEV_1$ across different health status groups.

The interaction between air pollution levels and the children's PFT change appears to be statistically significant (Roy's=2.37, $P<0.05$), indicating that the effect of air pollution does appear to depend on a child's health status at the beginning of the study period.

The analysis of the results of Scheffe's post-hoc test further indicates that $\Delta FEV_1$ of healthy children and that of children, suffering from chest symptoms, differs significantly across air pollution zones (Healthy: $F=10.16; P<0.01$; Chest symptoms: $F=11.33; P<0.01$), while no such an effect is found for children with asthma and spastic bronchitis ($F=1.48; P>0.1$)

3.3. Multivariate regression analysis

Table 3 shows the results of the multivariate regression analysis of the factors affecting the FEV$_1$ change across different health status groups of children between 1996 and 1999 (The results for the FVC change were found to be similar and are not reported here for brevity's sake). In the analysis, individual air pollution estimates (see Subsection 2.5) were controlled for several potential confounders: height, gender, duration of residence in the study area, parental smoking, housing density, parent’s education, and proximity to the closest main road (see Appendixes 1-2 ). The models appear to provide a reasonably good fit ($R^2=0.375-0.427$), and a high degree of generality ($F=22.244-37.092; P<0.001$).

As Table 3 shows, $\Delta FEV_1$ is significantly and positively related to the height of a child ($t=11.0-21.0; P<0.01$) and, in most models, with the duration of living in the study area ($t>2.0; P<0.05$). The effect of the NO$_x$*SO$_2$ interaction term on the children’s PFT performance appears to be negative and highly significant in most
models (P<0.01), implying that increasing air pollution levels do appear to have, *ceteris paribus*, a significant and negative effect on the children’s PF growth.

Characteristically, the strongest “NO\textsubscript{X}*SO\textsubscript{2} - \Delta FEV\textsubscript{1}” association is detected in the “chest symptoms” subgroup (t=-6.454; P<0.001) and among “healthy children” (t=-3.534; P<0.01). However, this association is much weaker for the “PF diseases” subgroup (t=-1.817; P>0.05), implying that PF growth in children with chronic pulmonary diseases appears to be least affected by air pollution, compared to other groups under study. A possible explanation for this phenomenon will be suggested in the following section.

4. Discussion

Since individuals differ in their metabolic rate and immune system functioning, some are likely to be more susceptible to ambient air pollution than others (Arshad et al., 2005; D’Amato et al., 2005; Kim, 2004; Paden, 2005). In the present study, a cohort of 1181 schoolchildren divided into three subgroups based on their health status was tested twice for their PFT performance in 1996 and again in 1999.

The results of the analysis indicate that the numbers of healthy children in the study cohort decreased over the study period overall, but most substantially in the most air polluted areas. In particular, the number of healthy children in the “low pollution” zone dropped during the study period by some 13.4%, from 268 children diagnosed as healthy in 1996 to 232 such children in 1999. Concurrently, the corresponding decrease in the number of healthy children in the “high air pollution” zone reached 25%. GLM analysis also indicated that the interaction between air pollution levels and the children's health status appeared to be highly significant (P>0.001), implying that the PF development of children in different health status groups appeared to respond differently to air pollution estimated for the places of the children’s residence.

Regression models, adjusted for the children's health and socio-demographic characteristics, also indicated that air pollution estimates (measured in this study by the NO\textsubscript{X}*SO\textsubscript{2} interaction term) appeared to have a stronger negative effect on the PFT change in healthy children and children with chest symptoms, than in children with already diagnosed chronic respiratory diseases (i.e., spastic bronchitis and asthma).
A sensitivity test of \( \Delta \text{FEV}_1 \) to plausible changes in NOx*SO\(_2 \) air-pollution levels is reported in Table 4. The test is based on the regression models reported in Table 3.

As Table 4 shows, the largest \( \Delta \text{FEV}_1 \) deficit (-21.6%) is observed in the “chest symptoms” subgroup, followed by healthy children (-11.5%) and children with chronic respiratory diseases (-8.7%), thus suggesting that healthy children and children with chest symptoms appear to be most susceptible to chronic respiratory effects associated with their exposure to air pollution.

