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Increases in Heart Rate during an Air Pollution Episode

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This paper assesses whether air pollution increases resting heart rates in 2,681 men and women aged 25–64 years who participated in the MONICA (monitoring of trends and determinants in cardiovascular disease) Augsburg cohort. Increases in heart rate were observed during the air pollution episode in January 1985 compared with non-episode days adjusted for cardiovascular risk factors and meteorologic parameters. Consistently, heart rates were also elevated at high concentrations of sulfur dioxide, total suspended particulates, or carbon monoxide. Acceleration in heart rates indicates an altered autonomic control of the heart in association with air pollution, which may contribute to the observed health effects in association with air pollution. *Am J Epidemiol* 1999;150:1094–8.

air pollution; cardiovascular diseases; heart rate; sulfur dioxide

Increases in total mortality have been consistently observed in association with concentrations of ambient air pollutants (1–3). The involvement of the cardiovascular system was strongly supported by recent analyses showing an association between particulate matter and hospital admissions for cardiovascular diseases (4–7).

In January 1985, an air pollution episode occurred throughout Central Europe (8), resulting in an elevated number of hospital admissions for cardiovascular diseases that were attributed to admissions for acute coronary syndromes and arrhythmia (9). Concurrently, the first MONICA survey (monitoring of trends and determinants in cardiovascular disease) was carried out in Augsburg (southern Germany) (10). This survey provides the unique opportunity to study the impact of an air pollution episode on markers of early biologic effects or altered function of the heart in a random sample of the adult population. Of particular interest are markers that could explain the increase in hospital admissions for cardiovascular diseases in association with air pollution (11, 12). An elevated resting heart rate has been recognized as a risk factor for all-cause mortality as well as cardiovascular mortality independent of other major risk factors (13–15). This paper assesses whether accelerations in heart rate, as a marker for altered autonomic control of the heart (16), can be observed in association with elevated levels of air pollution.

MATERIALS AND METHODS

The first MONICA survey in Augsburg (southern Germany) was carried out in 1984–1985. Taking part were 4,022 of the 5,069 randomly sampled eligible subjects, aged 25–64 years (response = 79.3 percent) (10). All survey methods were as in the MONICA protocol (17). A 12-lead resting electrocardiogram was derived from subjects in a supine position. The mean heart rate was determined from electrocardiogram records with a duration of 20 seconds using computerized electrocardiogram analysis systems that provided consistently operating measurement algorithms for all records analyzed. In 1987–1988, participants of the first MONICA survey were reexamined using the same methodology and protocols as 3 years earlier. A uniform age and sex distribution over time was preserved by design for both examinations. This report is based on a subsample of 2,681 men and women, from whom valid electrocardiogram readings were obtained each time and in whom no acute infection was present. There were no major differences in conventional cardiovascular risk factors between the subgroup of participants considered in this paper and the total sample (10, 18).

Sulfur dioxide (SO_2), carbon monoxide (CO), and total suspended particulates were measured as part of

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Abbreviation: MONICA, monitoring of trends and determinants in cardiovascular disease.

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the automated Bavarian air quality network (12). The monitoring station was located in the center of the city while temperature, relative humidity, and air pressure were measured on the outskirts of the city.

Linear regression models based on generalized estimating equations for clusters (19, 20) were used. Air pollution was considered in two ways in the analyses: 1) an indicator for the 1985 episode or 2) the continuous concentrations of SO_2, total suspended particulates, and CO. Categorical variables were constructed to control for age (10-year categories), body mass index (<25, 25–30, >30 kg/m^2), and current smoking. Systolic and diastolic blood pressure and total and high density lipoprotein cholesterol were entered into the models as continuous variables. Meteorologic parameters were considered as possible confounders in the regression analyses. Temperature was defined as below freezing, 0–10°C, and above 10°C; an indicator for relative humidity was one if the relative humidity exceeded 85 percent; and an indicator for high air pressure was one if the air pressure exceeded 1,020 mbar (765 mmHg or 102,000 Pa).

The analyses were conducted separately for men and women to allow different associations of the heart rate with the covariates (18). Summary estimates are presented to provide an estimate of the overall effect, taking into account the differences in risk profiles between men and women, and were calculated as the mean of the estimates for men and women weighed by the inverse of their variances.

