Effects of ambient air pollution on upper and lower respiratory symptoms and peak expiratory flow in children

H Marike Boezen, Saskia C van der Zee, Dirkje S Postma, Judith M Vonk, Jorrit Gerritsen, Gerard Hoek, Bert Brunekreef, Bert Rijcken, Jan P Schouten

Summary

Background Previous epidemiological studies have shown acute effects of increased amounts of ambient air pollution on the prevalence of respiratory symptoms in children with respiratory disorders. We investigated whether children with bronchial hyperresponsiveness (BHR) and relatively high serum concentrations of total IgE (>60 kU/L, the median value) are susceptible to air pollution.

Methods We collected data from children during three winters (1992–95) in rural and urban areas of the Netherlands. Lower respiratory symptoms (wheeze, attacks of wheezing, shortness of breath), upper respiratory symptoms (sore throat, runny or blocked nose), and peak expiratory flow were recorded daily for 3 months. The acute effects of airborne particulate matter with a diameter of less than 10 μm, black smoke, sulphur dioxide, and nitrogen dioxide were estimated by logistic regression.

Findings 459 (73%) of 632 children had complete data. Of these, 26% had BHR and relatively high (above median) serum total IgE, 36% had no BHR and total IgE of 60 kU/L or less, 15% had BHR and total IgE of 60 kU/L or less, and 23% had a total IgE of more than 60 kU/L but no BHR. In children with BHR and relatively high serum total IgE the prevalence of lower respiratory symptoms increased significantly by between 32% and 139% for each 100 $\mu m/m^3$ increase in particulate matter, and between 16% and 131% for each 40 $\mu m/m^3$ increase in black smoke, SO_2, or NO_2. Decrease in peak expiratory flow of more than 10% in that group was more common with increased airborne particulate matter and black smoke. There were no consistent positive or negative associations between increased air pollution and prevalence of respiratory symptoms or decrease in peak expiratory flow in the other three groups of children.

Interpretation Children with BHR and relatively high concentrations of serum total IgE are susceptible to air pollution. Although our odds ratios were rather low (range 1·16–2·39) the overall effect of air pollution on public health is likely to be substantial since these odds ratios refer to large numbers of people.

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Department of Epidemiology and Statistics, University of Groningen (H M Boezen PhD, J M Vonk MSc, B Rijcken MD, J P Schouten MSc); **Department of Environmental Sciences, Environmental and Occupational Health Units, University of Wageningen** (S C van der Zee MSc, G Hoek PhD, B Brunekreef PhD); **Department of Pulmonology, University Hospital Groningen** (D S Postma MD); **and Department of Pediatric Pulmonology, University Hospital Groningen** (J Gerritsen MD), **Netherlands**

Correspondence to: Dr H Marike Boezen, Department of Epidemiology and Statistics, University of Groningen, Ant Deusinglaan 1, 9713 AV Groningen, Netherlands (e-mail: h.m.boezen@med.rug.nl)

Introduction

Several epidemiological studies have described the adverse effects of ambient air pollution on respiratory health in children.[1–5] Most of these studies have focused on children with chronic respiratory symptoms. These children are judged to be the most vulnerable to the effects of air pollution. However, respiratory symptoms are an outcome of underlying processes. There is evidence that allergy plays an important part in the onset of respiratory symptoms in children. Therefore, allergy may be an important factor in assessment of whether a child will be susceptible to air pollution.

Bronchial hyperresponsiveness (BHR) is also associated with the development of respiratory symptoms in children. Population studies have shown a strong relation between raised serum concentrations of total IgE and the presence of BHR.[6–8] Children with BHR and raised serum concentration of total IgE may be vulnerable to the effects of air pollution, but they are not necessarily identified as such because they do not present themselves (yet) with chronic respiratory symptoms. We studied the effects of air pollution on lung function and the prevalence of lower and upper respiratory symptoms in children with BHR and relatively high serum concentrations of IgE, compared with children who did not have these characteristics.

Methods

Study population

We undertook a panel study of children aged 7–11 years living in rural areas (Bodegraven, Meppel, Nunspeet) and urban areas (Rotterdam, Amsterdam) of the Netherlands. The study took place over three consecutive winters (1992–95), and we used the PEACE study protocol.[9] The study was approved by the medical ethical committee of University Hospital, Groningen, and parents gave written consent.

Air-pollution measurements

Spirometry and methacholine challenge were done according to the protocol of the European Respiratory Health Survey.[10] Children who had a decline of 20% or more in forced expiratory volume in 1 s after inhalation of up to 2·0 mg/m^3 methacholine

	BHR+ high IgE (n=121)	No BHR and no high IgE (n=167)
Male/female	71/59	73/44
Mean (SD) age (years)	9·3 (1·0)*	9·8 (1·2)
Mean (SD) height (cm)	143 (9·00)*	145 (9·00)
Mean (SD) FEV_1 (L)	2·16 (0·36)*	2·32 (0·42)
Mean (SD) % predicted forced expiratory volume in 1 s	104 (11)*	107 (11)
Mean (SD) forced vital capacity (L)	2·51 (0·42)	2·56 (0·48)
Urban (%)	54 (45)	63 (38)
Mean (range) lower respiratory symptoms (%)	12 (0–35)	2 (0–38)
Mean (range) upper respiratory symptoms (%)	35 (0–64)	26 (5–71)
Mean (range) ⩾10% morning PEF decrease (%)	50 (18–88)	37 (13–76)
Mean (range) ⩾10% evening PEF decrease (%)	46 (0–82)	34 (13–65)

FEV_1=forced expiratory volume in 1 s. PEF=peak expiratory flow. *p<0·05.

