Improvements in PM$_{10}$ Exposure and Reduced Rates of Respiratory Symptoms in a Cohort of Swiss Adults (SAPALDIA)

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**Rationale:** Reductions in mortality following improvements in air quality were documented by several studies, and our group found, in an earlier analysis, that decreasing particulate levels attenuate lung function decline in adults.

**Objectives:** We investigated whether decreases in particulates with an aerodynamic diameter of less than 10 $\mu m$ (PM$_{10}$) were associated with lower rates of reporting respiratory symptoms (i.e., decreased morbidity) on follow-up.

**Methods:** The present analysis includes 7,019 subjects who underwent detailed baseline examinations in 1991 and a follow-up interview in 2002. Each subject was assigned model-based estimates of average PM$_{10}$ during the 12 months preceding each health assessment and the difference was used as the exposure variable of interest ($\Delta$PM$_{10}$). Analyses were stratified by symptom status at baseline and associations between $\Delta$PM$_{10}$ and change in symptom status during follow-up were adjusted for important baseline characteristics, smoking status at follow-up, and season. We then estimated adjusted odds ratios for symptoms at follow-up and numbers of symptomatic cases prevented due to the observed reductions in PM$_{10}$.

**Measurements and Main Results:** Residual exposure to PM$_{10}$ was lower in 2002 than in 1991 (mean decline 6.2 $\mu g/m^3$/SD = 3.9 $\mu g/m^3$). Estimated benefits (per 10,000 persons) attributable to the observed changes in PM$_{10}$ levels were: 259 (95% confidence interval [CI]: 102–416) fewer subjects with regular cough, 179 (95% CI, 30–328) fewer subjects with chronic cough or phlegm and 137 (95% CI, 9–266) fewer subjects with wheezing and breathlessness.

**Conclusions:** Reductions in particle levels in Switzerland over the 11-year follow-up period had a beneficial effect on respiratory symptoms among adults.

Several studies have reported cross-sectional associations between long-term exposure to air pollution and respiratory disease in adults (1–7). Recently three studies have observed substantial decreases in adult mortality after reductions in air pollution (8–10). However, with two exceptions (11, 12), the only studies to address whether gradual improvements in air quality can lead to improvements in respiratory symptoms or lung function have been in children. A number of cross-sectional studies of successive age cohorts of children have documented reductions in respiratory symptoms following decreases in air pollution levels (13–16). But because there was no follow-up of subjects, these results did not imply reversibility of symptoms. The gap was closed by a prospective cohort study in Californian children, which showed that moving to a less polluted area resulted in accelerated lung-function growth in children and suggested that previously documented impairments could be reversed (17). Similarly, we found attenuations in age-related lung-function decline among persons who had experienced decreases in ambient residential PM$_{10}$ levels during the 11 years of follow-up (12). These findings suggested that gradual decreases in air pollution levels might also lead to a concurrent reduction in respiratory morbidity among adults. This hypothesis gave rise to the present analysis. Its confirmation is relevant for public health because the significance of a decrease in air pollution exposure depends on the...
amount of time before health effects can be observed. If, contrary to our hypothesis, most of the damage caused by past exposure were irreversible, then beneficial effects of improvements in air quality would only manifest themselves gradually and predominantly in the younger cohorts.

The Swiss study on air pollution and lung disease in adults (SAPALDIA) cohort study was originally designed to assess respiratory health in the adult population of Switzerland and to study potential associations between long-term exposure to air pollution and various respiratory health parameters. In the original study sample of 9,651 subjects from eight areas, we found the average levels of forced vital capacity to be negatively associated with ambient levels of particulates with an aerodynamic diameter of less than 10 μm (PM₁₀) across areas (18) and with estimated home outdoor and personal exposure to nitrogen dioxide (NO₂) within areas (19). Moreover, ambient levels of PM₁₀ were positively associated with the prevalence of breathlessness at rest and of chronic bronchitis symptoms (3).

