Air Pollution and Emergency Room Visits for Asthma in Santa Clara County, California

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During the winters of 1986–1987 through 1991–1992, rainfall throughout much of Northern California was subnormal, resulting in intermittent accumulation of air pollution, much of which was attributable to residential wood combustion (RWC). This investigation examined whether there was a relationship between ambient air pollution in Santa Clara County, California and emergency room visits for asthma during the winters of 1988–1989 through 1991–1992. Emergency room (ER) records from three acute-care hospitals were abstracted to compile daily visits for asthma and a control diagnosis (gastroenteritis) for 3-month periods during each winter. Air monitoring data included daily coefficient of haze (COH) and every-other-day particulate matter with aerodynamic diameter equal to or less than 10 microns (PM_{10}, 24-hr average), as well as hourly nitrogen dioxide and ozone concentrations. Daily COH measurements were used to predict values for missing days of PM_{10} to develop a complete PM_{10} time series. Daily data were also obtained for temperature, precipitation, and relative humidity. In time–series analyses using Poisson regression, consistent relationships were found between ER visits for asthma and PM_{10}. Same-day nitrogen dioxide concentrations were also associated with asthma ER visits, while ozone was not. Because there was a significant interaction between PM_{10} and minimum temperature in this data set, estimates of relative risks (RRs) for PM_{10}-associated asthma ER visits were temperature-dependent. A 60 $\mu g/m^3$ change in PM_{10} (2-day lag) corresponded to RRs of 1.43 (95% CI = 1.18–1.69) at 20°F, representing the low end of the temperature distribution, 1.27 (95% CI = 1.13–1.42) at 30°F, and 1.11 (95% CI = 1.03–1.19) at 41°F, the mean of the observed minimum temperatures. ER visits for gastroenteritis were not significantly associated with any pollutant variable. Several sensitivity analyses, including the use of robust regressions and of nonparametric methods for fitting time trends and temperature effects in the data, supported these findings. These results demonstrate an association between ambient wintertime PM_{10} and exacerbations of asthma in an area where one of the principal sources of PM_{10} is RWC. *Key words*: asthma, emergency room visits, epidemiology, particulate air pollution, wood smoke. *Environ Health Perspect* 105:216–222 (1997)

Beginning with the winter of 1986–1987, Northern California experienced a 6-year drought during which many inland valleys were subject to intermittent air stagnation and decreased visibility. During this period, there were numerous anecdotal reports that smoke from residential wood combustion (RWC) was causing adverse respiratory effects. There are an estimated 1–1.5 million fireplaces and 300,000 wood-burning stoves in the San Francisco Bay Area (*1*). Source apportionment analysis sponsored by the Bay Area Air Quality Management District (BAAQMD) has shown that, on average, RWC accounts for approximately 45% of winter PM_{10} (particulate matter less than or equal to 10 μm in aerodynamic diameter) in Santa Clara County, located at the southern end of the Bay Area (*2*). During the source apportionment sampling, RWC was the largest single identified source of winter PM_{10} in Santa Clara County, quantitatively approximating the sum of motor vehicle emissions and entrained road dust. Wood smoke contains many respiratory irritants in addition to particles, including low molecular weight aldehydes and acids, nitrogen oxides, and sulfur dioxide (*3*). Indoor exposures to this complex mixture have previously been linked with increased risks of respiratory infection and otitis, increased symptoms of respiratory irritation, and exacerbations of asthma symptoms (*4–8*). Several studies undertaken in the Pacific Northwest suggest that ambient particles, many of which are attributable to RWC, are linked with decrements in children's lung function and increased hospital emergency room (ER) visits for asthma (*9–11*). This investigation was initiated to examine the relationship between ER visits for asthma and ambient air pollutant concentrations in Santa Clara County during the winters of 1988–1989 through 1991–1992.

Data and Methods

Visits for asthma to three acute-care facilities in Santa Clara County for the winters of 1988–1989 through 1991–1992 (1 November–31 January) were abstracted from ER logbooks by one of the coauthors (Hurley) and an epidemiology graduate student. Visits for gastroenteritis, a control diagnosis considered unlikely to be related to air pollution, were also abstracted. Daily counts of ER visits for asthma and gastroenteritis were compiled for each hospital. Summed hospital-specific counts were used as the primary dependent variable in the analysis, since preliminary analyses had indicated the presence of significant interhospital heterogeneity, possibly due to differences in diagnostic preferences and in the populations served (one facility was a county hospital, whereas the other two were private). Air monitoring data were obtained from the BAAQMD for the principal San Jose monitoring site, centrally located in the Santa Clara Valley. Particulate matter (PM) metrics included coefficient of haze (COH), a measure of light transmittance, which was recorded every 2 hr, and PM_{10}, which was recorded as a 24-hr average with a high-volume sampler every other day. For one 45-day period during the 1991–1992 winter, however, PM_{10} was measured only every sixth day. Ozone (O_3) and nitrogen dioxide (NO_2) were measured continuously and reported by the BAAQMD as hourly averages. We regressed measured PM_{10} on corresponding daily average values of COH in order to predict missing PM_{10} values so that we could conduct analyses with a daily PM_{10} metric. The R^2 value for this linear predic-

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tive model for the winters of 1988–1989 through 1991–1992 was 0.81. Meteorological data included daily temperature, relative humidity, and precipitation, which were obtained from the National Climatic Data Center in Asheville, North Carolina. The meteorological, air quality, and health data were entered and merged for analysis using PC-SAS (SAS Institute, Cary, NC), S-Plus (StatSci, Seattle, WA), and Stata (Stata Corp, College Station, TX) (*12–14*).