RESULTS

Concentrations of SO_2 and total suspended particulates were substantially higher during the air pollution episode compared with those on all other days of the MONICA study (table 1). The episode was characterized by low temperatures, stable relative humidity, and easterly winds (12). No differences in CO concentrations were observed in comparisons of episode days with non-episode days. Total suspended particulates and SO_2 were moderately correlated ($r = 0.42$) during the winter of 1984–1985. CO was weakly correlated with SO_2 ($r = 0.28$) but not with total suspended particulates ($r = 0.05$). Three years later, the mean concentrations of SO_2 had more than halved, while the concentrations of total suspended particulates and CO remained unchanged. The winter of 1987–1988 was warmer than the winter of 1984–1985. The correlation between SO_2 and total suspended particulates was similar ($r = 0.45$), and both were correlated with CO ($r = 0.45$ and $r = 0.52$, respectively).

During the air pollution episode, higher heart rates were observed in men and women (table 2). Adjustment for cardiovascular risk factors reduced the effect estimates for the episode. The heart rate was associated with cardiovascular risk factors such as body mass index, serum cholesterol, blood pressure, and smoking. Women had higher heart rates than did men, and several cardiovascular risk factors had different impacts on the heart rates in men and women. Regular leisure time activity reduced the heart rates as reported earlier (21). An indicator for cardiovascular disease medication was associated with a reduced heart rate, but it did not confound the association between heart rate and the air pollution. Further adjustment for the meteorologic conditions attenuated the estimates that were not statistically significant themselves.

Estimates from analyses considering the air pollution concentrations as continuous variables are given in table 2. Regression coefficients are expressed for increases from the 5th to the 95th percentile of each pollutant, and direct comparisons between the estimates are possible. Similar effects were detected for concentrations of SO_2 and total suspended particulates on the same day as well as for levels of the air pollutants on previous days. Effect estimates for 5-day moving averages were comparable in magnitude. In analyses that excluded the 1985 air pollution episode, an increase of 70 μg of SO_2 per m^3 (5th to 95th percentile) was associated with an increase in heart rate of 1.75 beats per minute (95 percent confidence interval: 0.93, 2.57), and an increase of 75 μg of total suspended particulates per m^3 (5th to 95th percentile) was associated with an increase in heart rate of 1.12 beats per minute (95 percent confidence interval: 0.35, 1.90). The estimate for CO was exactly the same, with only the lower bound of the confidence interval just crossing zero.

Effects of comparable size and stability were observed for participants without a history of myocardial infarction, those who were not taking medication to treat cardiovascular diseases, and nonsmokers. Participants who lived within the city limits of Augsburg, in contrast, showed slightly larger effect estimates consistent with higher exposures in the urban environment.

DISCUSSION

During the 1985 air pollution episode, increases in heart rate determined by a resting electrocardiogram were present in a random sample. Even after adjusting for cardiovascular risk factors and meteorologic parameters, the increases in heart rate were apparent. Elevated heart rates were consistently observed in association with concentrations of SO_2, total suspended particulates, and CO. The effects of the episode seemed to be stronger in women than in men. However, no difference was observed when SO_2, total

TABLE 1. Air pollution,* meteorologic parameters,* and heart rates on the days of the MONICA† cohort study, Augsburg, Germany, 1984–1985 and 1987–1988

	Sulfur dioxide			Total suspended particulates			Carbon monoxide		
	No.	Mean (μg/m³)	Range (μg/m³)	No.	Mean (μg/m³)	Range (μg/m³)	No.	Mean (mg/m³)	Range (mg/m³)
Winter 1984–1985‡									
Outside the air pollution episode	116	48.1 (23.1)§	13 to 103	112	47.4 (28.7)	7 to 135	116	4.51 (0.18)	0.91 to 11.51
During the air pollution episode¶	10	200.3 (26.6)	160 to 238	11	97.7 (31.7)	62 to 176	10	4.54 (0.47)	2.39 to 6.85
Winter 1987–1988#	158	23.6 (12.2)	6 to 71	142	48.3 (22.1)	12 to 134	158	4.10 (1.25)	1.72 to 8.19

	Temperature			Heart rate					
				Men			Women		
	No.	Mean (°C)	Range (°C)	No.	Mean (beats/minute)	Range (beats/minute)	No.	Mean (beats/minute)	Range (beats/minute)
Winter 1984–1985									
Outside the air pollution episode	133	3.4 (5.9)	−18.0 to 14.5	1,235	66.0 (10.9)	40 to 111	1,180	66.9 (9.9)	42 to 115
During the air pollution episode	11	−15.5 (6.1)	−24.8 to −5.1	148	66.8 (10.5)	46 to 101	118	69.4 (9.9)	46 to 106
Winter 1987–1988	153	6.0 (6.5)	−11.3 to 18.7	1,383	64.9 (10.2)	40 to 107	1,298	65.8 (9.7)	42 to 108

* Twenty-four-hour averages (midnight to midnight).
† MONICA, monitoring of trends and determinants in cardiovascular disease.
‡ Examinations on 144 days between October 9, 1984, and May 24, 1985.
§ Numbers in parentheses, standard deviation.
¶ January 7–19, 1985.
# Examinations on 158 days between October 12, 1987, and June 24, 1988.