In 2002, 8,047 subjects were reassessed following the same study protocol as in 1991. One of the primary objectives of the second survey was to study longitudinal changes in respiratory symptoms and in lung function and their relation to changes in air pollutant levels. For this purpose, we estimated individual PM₁₀-exposure trajectories for all subjects based on their residential histories before the first visit. This variable is referred to as estimated change in PM₁₀ exposure before the first visit. The marker of ambient air pollution considered was PM₁₀ (i.e., particulates with an aerodynamic diameter of less than 10 μm (PM₁₀) across areas). The shape of associations between PM₁₀ and new or persistent symptom reports was explored using natural splines (24). We also estimated the percentage of reduction in the number of symptom cases attributable to the observed decreases in PM₁₀ levels during follow-up. Sensitivity analyses were performed in different subsamples using interaction terms between specific individual characteristics and ΔPM₁₀.

### METHODS

#### Study Population and Health Examinations

At baseline in 1991 (SAPALDIA1), the cohort consisted of 9,651 randomly selected adults, 18 to 60 years of age, from eight diverse areas of Switzerland. Subjects underwent a detailed health examination consisting of an extensive computer-assisted interview on respiratory and allergic diseases and personal and environmental risk factors, measurements of pulmonary function, and assessments of atopic sensitizations (21). In 2002 (SAPALDIA2), most assessments were repeated and some were expanded (22). To be included in the present analysis, study subjects had to have lived at the same address for at least 1 year before the baseline assessment and had to provide information on respiratory symptoms and smoking status in the second assessment. All SAPALDIA study protocols were approved by the Ethics Committee of the Swiss Academy of Medical Sciences and by the local Ethics Committees.

#### Assignment of Individual Exposure Estimates

The marker of ambient air pollution considered was PM₁₀ (i.e., particulates with an aerodynamic diameter of less than 10 μm). PM₁₀ concentrations outside of each subject’s residence were estimated for the years 1990 and 2000 using a validated dispersion model (with different emissions outside of each subject’s residence were estimated for the years subjects’ residential histories. Details of these methods and the model evaluation were given in (23). The exposure variable used in the present analysis was the difference between the estimated average PM₁₀ level outside a subject’s home(s) in the 12 months before the second assessment in 2002 and the corresponding mean level in the 12 months before the first visit. This variable is referred to as estimated change in home outdoor PM₁₀ and will be abbreviated ΔPM₁₀.

#### Definition of Symptom Categories Considered

**Regular cough (regular phlegm)** was defined by an affirmative answer to at least one of the following questions “Do you usually cough (bring up phlegm from your chest) first thing in the morning?” and/or “Do you usually cough (bring up phlegm from your chest) during the day or at night?”

**Chronic cough or phlegm** was defined as chronic cough and/or chronic phlegm, with “chronic” being defined by the presence of the respective symptoms during at least 3 months per year for at least two years. **Wheezing** was defined by an affirmative answer to the question “Have you had wheezing or whistling in the chest at any time in the last 12 months?” **Wheezing with dyspnea** was defined according to the question “Have you had trouble breathing when you had this wheezing or whistling in your chest?” And **wheezing without a cold** was defined according to the question “Have you had this wheezing or whistling in your chest when you did not have a cold?”

### Statistical Analysis

Logistic regression models were used to describe the odds of reporting a given respiratory symptom in SAPALDIA2 as a function of ΔPM₁₀. Because symptom status at baseline is the strongest predictor of symptoms at follow-up and modifies the role of other predictor variables, analyses were stratified by symptom status at baseline. We also controlled for sex, age, smoking status at SAPALDIA1 (smoker, ex-smoker, never-smoker), smoking status at SAPALDIA2 (smoker, nonsmoker), the seasons of the two interviews, and a large number of baseline exposure variables and risk factors (see footnote of Table 3 and online supplement). The function GLLAMM from STATA (Stata, release 9; StataCorp LP, College Station, TX) was used to incorporate random area effects accounting for uncontrolled differences between the eight original study samples. For each outcome, the two associations with ΔPM₁₀ (for incidence and persistence) were subject to a Chi² test with two degrees of freedom to assess the global null hypothesis of absence of any association. The shape of associations between ΔPM₁₀ and new or persistent symptom reports was explored using natural splines (24). We also estimated the percentage of reduction in the number of symptom cases attributable to the observed decreases in PM₁₀ levels during follow-up. Sensitivity analyses were performed in different subsamples using interaction terms between specific individual characteristics and ΔPM₁₀.

### RESULTS

We could include up to 7,019 of the 8,047 participants of SAPALDIA2 in the present analysis. A total of 905 subjects did not provide complete information on symptoms and covariates. Geocoding of addresses failed in 57 subjects and 66 subjects were excluded because they had relocated less than a year before the first health assessment (Figure 1). For the majority of subjects in our sample, residential addresses at the time of the two surveys could be geocoded exactly (n = 5,772, 82.2%) using a nearest-neighbor address (n = 755, 10.8%) or the median house number of the respective street (n = 492, 7.0%).