The principal analysis relied on Poisson regression, which assumes that counts of independent, rare events follow a Poisson distribution, conditional on the explanatory variables. However, heterogeneity among the asthmatics' exposures, respiratory infections, and other factors may result in overdispersion of the data in relation to a classic Poisson distribution. This in turn may affect the standard errors of the regression coefficients, leading to incorrect significance tests. To address this issue, the extra-Poisson variability was modeled and incorporated into the estimates of the standard errors using PROC GENMOD in PC-SAS, which adopts the approach of McCullagh and Nelder (*15*). While the estimated coefficients remain the same as in Poisson regression, the standard errors are estimated by multiplying those obtained from the Poisson model by a dispersion parameter.

Initially we ran cross-correlations of the various explanatory variables to examine whether multicollinearity would be a concern in subsequent analyses. Pollutant variables found to be strongly correlated ($r \geq 0.6$) were not included together in the regression models. We then ran univariate regressions of asthma ER visits on each of the explanatory meteorological and pollutant variables. In general, covariates were examined in multivariate models if their t-statistics in the univariate regressions were equal to or greater than one. The PM variables were examined as daily average COH and PM_{10} (described above). Gaseous pollutants were modeled as daily peak 1-hr concentrations of O_3 and NO_2. The influence of temperature was examined by using daily minimum temperatures (including several lagged specifications) and by running models with locally weighted regression (loess) smooths of temperature (spans of 90 and 45 days) (see below). Percent relative humidity (measured daily at 4 P.M.) was examined as a continuous variable, with contemporaneous and lagged values of up to 4 previous days considered. Precipitation was modeled as a binary variable. To control for interhospital differences in ER utilization, the regression models also included indicator variables representing each hospital since, as noted above, preliminary analysis indicated the presence of significant interhospital heterogeneity. Separate sets of indicator variables were also included to model short-term (i.e., day of week) and long-term (i.e., annual) trends in ER utilization. Several terms were used to model potential interactions between temperature and the pollutant variables.

Numerous reports document a variety of lag structures relating ambient particle concentrations to both morbidity and daily mortality. Schwartz and colleagues recently reported that the strongest associations of PM_{10} and asthma ER visits in Seattle were found when the explanatory variable was defined as the mean of the previous 4 days' concentrations of PM_{10} (*11*). We investigated the effects of up to 5-day lags as well as multiday averages of PM_{10}.

Positive serial correlation is a common feature of time–series data, and can result in underestimation of the standard errors of regression coefficients. Examination of autocorrelation function (ACF) plots of the model residuals showed slight serial correlation only at lag 1 ($\rho = 0.12$–0.14 for the various models). This minimal degree of serial correlation is unlikely to produce biased significance tests (*16*). Nevertheless, to address this issue, we reran the models using the general estimating equations (GEE) of Liang and Zeger (*17*). In this approach, the covariance structure is incorporated into the estimation of the regression coefficients in addition to their variances, theoretically yielding robust estimators and correcting for serial correlation in the data. Though this method has been used in other time–series data sets (*11*), recent simulations suggest that when the data are structured in few independent blocks, the GEE model may overstate the significance of regression coefficients. Accordingly, when using the GEE, we structured the data into 12 blocks (by hospital and year). Unlike Burnett and colleagues, we did not structure these blocks as random effects variables (*18*).

Models that incorporate loess smooths of time also have been used successfully to reduce residual serial correlation in time–series data similar to these. When an explanatory variable, smoothed in this fashion, is incorporated into a regression, the measured value of the variable is replaced with a locally weighted moving average and the regression is conducted on the moving average. Thus, adding a loess smooth of time diminishes short-term fluctuations in the data, thereby helping to reduce the degree of residual serial correlation. This smoothing technique also allows for more parsimonious modeling of annual temporal trends in the data than the use of indicator variables in standard Poisson regressions. Furthermore, loess smoothing techniques can accommodate nonlinear patterns, offering a more flexible nonparametric modeling tool. Therefore, to control for temporal trends in the data and to allow for potential nonlinearities in the effect of temperature, we repeated the analysis using generalized additive models with loess smooths of time and temperature. Because our data represent discontinuous time series across years, we adjusted the span of the smooth to both 90 days (the length of one winter) and 45 days (half of the winter span).