A possible explanation of this trend may be attributed to differences in the PFT growth “trajectories” among different health status groups. The relation between pulmonary function and height is well known: The age-specific height is very similar for the preadolescent children, but shifts upward with age during the growth spurt (Wang et al., 1993). Some studies have shown that children with asthma exhibited impaired growth (Balfour-Lynn, 1986; Littlewood et al., 1988; Murray et al., 1976; Wales et al., 1991; Wolthers and Pedersen, 1991). This slow growth may lessen the effect of air pollution on PF changes and differences in measurements between “initial performance values” (FEV\(_1 \_1996 \)) and “final performance values” (FEV\(_1 \_1999 \)). As Wang and colleagues (1993) reported, FEV\(_1 \) of asthmatic children was, on the average, 1.5-3% lower than that of children who had no asthma or wheezing. In particular, asthmatic children tend to exhibit airway hyper-responsiveness and obstruction, which is reflected by low values of polluted air they inhale (GINA, 2008; Sears et al., 2003).

In our study the most affected population were the children not diagnosed as asthmatics but tended to experience chest symptoms. In fact, those children may belong to an “undiagnosed” and, therefore, “untreated-for-asthma” group. This group of children may experience the most severe insult on their respiratory system as reflected by a greater reduction of their PFT from the baseline.\(^1\)

Our results are generally consistent with the results of previous studies

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\(^1\) Medical surveillance and medicine intake were analyzed in our study as potential confounders, but were not found significant. These results are not reported here for brevity’s sake and can be obtained from the authors upon request.
carried out in the same region (Dubnov et al., 2007; Portnov et al., 2007), which reported a decline of children’s PFT performance in line with increasing air pollution levels. As in these previous studies, we used the individual exposure estimate approach, based on geographic information systems (GIS) technologies, which are more sensitive than zonal approaches, traditionally used in the epidemiological research (Jerrett et al., 2008; Sahsuvaroglu et al., 2009; Sun-Young et al., 2009).

Earlier evidence, accumulated during the 1990’s, also indicated a rise in the prevalence of acute pulmonary symptoms (upper respiratory tract infection (URTI), and lower respiratory tract infection (LRTI) among asthmatics and non-asthmatics children (Brauer et al., 2002; Goren et al., 1997). According to these studies, although preadolescent (6-10 year old) children exhibited a more acute prevalence of these symptoms, no parallel decline in their PFTs was reported. These adverse effects air pollution on lung development responses were also detected by studies carried out elsewhere (Gauderman et al., 2002, 2004; Horak et al., 2002).

Albeit there is no shortage of studies investigating the association between air pollution and children pulmonary function performance (Gauderman, et al., 2000, 2002, 2004, 2007; Horak et al., 2002; Jedrychowski et al., 1999, 2002; Lee et al., 2002; Peters et al., 1999a, b), we are unaware of other studies that evaluated the effect of air pollution on children subgroups characterized by different initial health status and used individual exposure estimate approach.

5. Study limitations

Population based epidemiologic studies are often susceptible to a selection bias arising from information loss and misclassification of exposure (Elliott and Watenberg, 2000; Greenland and Morgenstern, 1989). Differences in personal characteristics (physiological, genetic, diseases, lifestyle, smoking, alcohol, drugs, sexual orientation), and differences in pollution sources (industries and power plants, motor vehicles, naturally occurring dust storms), as well as differences in topography, meteorology, etc. could lead to these biases (Elliot et al., 1992; Morgenstern and Thomas, 1993; Lash et al., 2009). Since the same cohort of children was tested twice (in 1996 and again in 1999), and their health status and
residential conditions were estimated by individual questionnaires, these sources of bias are less likely to occur in the present study.

An additional sort of bias, attributed to exposure misclassification, can also arise from an assumption that average exposure levels estimated for townships and SCAs are used to approximate the air pollution exposure of individuals (Jerrett et al., 2008; Portnov et al., 2007; Sun-Young et al., 2009). Since, in the present study, we used individual exposure estimates, this source of bias was also minimized.