Differences between the sample of the present analysis and subjects who only participated in SAPALDIA1 or provided incomplete data are shown in Table 1. Compared with the nonparticipants, the study sample had a higher mean age (by 1.2 yr) and a lower mean body mass index (by 0.5 kg/m²). Moreover, it included higher proportions of women, of non-smokers at SAPALDIA1, and of Swiss nationals. However, there was no statistically significant difference between the two samples with respect to the average change in home outdoor PM₁₀ between surveys. Differences between SAPALDIA2 participants and subjects having only participated in the baseline survey were previously described in detail (22).

Table 2 shows the respective fractions of subjects with persistent, remitting, and newly emerging symptoms. Compared with 1991, symptom reports were slightly more frequent in 2002, with the prevalence of regular cough, regular phlegm, and wheezing ranging between 14 and 17%, and the prevalence of chronic cough or phlegm, wheezing with dyspnea, and wheezing without cold reaching 12.9, 6.6, and 8.2%, respectively. The largest relative increase (i.e., by 45%) was observed for chronic...
cough or phlegm. All symptoms were labile: more than 50% of subjects reporting a given symptom in 1991 no longer reported it in 2002. The remission rate was highest for wheezing without cold (64%). In persistent smokers, however, remission rates were less than 50% for regular cough, regular phlegm, chronic cough or phlegm and wheezing. In both surveys, prevalence of symptoms was highest among persistent smokers. Among subjects who quit smoking between 1991 and 2002, reports of all symptoms except wheezing substantially decreased from 1991 to 2002.

On average, home outdoor levels of PM$_{10}$ decreased by 6.2 $\mu$g/m$^3$ in our study population during the 11 years of follow-up (Table 1). Figure 2A shows the distribution of average PM$_{10}$ levels outside the subjects’ homes in the 12 months preceding the two examinations, grouped by the area of residence at SAPALDIA1. For all study areas, the distribution shifted toward lower values from the first to the second survey. This shift was particularly pronounced in Lugano (with a mean decrease of 12.0 $\mu$g/m$^3$) and smallest in the alpine areas of Davos and Montana (with mean decreases of 1.5 $\mu$g/m$^3$ and 3.3 $\mu$g/m$^3$, respectively). Figure 2B shows the distribution of individual differences between follow-up and baseline levels. The interquartile ranges (boxes) were very narrow for the four rural areas of Wald, Payerne, Davos, and Montana and widest for the

<p>| TABLE 1. BASELINE CHARACTERISTICS (ASSESSED IN THE SURVEY OF 1991) OF THE PRESENT STUDY SAMPLE AND OF THE SAMPLE OF SUBJECTS WHO DID NOT PARTICIPATE IN THE SECOND SURVEY OR HAD INCOMPLETE DATA FOR THE PRESENT ANALYSIS |</p>
<table>
<thead>
<tr>
<th>Study Sample</th>
<th>Complementary Sample</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women, %</td>
<td>52.1 (n = 7,019)</td>
<td>47.5 (n = 2,632)</td>
</tr>
<tr>
<td>Age, yr</td>
<td>41.4 [11.4] (n = 7,019)</td>
<td>40.2 [12.3] (n = 2,632)</td>
</tr>
<tr>
<td>BMI, kg/m$^2$</td>
<td>23.8 [3.7] (n = 7,019)</td>
<td>24.3 [4.2] (n = 2,533)</td>
</tr>
<tr>
<td>Smoking history, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never smokers</td>
<td>46.6</td>
<td>36.5</td>
</tr>
<tr>
<td>Former smokers</td>
<td>22.8</td>
<td>21.9</td>
</tr>
<tr>
<td>Current smokers</td>
<td>30.5 (n = 7,019)</td>
<td>41.7 (n = 2,617)</td>
</tr>
<tr>
<td>Education, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low education</td>
<td>14.7</td>
<td>22.7</td>
</tr>
<tr>
<td>Medium education</td>
<td>68.3</td>
<td>61.5</td>
</tr>
<tr>
<td>High education</td>
<td>17.0 (n = 7,019)</td>
<td>15.8 (n = 2,608)</td>
</tr>
<tr>
<td>Non-Swiss nationality, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non-Swiss</td>
<td>14.8 (n = 7,019)</td>
<td>27.2 (n = 2,621)</td>
</tr>
<tr>
<td>$\Delta$PM$_{10}$, $\mu$g/m$^3$</td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\Delta$PM$_{10}$</td>
<td>-6.2 [3.9] (n = 7,019)</td>
<td>-6.1 [5.9] (n = 931)*</td>
</tr>
<tr>
<td>Study area at baseline, %</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Basel</td>
<td>69.7</td>
<td>30.3</td>
</tr>
<tr>
<td>Wald</td>
<td>84.8</td>
<td>15.2</td>
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<tr>
<td>Davos</td>
<td>74.8</td>
<td>25.2</td>
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<tr>
<td>Lugano</td>
<td>75.2</td>
<td>24.8</td>
</tr>
<tr>
<td>Montana</td>
<td>54.7</td>
<td>45.3</td>
</tr>
<tr>
<td>Payerne</td>
<td>69.4</td>
<td>30.6</td>
</tr>
<tr>
<td>Aarau</td>
<td>77.1</td>
<td>22.9</td>
</tr>
<tr>
<td>Geneva</td>
<td>67.9 (n = 7,019)</td>
<td>32.1 (n = 2,632)</td>
</tr>
</tbody>
</table>