We undertook several additional sensitivity analyses, which included 1) conducting robust regressions (to minimize the effects of outliers and other potentially influential data points) using an iteratively reweighted least squares methodology; 2) incorporating several combinations of trigonometric terms to model and thereby reduce the impact of any long-wave trends within the winter seasons that were not obvious by visual inspection of the data; and 3) fitting the same models to ER visits for gastroenteritis (the control diagnosis) as those used for asthma.

Results

Descriptive statistics for the variables used in this investigation are displayed in Table 1. The mean PM_{10} concentration during the study period was 61.2 $\mu g/m^3$. During the 368 observation-days of this investigation, there were 2 measured exceedances of the federal ambient air quality standard for PM_{10} (150 $\mu g/m^3$, 24-hr average), and 85 of the California standard (50 $\mu g/m^3$, 24-hr average). Using COH readings to predict

Table 1. Descriptive statistics for health, pollutant, and meteorological variables, Santa Clara County, California, in the winters of 1988–1989 through 1991–1992

Variable	Mean ± SD	Range
Asthma ER visits/day	7.6 ± 3.3	0–19
Gastroenteritis ER visits/day	9.3 ± 4.4	1–28
COH (average × 10)	11.4 ± 6.5	1.3–31.3
PM_{10} ($\mu g/m^3$)[a]	61.2 ± 32.6	9–165
O_3 (1-hr peak; ppm)	0.024 ± 0.01	0.005–0.07
NO_2 (1-hr peak; ppm)	0.069 ± 0.028	0.029–0.15
Minimum temperature (°F)	41.6 ± 6.6	19–60
Maximum temperature (°F)	60.4 ± 6.7	38–80
Relative humidity (%)	49.8 ± 17.8	13–100
Precipitation (inches)	0.03 ± 0.13	0–0.9

Abbreviations: SD, standard deviation; ER, emergency room, COH coefficient of haze; PM_{10}, particulate matter with aerodynamic diameter equal to or less than 10 microns.

[a]Complete PM_{10} data set, including both measured values and those predicted from COH. The corresponding values for measured PM_{10} (every other day) were $\mu = 60.7$, range = 9–165, SD = 32.3.

PM_{10} on days when it was not measured, however, resulted in estimates of 3 and 202 exceedances of the federal and state standards, respectively. O_3 concentrations were generally at or near background levels throughout most of the observation period, as expected, since ground-level O_3 is generated photochemically and attains elevated concentrations primarily from April to October in California. NO_2 concentrations were substantially below the California 1-hr ambient air quality standard (0.25 ppm) during the entire study. On most days there was little or no precipitation.

Table 2. Correlation matrix for pollutant and meteorological variables, Santa Clara County, California, in the winters of 1988–1989 through 1991–1992[a]

	COHAv	NO_2[b]	Ozone[b]	Min temp
PM_{10}[c]	0.9496	0.8173	-0.1536	-0.4751
COHAv	1.0000	0.8026	-0.1655	-0.5024
NO_2	—	1.0000	-0.0148	-0.2584
Ozone	—	—	1.0000	0.2260

Abbreviations: COHAv, 24-hr average COH; COH, coefficient of haze; NO_2, nitrogen dioxide; Min temp, minimum daily temperature; PM_{10}, particulate matter with aerodynamic diameter equal to or less than 10 microns.
[a]Pearson coefficients; all correlations are significant ($p<0.01$) except r_{NO_2,O_3} ($p = 0.623$).
[b]Maximum 1-hr average.
[c]Daily 24-hr average, complete PM_{10} data set, including both measured values and those predicted from COH.

Table 2 is a correlation matrix of pollutant and meteorological variables. Minimum temperature was negatively associated with PM_{10}, COH, and NO_2, which would be expected to occur in the presence of the shallow thermal inversions common during Northern California winters. NO_2 was strongly correlated with the particulate measures and thus was not used in any initial regressions with PM_{10} as an explanatory variable.

In this paper, a relative risk (RR) reported for a pollutant variable represents the change in daily risk of seeking ER treatment for asthma associated with a given unit change in the ambient level of that pollutant. For PM_{10}, we calculated RRs associated with a 60-unit change. Specifically, the RRs we report for PM_{10} represent ratios of the expected counts of asthma ER visits on days when 24-hr average ambient levels of PM_{10} were 60 $\mu g/m^3$ higher than on comparison days. While this is a moderately large pollution difference, it is well within the range of PM_{10} observed and is approximately equal to the mean PM_{10} levels for the study period. The initial estimates of the PM_{10}-associated RRs for asthma ER visits were based on regressions that did not include a term for interaction between PM_{10} and minimum temperature. These RRs ranged from 1.52 to 1.79, depending on the PM_{10} metric used (i.e., unlagged and lagged pollutant variables as well as multiday averages), when calculated at the approximate mean concentration of PM_{10} (60 $\mu g/m^3$). The 3-day average PM_{10} concentration attained statistical significance (RR = 1.73, 95% CI = 1.00–2.97, $p = 0.048$); all other PM_{10} regression coefficients were of borderline significance ($p = 0.06$).