Lastly, the effect of air pollution on asthmatic children, who receive medical treatment, and the effect of air pollution on children suffering from chest symptoms, but not taking medications, may be different (Delfino et al., 2008; Liu et al., 2009). However, we had no robust information on such “undiagnosed” asthmatic children and had to rely on information obtained from questionnaires filled out by the children’s parents.

6. Conclusion

As the present study indicates, the long-term exposure to ambient air pollution appears to have more detrimental effects on pulmonary function growth among children with chest symptoms and healthy than among children diagnosed with asthma and spastic bronchitis, with the estimated PFT deficit ranging from -8.7% for asthmatics to -21.6% for children exhibiting chest symptoms. A likely explanation of this trend is that different effects of exposure to ambient air pollution on the airways in these groups when lungs rapidly develop. In asthmatic children, the height growth is often stunned. As a result, the prediction pulmonary function values can be less accurate than for children whose growth track is normal. Since asthmatic children had lower baseline of PF and lower growth rates, the reduction of the PFT values may be less affected. Therefore, the effect of air pollution on this children subgroup may be less pronounced. However, further studies are needed to understand better this phenomenon. Nevertheless, the data already available provide evidence that children, particularly those with increased susceptibility, such as untreated children with chest symptoms, may benefit substantially from a reduction in the current levels of air pollution, especially in densely populated urban areas.
7. References


Figure 1: Map of the study area
Table 1: Mobility of children across health status groups in different air pollution regions in 1996-1999*

<table>
<thead>
<tr>
<th>Air pollution region</th>
<th>Health group</th>
<th>Number of children in 1996</th>
<th>Number of children in 1999</th>
<th>% Change</th>
<th>Prob.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>Healthy</td>
<td>268</td>
<td>232</td>
<td>-13.4</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Chest symptoms</td>
<td>170</td>
<td>208</td>
<td>+22.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulmonary diseases</td>
<td>136</td>
<td>134</td>
<td>-1.5</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total:</td>
<td>574</td>
<td>574</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Medium</td>
<td>Healthy</td>
<td>97</td>
<td>74</td>
<td>-23.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>Chest symptoms</td>
<td>77</td>
<td>84</td>
<td>+9.1</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulmonary diseases</td>
<td>40</td>
<td>56</td>
<td>+40.0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total:</td>
<td>214</td>
<td>214</td>
<td></td>
<td></td>
</tr>
<tr>
<td>High</td>
<td>Healthy</td>
<td>40</td>
<td>30</td>
<td>-25.0</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td></td>
<td>Chest symptoms</td>
<td>20</td>
<td>37</td>
<td>+85.0</td>
<td></td>
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<tr>
<td></td>
<td>Pulmonary diseases</td>
<td>27</td>
<td>20</td>
<td>-25.9</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total:</td>
<td>87</td>
<td>87</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Includes only children with reported health status in both 1996 and 1999 (875 cases out of 1181 children in the sample).
Table 2: Interaction of health status with air pollution levels (Method: GLM-univariate; dependent variables - $\Delta FEV_1$)\(^1\)

<table>
<thead>
<tr>
<th>Health status group</th>
<th>Air pollution zone</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Low</td>
<td>Mid</td>
<td>High</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mean (S.D.)</td>
<td>N</td>
<td>Mean (S.D.)</td>
<td>N</td>
</tr>
<tr>
<td>Healthy</td>
<td>-2.12 (12.77)</td>
<td>360</td>
<td>-4.44 (13.22)</td>
<td>136</td>
</tr>
<tr>
<td></td>
<td>$P&lt;0.01$</td>
<td></td>
<td>$P&lt;0.01$</td>
<td></td>
</tr>
<tr>
<td>Chest symptoms</td>
<td>-2.75 (12.95)</td>
<td>230</td>
<td>-1.40 (11.18)</td>
<td>104</td>
</tr>
<tr>
<td></td>
<td>$11.33$</td>
<td></td>
<td>$P&lt;0.01$</td>
<td></td>
</tr>
<tr>
<td>Pulmonary diseases</td>
<td>-1.82 (11.96)</td>
<td>194</td>
<td>-3.23 (12.87)</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td>$1.48$</td>
<td></td>
<td>$P&gt;0.05$</td>
<td></td>
</tr>
</tbody>
</table>