**Definition of abbreviations:** PM$_{10}$ = particulates with an aerodynamic diameter of less than 10 $\mu$m; $\Delta$PM$_{10}$ = difference in home outdoor PM$_{10}$ between the two surveys. Values are expressed as percentage or mean [SD].

* Information available only for participants of SAPALDIA2.
three urban areas of Basel, Lugano, and Geneva. The widest
distribution of change was observed in Lugano.

Table 3 shows the estimated adjusted odds ratios for the
symptom reports in 2002 for a 10 μg/m³ decrement in PM$_{10}$
exposure between the two surveys stratified by symptom status
in 1991. The variables adjusted for are listed in a footnote of
the table. For persistent reports of symptoms, the odds ratios
ranged between 0.50 and 0.82. Furthermore, declining levels
of PM$_{10}$ were associated with fewer new reports of most of the
symptoms (except wheezing and wheezing without a cold). Decreased PM$_{10}$ was protective against persistence of regular
cough, chronic cough or phlegm, wheezing, and wheezing
without a cold both in the entire sample and in the subsample
of persistent nonsmokers. In this subsample, effects on persis-
tence (Table 4), significant results were obtained for regular
cough, chronic cough or phlegm and wheezing. A sensitivity
analysis, including additional follow-up variables (e.g., pack-
years smoked between studies) at the expense of a reduction in sample
size estimates generally between those found for incidence and
a longitudinal model. We found similar associations, with effect
size estimates generally between those found for incidence and
persistence at visit two, controlling for symptom status at visit one in
the stratified models (results not shown).

We also found that associations got weaker with increasing
age of the subjects. But none of these differences or interactions
was statistically significant. However, associations were stronger
in subjects who had always lived in the same area. Most of these
differences were nonsignificant, but the beneficial effect of
decreases in PM$_{10}$ on wheezing with dyspnea was seen only in
subjects having always lived in the same area. Similarly, most
associations were slightly stronger among subjects whose addresses
could be geocoded exactly. The decline in PM$_{10}$ and the baseline
level of PM$_{10}$ interacted negatively in their joint effect on most of
the symptom reports, indicating that the protective effects of
a fixed decrease in PM$_{10}$ exposure tended to be larger if initial
levels were lower. These interactions were marginally significant
for new reports of regular phlegm (results not shown).

For further sensitivity analysis, we examined symptom prev-
ance at visit two, controlling for symptom status at visit one in
a longitudinal model. We found similar associations, with effect
size estimates generally between those found for incidence and
persistence in the stratified models (results not shown).