Figure 1 is a plot of the joint influence of PM_{10} and minimum temperature on asthma ER visits. This graph suggests an interactive effect of PM_{10} and temperature primarily on days when the minimum temperature was less than the mean. Inclusion

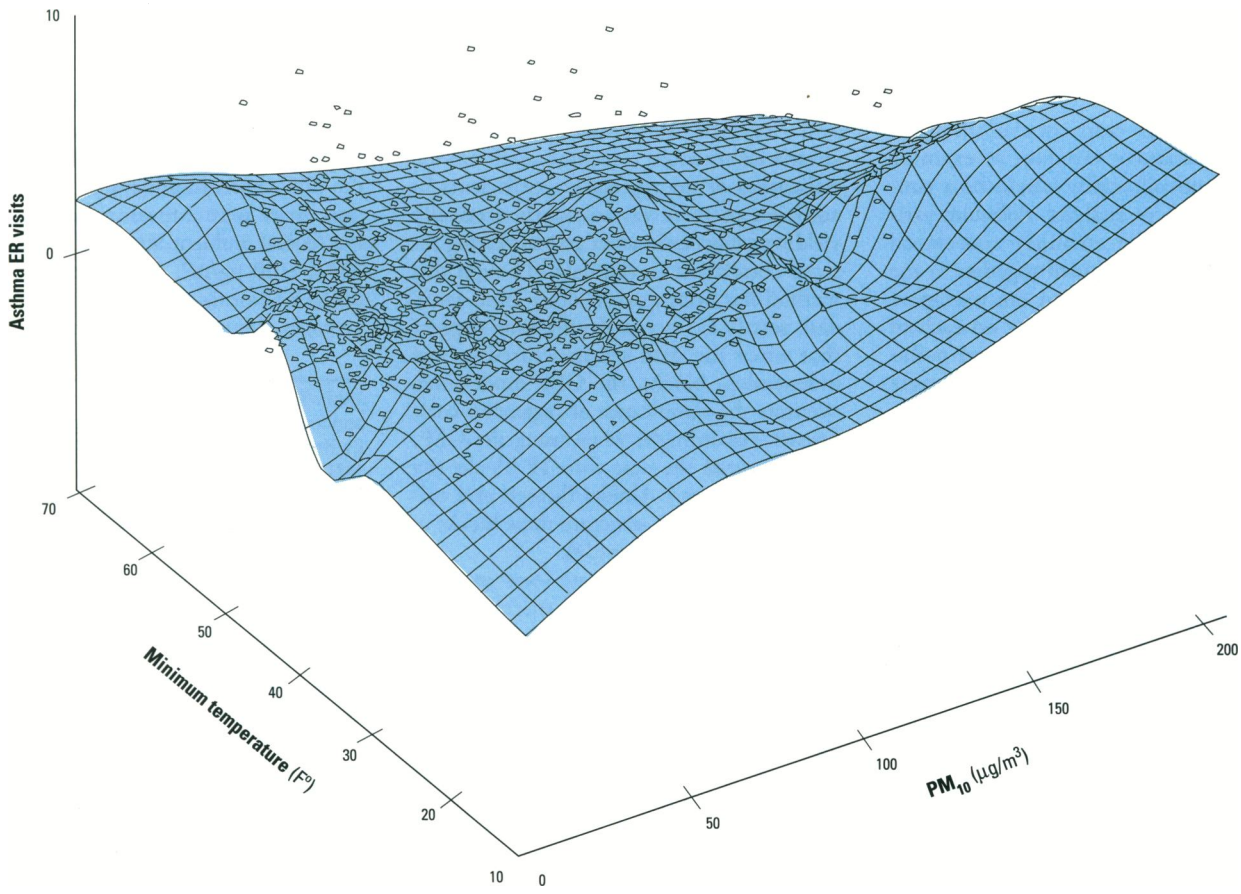

Figure 1. The joint influence of PM_{10} and minimum temperature on asthma emergency room visits, Santa Clara County, California, in the winters of 1988–1989 through 1991–1992.

of an interaction term (PM × minimum temperature) increased the magnitude of the PM_{10} regression coefficients and resulted in statistical significance for all specifications of PM_{10}, as displayed in Table 3. The coefficient for the interaction term included in the regression models, though of small magnitude, was significant ($p<0.05$ in all but one model). In these models, the RR estimates for PM_{10} depend on the value of the interacting variable (i.e., minimum temperature). Therefore, we estimated RRs and confidence intervals using formulas that incorporated specific values for minimum daily temperature and the covariance of PM_{10} and the interaction term (*19*).