Note: Total number of cases – 1181; number of valid cases list-wise - 1177 (excluding four cases with missing values); \(^a\) F-statistic and its degree of freedom (df), post-hoc differences.
### Table 3. Factors affecting the pulmonary function change by children subgroups

(Independent variable - $\Delta$FEV₁; Method – Multiple regression analysis)

<table>
<thead>
<tr>
<th>Variable</th>
<th>All population</th>
<th>Healthy</th>
<th>Chest symptoms</th>
<th>Pulmonary diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$B^a$</td>
<td>$T^{b,c}$</td>
<td>$B^a$</td>
<td>$T^{b,c}$</td>
</tr>
<tr>
<td>Father’s education</td>
<td>0.160</td>
<td>1.378</td>
<td>0.413</td>
<td>2.231*</td>
</tr>
<tr>
<td>Gender</td>
<td>-0.208</td>
<td>-0.338</td>
<td>0.032</td>
<td>0.034</td>
</tr>
<tr>
<td>Height</td>
<td>0.669</td>
<td>21.110**</td>
<td>0.632</td>
<td>12.605**</td>
</tr>
<tr>
<td>Housing density</td>
<td>0.498</td>
<td>0.987</td>
<td>0.438</td>
<td>0.547</td>
</tr>
<tr>
<td>NO₅*SO₂</td>
<td>-0.004</td>
<td>-6.558**</td>
<td>-0.003</td>
<td>-3.534**</td>
</tr>
<tr>
<td>Passive smoking</td>
<td>0.945</td>
<td>1.515</td>
<td>1.591</td>
<td>1.690</td>
</tr>
<tr>
<td>Proximity to the main road</td>
<td>-1.166</td>
<td>-1.807</td>
<td>0.047</td>
<td>0.048</td>
</tr>
<tr>
<td>Year in study area</td>
<td>0.409</td>
<td>3.158*</td>
<td>0.671</td>
<td>3.011*</td>
</tr>
<tr>
<td>(Constant)</td>
<td>-98.645</td>
<td>-21.822**</td>
<td>-100.918</td>
<td>-14.439**</td>
</tr>
<tr>
<td>$R^2$</td>
<td>0.378</td>
<td>0.375</td>
<td>0.427</td>
<td>0.398</td>
</tr>
<tr>
<td>$R^2$– adjusted</td>
<td>0.373</td>
<td>0.365</td>
<td>0.412</td>
<td>0.380</td>
</tr>
<tr>
<td>$F$</td>
<td>83.611**</td>
<td>37.092**</td>
<td>29.309**</td>
<td>22.244**</td>
</tr>
<tr>
<td>No of cases (total)</td>
<td>1181</td>
<td>537</td>
<td>357</td>
<td>287</td>
</tr>
<tr>
<td>No of case (valid list wise)</td>
<td>1110</td>
<td>504</td>
<td>324</td>
<td>282</td>
</tr>
</tbody>
</table>

Note: Variables are reported in alphabetical order; $^a$ unstandardized regression coefficient; $^b$ t-statistic; $^c$ significance of $t$-statistic: * $p<0.05$; ** $p<0.001$
Table 4. Sensitivity test of ΔFEV<sub>1</sub> to plausible changes in NOx*SO<sub>2</sub> air pollution levels (\%)

<table>
<thead>
<tr>
<th>Air pollution levels (ppm)</th>
<th>All population</th>
<th>Healthy symptoms</th>
<th>Chest symptoms</th>
<th>Pulmonary diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td>No pollution (NOx*SO&lt;sub&gt;2&lt;/sub&gt; = 0)</td>
<td>-2.395</td>
<td>-3.239</td>
<td>-0.399</td>
<td>-3.191</td>
</tr>
<tr>
<td>Medium pollution (NOx*SO&lt;sub&gt;2&lt;/sub&gt; = 312)</td>
<td>-3.630</td>
<td>-4.210</td>
<td>-2.897</td>
<td>-3.847</td>
</tr>
<tr>
<td>High pollution (NOx*SO&lt;sub&gt;2&lt;/sub&gt; = 2640)</td>
<td>-12.834</td>
<td>-11.445</td>
<td>-21.518</td>
<td>-8.742</td>
</tr>
</tbody>
</table>