**DISCUSSION**

Data from our population-based cohort study confirm for the
first time that sustained improvements in ambient PM$_{10}$ levels
can lead to decreases in respiratory symptoms among adults.
Not only did decreases in PM$_{10}$ reduce the incidence of res-
piratory symptoms, but they also increased the likelihood of
recovery in persons symptomatic at baseline. Although changes
Interpretation of Results

We observed a moderate increase in the prevalence of respiratory symptoms after 11 years of follow-up and considerable fluctuations in reporting symptoms between the two surveys in 1991 and 2002. Compared with SAPALDIA, the longitudinal Hordaland study in Western Norway found a smaller increase in the prevalence of cough symptoms but a similar increase in wheezing prevalence among its 2,800 adult study participants between 1985 and 1996/97 (26), whereas the European Community Respiratory Health Survey saw no increase in wheezing prevalence, but it did observe an increase in asthma within its younger cohort (27). The rate of symptom remissions in Norway was similar to our findings, with cumulative remission rates between 42 and 58% for chronic cough, cough with phlegm, and wheezing. As in SAPALDIA, the remission rate was lower in smokers, and smoking cessation had a beneficial effect (28). High fluctuations and remission rates of reports of chronic respiratory symptoms were also reported from studies in The Netherlands (29), the United States (30), and from an early study in London (31). The authors of the London study interpreted phlegm production as a temporary effect of bronchial infection, remitting with decreasing exposure to smoking and/or air pollution.

The prevalence of chronic cough or phlegm but not of wheezing symptoms was associated with PM\(_{10}\) in the SAPALDIA baseline study (3). At that time, PM\(_{10}\) measurements from only one central monitoring station per study area were available. In the present study, with individual estimates of home outdoor PM\(_{10}\) exposure, we could show that decreases in outdoor PM\(_{10}\) levels over the 11 years of follow-up were associated with lower rates of new and persistent reports of chronic cough or phlegm. Similar longitudinal associations were found for the other symptoms (except wheezing without a cold) and we estimated that their prevalence would have been between 8 and 17% higher in 2002 if mean PM\(_{10}\) levels had remained constant over time. These effects were seen despite the moderate to low air pollutant levels in Switzerland. They suggest that the respiratory health of adult populations may adapt relatively fast to changing levels of ambient air pollution. This is also consistent with a report from the Harvard Six City Study indicating that changes in PM\(_{2.5}\) exposure over a similar time interval were associated with changes in mortality risk on follow-up (10). A more recent analysis of these data showed that the effects of particles on mortality were driven by exposure in the previous 2 years (32).

Previously, our group reported associations between respiratory symptoms and traffic exposure indicators (e.g., closeness of streets and street densities) for both SAPALDIA surveys (33). These associations were cross-sectional and suggested potentially stronger effects from traffic on the prevalence of regular cough, regular phlegm, wheezing, and attacks of breathlessness. However, due to a lack of data on changes in traffic frequencies and emissions, we were unable to address whether changes in the level of traffic-related particles were associated with changes in symptom reports between the two surveys. Moreover, given that our model-based PM\(_{10}\) estimates do not capture local traffic particles (23), such associations would be largely independent of the ones observed in the present study.

Potential Sources of Bias

A potential source of bias in all cohort studies is loss to follow-up. By including only a minimum of follow-up covariates in our model, we could include 70% of the original participants in our analyses. To examine whether more detailed information on follow-up covariates would change the results, we also performed analyses in a restricted sample with more complete information. This led to only minor changes in the results. Moreover, these
changes were mostly explained by the restriction of the sample and not by the inclusion of additional covariates. An alternative analysis, including the likelihood of participation in the second survey as a potential effect modifier, suggested that we might have slightly underestimated the effects on cough and phlegm and slightly overestimated the effects on wheezing. This would be consistent with the slightly stronger effects for wheezing in the model with more covariates and fewer subjects (see Figure E6).

Our exposure variable was derived from a dispersion model for home outdoor PM$_{10}$. Generally, associations are weakened if exposure is misclassified. But effect sizes may also be overestimated if exposure contrasts are systematically underestimated. This is unlikely in our case, because validation of our exposure model has shown good average agreement between modeled and measured PM$_{10}$ values across and within study areas (23).

Because changes in exposure were generally larger in persons who had moved between surveys, they might have been correlated with socioeconomic factors. In as much as these factors could be assessed at baseline, they were controlled for in our analyses. Moreover, the observed beneficial effects of reduced exposure to PM$_{10}$ were slightly stronger among nonmovers than among the entire sample, showing that our results were not driven by the movers.