The formulas for calculating RR given the interaction between PM_{10} and temperature took the following form:

$$RR = \exp\left(\hat{\beta}_1(PM_{10}) + \hat{\beta}_1 \text{ (minimum temperature) } (PM_{10})\right] \pm t1\text{-}0.95/2\ \hat{SE}\{\ln \hat{RR}\})$$

where $\hat{\beta}_1$ = regression coefficient for a given PM_{10} increment

and $\hat{\beta}_3$ = regression coefficient for the minimum temperature × PM_{10} interaction term.

The estimate of the variance from which the SE was derived was given by:

$$\hat{var}\{\ln(\hat{RR})\} = \hat{var}(\hat{\beta}_1)(PM_{10})^2 + \hat{var}(\hat{\beta}_3)[(\text{minimum temperature})(PM_{10})]^2 + 2\hat{cov}(\hat{\beta}_1, \hat{\beta}_3)(\text{minimum temperature})(PM_{10})^2$$

The results of these analyses are displayed in Table 3. As expected, the estimated RRs were strongly temperature-dependent. At the low end of the temperature distribution (20°F), the RRs ranged from 1.33 (3-day average) to 1.66 (1-day lag of PM_{10}). At 30°F, the RRs for most of the specifications of PM_{10} were statistically significant, though the magnitudes of the RRs were less than those calculated at the lower temperature. At 41°F, the mean minimum daily temperature for the study period, only the 2-day lag of PM_{10} remained significant (RR = 1.11, 95% CI = 1.03–1.19). The GEE models produced somewhat lower estimates of the RRs, and none calculated at 41°F were significant. In contrast, the robust regressions resulted in somewhat higher estimates of PM_{10}-associated RRs at 20°F and 30°F, but not at 41°F. Specification of a variety of trigonometric terms in the regression equations did not substantially affect the results. Table 4 compares the results from the Poisson, robust, GEE, and trigonometric model regressions for a 2-day lagged specification of PM_{10}.

Models run with loess smooths of temperature and time (with spans of 90 or 45 days) did not markedly change the magnitude of the PM_{10} coefficients, though in several instances their significance decreased slightly. However, in these instances, coefficients for all various specifications of PM_{10} remained significant at $p\leq0.05$, except the 4-day lag. When the regressions were analyzed with temperature stratified into two levels (at the mean), the PM_{10} coefficients remained significant only for temperatures ≤41°F. Point estimates for RRs corresponding to a 60 μg/m³ change in PM_{10} (at 20°F), using only the days when $T<41$°F, ranged from 1.41 (4-day lag) to 2.17 (no lag), somewhat higher than the estimates derived using the entire data set. Insufficient statistical power precluded additional stratification on temperature. The addition of loess smooths of temperature and time to the regression models did not eliminate the small degree of autocorrelation at lag one.

Other terms that were consistently associated with the outcome variable included the indicators for precipitation, hospital, day of week, day of study, and year. Relative humidity was not significantly related to ER visits for asthma in any of the initial models (p-values ranged from 0.5–0.7) and therefore was dropped from all subsequent runs. The models using COH as the particulate metric were not markedly different from those using PM_{10}; in general, models with PM_{10} fit the data slightly better (data not shown). Using similar models with contemporaneous and lagged exposures, asthma ER visits were not significantly associated with O_3 (e.g., for same-day ozone, using the full model, β_{O_3} = -0.003 [$p>0.29$]). However, same-day NO_2 was associated with ER visits for asthma (β_{NO_2} = 0.013 [p = 0.024]). When included as an explanatory variable in regressions along with PM_{10}, the NO_2 coefficient became insignificant, while that for PM_{10} did not change in magnitude and remained statistically significant. In the models that included NO_2 or O_3 rather than PM_{10} as the pollutant variable, the temperature coefficient became highly significant ($p<0.01$). In models using unlagged, lagged, and multiday averages of pollutant variables, gastroenteritis ER visits were not significantly associated with PM_{10}, COH, NO_2 or O_3 (data not shown).

Table 3. Regression coefficients and relative risks (RRs) for asthma-related emergency room visits, Santa Clara County, California, in the winters of 1988–1989 through 1991–1992[a]