Note: Based on models in Table 3; input constants: Proximity to the main road =0 (> 50 m); passive smoke = 0 (no); Gender = male; Duration of living in the study area = 8 years (average for the sample); Housing density=1.3 persons per room (ibid.); Father's education = 12.71 years (ibid.); Height = 136.28 cm (ibid.).
APPENDIX 1: General characteristics of the schoolchildren surveyed in 1996 and 1999

<table>
<thead>
<tr>
<th>Variable</th>
<th>1996</th>
<th>1999</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>Observed FVC (liters)</td>
<td>2.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Predicted FVC (%)</td>
<td>94.3</td>
<td>12.4</td>
</tr>
<tr>
<td>Observed FEV₁ (liters)</td>
<td>1.8</td>
<td>0.4</td>
</tr>
<tr>
<td>Predicted FEV₁ (%)</td>
<td>100.7</td>
<td>13.7</td>
</tr>
<tr>
<td>Female gender (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>54</td>
<td></td>
</tr>
<tr>
<td>Age (yr)</td>
<td>9.2</td>
<td>1.5</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>136.3</td>
<td>10.6</td>
</tr>
<tr>
<td>Father's education (years of schooling)</td>
<td>12.7</td>
<td>2.7</td>
</tr>
<tr>
<td>Passive smoking in the family % a</td>
<td>53</td>
<td></td>
</tr>
<tr>
<td>Length of residence in the study area (yr)</td>
<td>8.0</td>
<td>2.7</td>
</tr>
<tr>
<td>Proximity to the main road (%) b</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Housing density (person/room)</td>
<td>1.3</td>
<td>0.7</td>
</tr>
</tbody>
</table>

Note: a At least one person smokes in the child’s home. b Living within 50-m from a main road (first raw of buildings).
### APPENDIX 2: Characteristics of "health status" groups (according to the 1996 survey)

<table>
<thead>
<tr>
<th>Variable</th>
<th>Healthy (N=537)</th>
<th>Chest symptoms (N=357)</th>
<th>Pulmonary diseases (N=287)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>S.D.</td>
<td>Mean</td>
</tr>
<tr>
<td>Age (years)</td>
<td>9.3</td>
<td>1.6</td>
<td>9.1</td>
</tr>
<tr>
<td>Father's education (years of schooling)</td>
<td>12.8</td>
<td>2.6</td>
<td>12.5</td>
</tr>
<tr>
<td>Female gender (%)</td>
<td>56</td>
<td></td>
<td>53</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>136.6</td>
<td>10.7</td>
<td>135.9</td>
</tr>
<tr>
<td>Housing density (person/room)</td>
<td>1.3</td>
<td>0.7</td>
<td>1.4</td>
</tr>
<tr>
<td>Length of residence in the study area (yr)</td>
<td>8.4</td>
<td>2.5</td>
<td>7.7</td>
</tr>
<tr>
<td>Observed FEV₁ (liters)</td>
<td>1.9</td>
<td>0.4</td>
<td>1.8</td>
</tr>
<tr>
<td>Observed FVC (liters)</td>
<td>2.0</td>
<td>0.5</td>
<td>2.0</td>
</tr>
<tr>
<td>Passive smoking in the family (%)</td>
<td>46.0</td>
<td></td>
<td>55.5</td>
</tr>
<tr>
<td>Predicted FEV₁ (%)</td>
<td>101.6</td>
<td>13.8</td>
<td>100.1</td>
</tr>
<tr>
<td>Predicted FVC (%)</td>
<td>94.9</td>
<td>12.0</td>
<td>93.5</td>
</tr>
<tr>
<td>Proximity to the main road (%)</td>
<td>39.0</td>
<td></td>
<td>32.5</td>
</tr>
</tbody>
</table>

Note:  
- a At least one person smokes in the child’s home;  
- b percent of children living within 50-m from a main road