The magnitude and distribution of change in exposure differed across areas. Thus, there is a potential risk that associations could have been confounded by unexplained factors varying between areas. However, area-specific slopes did not improve the model for most of the outcomes studied except for new reports of wheezing with dyspnea. Moreover, the results from models with fixed area intercepts adjusting for any previously unexplained differences between areas were only slightly weaker than the ones of the random intercept models (results not shown).

We previously reported that change in PM$_{10}$ exposure was associated with change in lung function in this cohort, with reduced exposure associated with a slower rate of pulmonary function decline. These results strengthen the evidence that reducing exposure to particles improves respiratory health. They also raise the question as to how independent the results are. Whereas respiratory symptoms and level of lung function are correlated, the correlation is modest. To test the hypothesis that change in FEV$_1$ is on the causal pathway between change in PM$_{10}$ exposure and respiratory symptoms, we repeated our analyses controlling for FEV$_1$ (results not shown). Control for FEV$_1$ had little impact on the protective effects of air pollution reduction on symptom incidence or persistence, suggesting these are independent effects.

**Strengths and Limitations of the Present Study**

Our study involves a large population-based cohort including more than 70% of the initially recruited subjects. Moreover, the availability of detailed individual residential histories and a validated PM$_{10}$ dispersion model enabled the study of associations between changes in respiratory symptom reports and changes in individual estimates of home outdoor PM$_{10}$.

This study also has several limitations. We do not know the time course of different size and source fractions of particles between 1991 and 2002. Measurements of PM$_{2.5}$ were initiated in Switzerland only in 1998 after most of the decrease in PM$_{10}$ levels had already happened. However, PM$_{2.5}$ levels were highly correlated with PM$_{10}$ levels between 1998 and 2001 and there is no reason to think that this was different before 1998 (34). PM$_{10}$ levels outside individual residences capture only part of individual exposure to particles. However, they have been shown to strongly correlate with total individual exposure to particulate matter (35, 36). Thus, the direction of bias of our effect estimates depends on the difference in exposure contrasts between the two measures. If contrasts were actually larger for residential than for average personal levels, as some previous results of ours on individual exposure to NO$_2$ might indicate (19), then our effect estimates would be too low.

Because PM$_{10}$ exposure contrasts have decreased during the follow-up period, there is a strong negative correlation between individual change in PM$_{10}$ exposure and individual average exposure between the two studies ($r = -0.63$). Thus, we could

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**TABLE 3. ESTIMATED ODDS RATIOS (95% CONFIDENCE INTERVALS) OF REPORTING THE RESPECTIVE SYMPTOMS AT SAPALDIA2 BY 10 μg/m$^3$ DECREMENT IN ANNUAL HOME OUTDOOR PM$_{10}$ BETWEEN THE TWO INTERVIEWS$^*$**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Entire Sample</th>
<th>Persistent Non-smokers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>New Reports</td>
<td>Persistent Reports</td>
</tr>
<tr>
<td></td>
<td>P Value$^2$</td>
<td>P Value$^2$</td>
</tr>
<tr>
<td>Regular cough</td>
<td>0.77 (0.62 to 0.97)</td>
<td>0.55 (0.39 to 0.78)</td>
</tr>
<tr>
<td>Regular phlegm</td>
<td>0.74 (0.56 to 0.99)</td>
<td>0.82 (0.52 to 1.33)</td>
</tr>
<tr>
<td>Chronic cough or phlegm</td>
<td>0.78 (0.62 to 0.98)</td>
<td>0.67 (0.40 to 1.15)</td>
</tr>
<tr>
<td>Wheezing</td>
<td>1.01 (0.74 to 1.39)</td>
<td>0.50 (0.32 to 0.80)</td>
</tr>
<tr>
<td>Wheezing with dyspnea</td>
<td>0.70 (0.49 to 1.01)</td>
<td>0.59 (0.30 to 1.23)</td>
</tr>
<tr>
<td>Wheezing without cold</td>
<td>1.06 (0.76 to 1.50)</td>
<td>0.61 (0.35 to 1.12)</td>
</tr>
<tr>
<td>Wheezing without cold</td>
<td>0.86 (0.63 to 1.19)</td>
<td>0.28 (0.14 to 0.60)</td>
</tr>
<tr>
<td>Chronic cough or phlegm</td>
<td>0.70 (0.49 to 1.01)</td>
<td>0.59 (0.30 to 1.23)</td>
</tr>
<tr>
<td>Wheezing with dyspnea</td>
<td>1.01 (0.74 to 1.39)</td>
<td>0.50 (0.32 to 0.80)</td>
</tr>
<tr>
<td>Wheezing without cold</td>
<td>0.70 (0.49 to 1.01)</td>
<td>0.59 (0.30 to 1.23)</td>
</tr>
</tbody>
</table>

$^*$ Annual home outdoor PM$_{10}$ was defined as the estimated 12-month mean of home outdoor PM$_{10}$ before the respective interview.