Specification of PM_{10}	PM_{10} β	PM_{10} p-value	RR (95% CI) Min temp = 20°F	Min temp = 30°F	Min temp = 41°F
Same Day	0.0115	0.012	1.39 (1.11–1.67)	1.16 (1.00–1.32)	0.95 (0.86–1.05)
1-Day lag	0.0113	0.009	1.66 (1.27–2.06)	1.53 (1.06–2.00)	1.39 (0.78–2.01)
2-Day lag	0.0100	0.018	1.43 (1.18–1.69)	1.27 (1.13–1.42)	1.11 (1.03–1.19)
3-Day lag	0.0127	0.002	1.49 (1.24–1.75)	1.25 (1.10–1.39)	1.02 (0.94–1.11)
4-Day lag	0.0093	0.047	1.37 (1.09–1.66)	1.22 (1.06–1.38)	1.07 (0.98–1.16)
2-Day average	0.0095	0.051	1.39 (1.10–1.69)	1.23 (1.07–1.40)	1.08 (0.99–1.17)
3-Day average	0.0108	0.030	1.33 (1.03–1.64)	1.11 (0.94–1.29)	0.91 (0.81–1.01)
4-Day average	0.0119	0.020	1.42 (1.11–1.74)	1.19 (1.01–1.37)	0.98 (0.88–1.07)
5-Day average	0.0122	0.024	1.45 (1.12–1.78)	1.21 (1.03–1.40)	0.99 (0.89–1.10)
Average of previous 2 days	0.0094	0.037	1.38 (1.11–1.66)	1.23 (1.07–1.38)	1.07 (0.99–1.16)
Average of previous 3 days	0.0112	0.018	1.37 (1.08–1.65)	1.14 (0.98–1.30)	0.94 (0.85–1.02)
Average of previous 4 days	0.0113	0.027	1.37 (1.06–1.69)	1.15 (0.97–1.32)	0.94 (0.85–1.03)

PM_{10}, particulate matter with aerodynamic diameter equal to or less than 10 microns.
Coefficients and RRs derived from multivariate Poisson regressions, which included terms for pollutant metric, minimum temperature (min temp), an interaction term for PM_{10} and min temp, day of study, and indicator variables for precipitation, hospital, day of week, and year, and an overdispersion parameter.
[a]Calculated at the approximate mean value of PM_{10} (60 μg/m³).

Table 4. Regression coefficients and relative risks (RRs) for asthma-related emergency room visits estimated from different models, Santa Clara County, California, in the winters of 1988–1989 through 1991–1992[a]

Model	PM_{10} β	PM_{10} p-value	RR (95% CI) Min temp = 20°F	Min temp = 30°F	Min temp = 41°F
Poisson regression	0.010	0.018	1.43 (1.18–1.69)	1.27 (1.13–1.42)	1.11 (1.03–1.19)
Robust regression	0.024	0.019	2.13 (1.52–2.75)	1.52 (1.17–1.87)	1.05 (0.85–1.25)
GEE	0.009	0.003	1.33 (1.15–1.50)	1.15 (1.04–1.27)	0.99 (0.90–1.09)
Model with trigonometric terms	0.009	0.028	1.37 (1.12–1.63)	1.22 (1.08–1.36)	1.07 (0.99–1.15)

Abbreviations: PM_{10}, particulate matter with aerodynamic diameter equal to or less than 10 microns; GEE, generalized estimating equations of Liang and Zeger (*17*).
All models included terms for lag 2 of PM_{10} and minimum temperature (min temp), an interaction term for PM_{10} and min temp, day of study, and indicator variables for precipitation, hospital, day of week, and year, and an overdispersion parameter.
[a]Calculated at the approximate mean value of PM_{10} (60 μg/m³).

Discussion

In this investigation we found that a variety of specifications of PM_{10} were consistently associated with ER visits for asthma, but not for gastroenteritis. Several lagged specifications for PM_{10} provided modestly stronger associations with asthma ER visits than did same-day PM_{10}. One explanation for this observation may lie in the pattern of exposure to winter particles in the Santa Clara Valley. Visual inspection of the 2-hr COH values and the results of more recent real-time monitoring data indicate that PM levels generally tend to increase markedly in the late afternoon and evening, a pattern that is consistent with RWC emissions. Peak levels, therefore, often occur in the evening, suggesting that if severe symptomatic reactions to exposure (manifested by a visit to the ER) were delayed by more than a few hours, they would not be observed until the following day or later.

In Santa Clara County, the main source of winter particle concentrations is RWC, though motor vehicle exhaust and entrained road dust also make significant contributions (*2*). Wood smoke particles arise mainly from condensation of combustion gases and therefore tend to be distributed mainly in the submicron range, allowing substantial penetration to the indoor environment from outdoors (*3*). Wood smoke can also enter a residence directly via backdrafting from a fireplace or wood stove: It is possible that some of the asthmatics who later sought care at the local ER were exposed to smoke emitted from wood-burning devices in their own homes. A recent report indicates that use of a fireplace or wood stove on a given day strongly predicts exacerbation of respiratory symptoms in adults who have moderate or severe asthma (*8*). However, because of the ecological design of this ER visit study, we could not assess individual exposures.

Others have also found delayed asthmatic responses to particulate or smoke exposure (*20,21*). In an analysis of ER visits after the Berkeley-Oakland hills firestorm of 1991, Shusterman and colleagues found that the mean lag between exposure to smoke and a visit to the ER was between 1 and 2 days, which is also consistent with our findings [D. Shusterman, personal communication; (*21*)]. In Seattle, an area that has also experienced substantial wood smoke pollution, Schwartz and co-workers found that the mean PM_{10} concentration averaged over a 4-day period was the best predictor of ER asthma visits, also suggesting a delayed response (*11*). In a study of daily mortality in Santa Clara County in relation to particulate air pollution, Fairley found that the strongest association with the outcome was a 2-day lag (*22*). Although daily mortality is clearly not directly comparable to asthma ER visits, finding delayed particle-associated adverse health events in the same locale lends additional plausibility to each report.