TABLE 2. Mean change in heart rates associated with air pollution in 1,383 men and 1,298 women from the MONICA* cohort, Augsburg, Germany, 1984–1985 and 1987–1988

Pollution variable	Men		Women		Men and women†	
	Heart rate‡	95% CI*	Heart rate	95% CI	Heart rate	95% CI
Crude analysis						
1985 episode§	1.75	0.43, 3.07	2.87	1.42, 4.32	2.26	1.28, 3.23
Analysis adjusted for cardiovascular risk factors ¶						
1985 episode§	1.81	0.48, 3.15	2.43	0.97, 3.88	2.09	1.11, 3.08
Analyses adjusted for cardiovascular risk factors and meteorologic parameters #						
1985 episode§	1.38	−0.08, 2.83	2.29	0.71, 3.88	1.79	0.72, 2.87
Same-day concentrations #						
Sulfur dioxide (per 80 μg/m³**)	1.02	0.41, 1.63	1.07	0.41, 1.73	1.04	0.60, 1.49
Total suspended particulates (per 90 μg/m³**)	1.61	0.38, 2.85	1.50	0.26, 2.73	1.56	0.68, 2.43
Carbon monoxide (per 6.6 mg/m³**)	0.95	−0.37, 2.27	0.98	−0.37, 2.34	0.97	0.02, 1.91
5-day-averages for the air pollutants #						
Sulfur dioxide (per 75 μg/m³**)	1.29	0.68, 1.90	1.26	0.57, 1.95	1.28	0.82, 1.74
Total suspended particulates (per 70 μg/m³**)	1.55	0.45, 2.65	2.08	0.84, 3.35	1.78	0.96, 2.60
Carbon monoxide (per 3.5 mg/m³**)	0.91	−0.25, 2.07	0.52	−0.55, 1.59	0.70	−0.09, 1.48

* MONICA, monitoring of trends and determinants in cardiovascular disease; CI, confidence interval.
† Adjusted for sex.
‡ Regression coefficient from linear regression analyses considering repeated measurements.
§ Compares the heart rate during the episode (January 7–19, 1985) with the heart rate measured on the second occasion within the same individual and with the heart rates measured on both occasions for persons measured on non-episode days at the first examination.
¶ Analyses adjusted for age, body mass index, high density lipoprotein cholesterol, total cholesterol, diastolic blood pressure, smoking, leisure time activity, and medication intake.
# Analyses adjusted for ¶ and temperature, relative humidity, and air pressure.
** Change in air pollutant concentration from the 5th to the 95th percentile.

suspended particulates, and CO were considered, which reduces the suspicion that an effect modification by sex might be present. SO_2 concentrations, unusually high for Augsburg, were recorded during the episode, but 3-hour mean concentrations never exceeded 600 μg/m³ at which a public alert would have been issued. Models for long range transport of air pollution suggest that SO_2 and respirable particulate matter had been transported to Augsburg in addition to pollution by local sources (8). Unfortunately, no data on the concentrations of fine and ultrafine particles were available, so that SO_2 and total suspended particulates serve as surrogates for the levels of inhalable particles. Even after exclusion of the air pollution episode, increases in heart rate were associated with elevated concentrations of the air pollutants.

Animal studies currently underway find increasing evidence for changes in the autonomic control of the heart and changes in hematologic parameters in animals exposed to concentrated ambient particles (22, 23) or after instillation of fly ash (24, 25). An elevated resting heart rate has been recognized as a risk factor for all-cause mortality as well as for cardiovascular mortality independent of other major risk factors (13–15). Data from the Framingham Study suggest that, during a longer follow-up among subjects with hypertension, an elevated heart rate is a strong predictor of death and fatal heart disease in both men and women (26). Elevated heart rates can serve as a marker for altered autonomic activity (16) and thereby identify patients at higher risk for sudden deaths during ischemic events (27). An increased risk for sudden death in association with elevated heart rates was observed in survivors of a myocardial infarction (28). Transient increases in heart rate before ischemic episodes have been noted in patients with unstable angina pectoris or non-Q-wave infarction undergoing Holter monitoring (29). A review of 72 sudden deaths occurring during Holter monitoring detected tachyarrhythmias preceding ventricular fibrillation in 90 percent of the cases (30).

The epidemiologic evidence presented in this paper points toward a modification of the autonomic control of the heart that might be in part responsible for the adverse health effects seen in association with air pollution episodes.

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