$^2$ Chi-squared test with two degrees of freedom using the sum of the squared t-values of the two stratum-specific parameter estimates as test statistic.
not estimate the independent role of interval exposure reliably. The use of the change in annual PM10 levels before health assessments as the primary exposure measure may be justified if the health parameters studied adapt relatively fast to environmental changes and reflect effects of more recent rather than of long-term exposure. Our results suggest that this may indeed be the case for most of the outcomes studied. Nevertheless, our interaction results suggest that the beneficial effects of reduced PM10 exposure were slightly weaker if initial levels were higher and if persons were older. This may indicate that the degree of reversibility of respiratory conditions decreases with age and with increasing level of past exposure. Alternatively, the presence of an adaptation mechanism might make the percentage of change in exposure more important than the absolute change, which would result in larger improvements in health for the same exposure decrement among individuals with lower baseline exposure.

In conclusion, the present results on respiratory symptoms are in line with our previous findings that decreasing PM10 levels were associated with an attenuated age-related lung-

**TABLE 5. ESTIMATED CHANGES IN THE NUMBERS OF CASES WITH SYMPTOMS AT SAPALDIA2 ATTRIBUTABLE TO THE OBSERVED CHANGES IN ANNUAL HOME OUTDOOR PM10 BETWEEN THE TWO SURVEYS**

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Number of Cases*</th>
<th>Change in Number of Cases †</th>
<th>Number of New Cases Prevented‡</th>
<th>Number of Persistent Cases Prevented§</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regular cough</td>
<td>1,911</td>
<td>-259 [13.6%] (-416, -102)</td>
<td>-139/8,511 [-1.6%]</td>
<td>-119/1,489 [-8.0%]</td>
</tr>
<tr>
<td>Regular phlegm</td>
<td>1,639</td>
<td>-182 [11.1%] (-366, 3)</td>
<td>-145/8,802 [-1.6%]</td>
<td>-37/1,198 [-3.1%]</td>
</tr>
<tr>
<td>Chronic cough or phlegm</td>
<td>1,469</td>
<td>-179 [12.2%] (-328, -30)</td>
<td>-127/9,111 [-1.4%]</td>
<td>-52/889 [-5.8%]</td>
</tr>
<tr>
<td>Wheezing</td>
<td>1,531</td>
<td>-129 [8.4%] (-300, 43)</td>
<td>5/8,719 [+0.1%]</td>
<td>-133/1,281 [-10.4%]</td>
</tr>
<tr>
<td>Wheezing with dyspnea</td>
<td>799</td>
<td>-137 [17.1%] (-266, -9)</td>
<td>-99/9,426 [-1.1%]</td>
<td>-38/574 [-6.6%]</td>
</tr>
<tr>
<td>Wheezing without cold</td>
<td>862</td>
<td>-26 [3.0%] (-152, 101)</td>
<td>19/9,310 [+0.2%]</td>
<td>-45/690 [-6.5%]</td>
</tr>
</tbody>
</table>

For definition of abbreviation, see Table 3.

* Estimated number of cases in 10,000 persons under counterfactual scenario of constant PM10

† Estimated change in number of cases (with 95% confidence interval) among 10,000 persons attributable to the observed changes in PM10. The percentage in brackets gives the relative decrease in the number of cases compared to the counterfactual scenario.

‡ Estimated number of new cases prevented in 2002/number of subjects without the respective symptom in 1991 (per 10,000 subjects)

§ Estimated number of persistent cases prevented in 2002/number of subjects with the respective symptom in 1991 (per 10,000 subjects)
function decline. Both results indicate that reductions in ambient particle concentrations may have beneficial effects on the respiratory health of adults within few years even in areas with moderate to low levels of air pollution. Moreover, they suggest that efforts to reduce particulate pollution should be sustained irrespective of improvements already achieved, as the potential for reversibility of symptoms was even higher at lower initial exposure levels in our study.

Conflict of Interest Statement: None of the authors has a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

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