Our analysis also controlled for meteorological factors, and suggested that the combination of low temperature and particle concentrations is also an important predictor of asthma ER visits. Inclusion of the $PM_{10} \times$ minimum temperature interaction term increased both the magnitude and statistical significance of the PM_{10} coefficient, while the opposite was true for the coefficient for minimum temperature. In most model specifications the interaction term itself, though of small magnitude, was significant ($p<0.05$) and its inclusion improved the overall model fit. The significance of the PM $\times$ minimum temperature term in these regressions may be partly attributable to not having specified temperature adequately in other models. However, other specifications, including loess smooths of temperature, did not significantly improve the model fit without the interaction term.

Interpretation of this statistical interaction in meaningful biological terms is somewhat speculative. While exposure to cold dry air can precipitate bronchospasm in asthmatics, temperature was not strongly correlated with asthma ER visits in models that also included PM_{10}. However, in models where the pollutant variable was O_3 or NO_2, temperature was strongly associated with asthma ER visits. This difference may be attributable in part to the inverse correlation between PM_{10} and temperature ($r \approx -0.41$ for measured PM_{10} and $r \approx -0.48$ for the full PM_{10} data set, including the days predicted from COH). During Bay Area winters, radiative inversions associated with low temperature frequently limit the vertical mixing depth to less than 100 ft, trapping pollutants near ground level. Moreover, people tend to use their fireplaces and wood stoves more often as the temperature drops. Thus, the association of asthma ER visits with higher PM_{10} concentrations in conjunction with colder days and nights may be partially explicable by both meteorology and human behavior.

Decreasing ambient temperatures tend to be associated with increasing time spent inside, thus enhancing the likelihood of exposure to indoor allergens and pollutants (including backdrafted wood smoke). Acute exposures to several indoor sources of combustion (gas stoves, cigarette smoke, and wood-burning devices) have been reported to enhance the probability of an exacerbation of asthma in those with moderate to severe disease, which would include individuals likely to require intermittent urgent care (*8*). However, as noted earlier, the ecological design of this investigation precluded evaluation of individual exposures, which may confound or modify the relationships observed between ambient PM_{10} levels and asthma ER visits.

The results are not likely to have been confounded by other measured pollutants or meteorological factors. Ozone has been associated with ER visits or increased symptoms of asthma in other settings, but in those situations the concentrations were substantially higher than here (*23–25*). The strong correlation of NO_2 with PM_{10} and COH probably reflects in part the contribution of motor vehicle emissions to winter particle loading. Nevertheless, NO_2 was associated with the outcome only for same-day exposures. Although NO_2 is a strong respiratory irritant, chamber studies of asthmatics suggest that the NO_2 concentrations observed in this study would be unlikely to elicit a bronchoconstrictive response (*26,27*). However, participants in chamber studies do not represent the spectrum of disease in the general population and the short (usually 1- or 2-hr) durations of the controlled studies cannot adequately capture the complexity of real-world exposures. Others have reported a relationship between ambient NO_2 and ER visits for asthma and with pulmonary function changes in asthmatics (*28,29*). Nevertheless, the absence of an association between lagged or multiday specifications of NO_2 and asthma ER visits in this data set, in addition to the observation that the NO_2 regression coefficient lost its statistical significance in models that also included PM_{10}, suggest that the same-day association may be an artifact of covariation with PM_{10}. This association, in turn, could also be due to the shallow thermal inversions in the Santa Clara Valley.

Another possible explanation for the apparently stronger association of PM_{10} than NO_2 with asthma ER visits may lie in the greater likelihood of exposure misclassification for the latter pollutant. High-temperature combustion results in the formation of NO_2 and other nitrogen oxides, which tend to be elevated near streets and freeways with substantial traffic volume. Thus, a single NO_2 monitoring site, as was used in this analysis, may not be representative of regional concentrations or personal exposures. In examining this issue, we ran pair-wise correlations among peak hourly NO_2 concentrations at three regional fixed-site monitors, including the one used in this analysis, the coefficients of which ranged from 0.73 to 0.78 during the study period. Similar correlation coefficients (based on every-sixth-day sampling) among

24-hr averages for PM_{10} at the same sites ranged from 0.90 to 0.92. Thus, even if NO_2 exposure was causally related to serious asthma exacerbations, these results suggest that exposure misclassification alone could have resulted in an apparently stronger relationship of ER asthma visits with PM_{10} than with NO_2, since such misclassification tends to bias the results towards the null hypothesis of no effect.

Sulfur dioxide (SO_2), another respiratory irritant to which asthmatics tend to be susceptible, was not measured by the BAAQMD during the study period because SO_2 concentrations in prior years had been far below both the California and the federal ambient air quality standards. In 1988, for example, the last year for which SO_2 was measured at this site, the peak 1-hr concentration was 4 ppb, whereas the annual average was 0.55 ppb. [For purposes of comparison, the California ambient air quality standard for SO_2, which is intended to protect individuals with asthma, is 250 ppb (1-hr average)]. With such low SO_2 concentrations, the sulfate fraction of PM_{10} also tends to be quite low compared with other regions in the United States. During the winters of 1988–1991, sulfates composed only about 4–5% of PM_{10} mass measured at the same monitoring site as that used in this analysis (*30*). In contrast, in urban areas on the East Coast, the comparable percentage is closer to 30% (*31*).

Since some fungal spores and pollen fragments are within the size range encompassed by PM_{10}, it is possible that the increased RRs reported here may have been confounded by exposure to aeroallergens, some of which are well recognized to bear a causal relationship to seasonal and epidemic asthma exacerbations. While theoretically possible, such confounding is unlikely to explain the associations observed here, where the PM_{10}-associated risk increased with decreasing temperature. Viral epidemics are also often cited as potential confounders of associations between air pollution and asthma or other respiratory conditions. We did not have data available to control for this potential confounder. Moreover, to the extent that respiratory viral infections may constitute an intermediate stage on a causal pathway between exposure to PM_{10} and asthma exacerbations, it would be inappropriate to control for this variable in an analysis of potential air pollution effects (*32*).

Our results indicate that a 10 $\mu g/m^3$ increase in PM_{10} (2-day lag) would correspond to a relative risk of 1.06 (95% CI = 1.02–1.10) at 20°F, declining to 1.04 (95% CI = 1.02–1.06) at 30°F, and 1.02 (1.00–1.03) at the mean observed minimum temperature (41°F) using the Poisson model. These estimates were slightly lower using the GEE model (RR = 1.05; 95% CI = 1.02–1.08) and slightly higher using robust regressions (RR = 1.13; 95% CI = 1.03–1.24). In Seattle, where RWC has also been a major source of winter particles, Schwartz and co-workers reported a RR of 1.12 for a 30 $\mu g/m^3$ increase in PM_{10} for asthma ER visits, but their estimate was based on a multiday average of PM_{10} concentrations throughout the year (*11*). Using our data, a 30 $\mu g/m^3$ change in PM_{10} (lag 2) would result in a RR of 1.20 (95% CI = 1.07–1.33) at 20°F, a RR of 1.13 (95% CI = 1.06–1.20) at 30°F, or 1.06 (95% CI = 1.02–1.10) at 41°F. Though Schwartz et al. did not report any PM_{10}-temperature interactions, their RR estimates are of comparable magnitude to ours.

Whether these results could be replicated in seasons other than winter has not been examined. Given that the relationship between ER visits and PM_{10} concentrations was observed primarily at lower temperatures, it is possible that this association would not hold during other seasons. Moreover, though RWC takes place throughout the year in the Bay Area, overall PM_{10} levels and absolute concentrations of RWC-associated particles are generally lower in the spring, summer, and fall. On the other hand, during other seasons ambient levels of ozone and aeroallergens are more likely to be elevated, which could also increase the risk of ER visits for asthma. We are currently examining the relationships of PM_{10} and other ambient pollutants to respiratory and cardiovascular morbidity in the Bay Area throughout the year.

Our results are consistent with numerous other recent reports linking airborne particles to adverse respiratory outcomes when measured outdoor concentrations are lower than the federal ambient air quality standard for PM_{10} (*33*). To the extent that the associations noted in this report represent causal relationships, it is plausible that the heterogeneous categories of substances subsumed by PM_{10} (or fine particles, which represent the bulk of RWC-related particles and which also are capable of substantial penetration to the indoor environment) may be responsible. In this study of winter air pollution and asthma, however, airborne particles represent but one of many respiratory irritants in wood smoke, including formaldehyde, acrolein, acetaldehyde, acetic acid, phenol, and nitrogen oxides, among others (*3*). Though the mean contribution of wood smoke to particle mass was reported to be approximately 45%, in some of the narrow inland valleys in Santa Clara County the percentage is likely to have been substantially higher. Thus, though PM_{10} is a routinely monitored indicator for wood smoke, the respiratory toxicity of the mixture, rather than particles per se, may have driven the relationships reported here. On the other hand, since approximately half of the winter particle mass during this study period was attributable to sources other than RWC, one cannot directly link the elevated RRs observed here exclusively to the latter.

Unlike most other human activities that generate air pollution, RWC remains largely unregulated, despite substantial contributions to atmospheric particle loading in many areas of North America, especially in the western United States. It is also a major source of indoor pollution in many developing countries. Though wood combustion is probably the oldest form of anthropogenic pollution, there is a paucity of data on health impacts of smoke inhalation except at high concentrations. Future research should address effects of ambient levels of wood smoke on sensitive subpopulations (i.e., asthmatics) in chamber studies and in epidemiologic investigations in areas where RWC is the primary source of air pollution.

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