Table 1: **Baseline characteristics of children with and without BHR and relatively high serum total IgE**

	Urban areas			Rural areas		
	n	Mean	Median (range)	n	Mean	Median (range)
Winter 1992–93						
Particulate matter ≤10 μm	72	54·8	51·7(4·7–145·6)	67	44·7	35·0 (4·8–103·8)
Black smoke	72	17·8	14·8 (2·5–55·9)	68	13·4	10·3 (1·2–37·6)
SO_2	73	22·5	21·6 (1·4–61·3)	72	9·8	9·0 (1·3–34·2)
NO_2	73	54·2	51·6 (21·8–94·0)	68	37·5	33·1 (5·9–83·2)
Winter 1993–94						
Particulate matter ≤10 μm	89	41·5	34·6 (12·1–112·7)	87	44·1	30·4 (7·9–242·2)
Black smoke	90	14·2	11·3 (0–47·8)	92	13·5	9·5 (1·5–58·1)
SO_2	91	11·8	10·2 (2·7–33·5)	92	8·2	4·4 (0·8–41·5)
NO_2	92	46·0	47·0(22·2–75·9)	92	26·6	24·0 (6·5–54·3)
Winter 1994–95						
Particulate matter ≤10 μm	82	31·1	28·9 (8·8–89·9)	81	26·6	23·7 (7·1–96·9)
Black smoke	74	8·8	6·7 (2·0–28·0)	81	8·7	5·6 (0–43·0)
SO_2	82	8·3	7·4 (0·6–24·4)	80	4·3	3·7 (0·5–17·0)
NO_2	82	46·7	48·3 (26·0–82·3)	80	23·7	21·4 (1·6–57·1)

Table 2: **24 h mean and median winter air pollution (μg/m³)**

were judged to have BHR. Serum total IgE was measured,[11] and measurements higher than the median value of serum total IgE (60kU/L) were defined as "relatively high".

Children were given a diary in which they recorded their lower respiratory symptoms, upper respiratory symptoms, and their morning and evening peak expiratory flow every day for 3 months. We included children who had data for at least 60% of those days in our analysis. The prevalence of lower respiratory symptoms (wheeze, attacks of wheezing with shortness of breath, shortness of breath) on a given day was defined as the number of children who reported such symptoms on that day divided by the number of children who provided diary data for that given day. The prevalence of upper respiratory symptoms (sore throat, runny or stuffed nose) was assessed in the same way.

The children made three daily measurements of their peak expiratory flow on a mini-Wright PEF meter (Clement Clarke International, London, UK) every morning on waking, and again in the evening at bedtime. We used the highest of each of the three daily measurements in our analysis. Children were excluded if they reported exactly the same peak expiratory flow for more than 1 week, since that would suggest cheating. For each child, the distributions of morning and evening peak expiratory flow were determined, and decreases of 10% or more below the 95th percentile of an individual child's morning or evening peak expiratory flow distribution were judged to be clinically relevant to our analysis.[12] The effects of air pollution on morning and evening peak expiratory flow were expressed as changes in the prevalence of morning or evening decrease in peak expiratory flow of 10% or more.[13]

Air pollution was measured continually at fixed sites in each urban and rural area. We measured atmospheric concentrations of particulate matter smaller than 10 μm, black smoke, sulphur dioxide (SO_2), and nitrogen dioxide (NO_2). Amounts of air pollution were defined as the mean 24 h concentration for the day on which effects on health were measured (lag 0), for the 2 days preceding that day (lag 1, lag 2), and by the 5-day mean concentration of lag 0 to lag 4 preceding that day.

Statistical analysis

We compared the acute effect of air pollution on the prevalence of respiratory symptoms, and on peak expiratory flow in four groups of children: those with both BHR and a relatively high (>60 kU/L) serum total IgE; those without BHR with relatively low (≤60 kU/L) serum total IgE; those with BHR but without relatively high serum total IgE; and those without BHR and with relatively high serum total IgE.

We did logistic regression using PROC MODEL (SAS version 6.12). Additional modelling to take into account the non-independence of residuals on successive days (autocorrelation) used the AR-macro with method equal to maximum likelihood. We adjusted the logistic model for daily minimum temperature, for linear, quadratic, and cubic time trend, and for weekends and holidays. We also adjusted for influenza incidence[14] for each area and each winter, which gave us six estimates of the effects of air pollution on symptoms and peak expiration flow. These estimates were weighted with the inverse of the square of their SE to give a weighted mean estimate. Odds ratios were calculated for

	Lower respiratory symptoms	Upper respiratory symptoms	≥10 % morning PEF decrease	≥10% evening PEF decrease
Particulate matter <10 μm				
Lag 0	1·32* (1·07–1·63)	1·13 (0·97–1·32)	1·10 (0·92–1·33)	1·37* (1·16–1·63)
Lag 1	1·36* (1·13–1·64)	1·00 (0·87–1·16)	1·08 (0·90–1·28)	1·09 (0·92–1·29)
Lag 2	1·36* (1·13–1·65)	0·96 (0·84–1·11)	1·03 (0·87–1·23)	1·16 (0·98–1·36)
5-day mean	2·39* (1·71–3·35)	0·91 (0·70–1·18)	1·10 (0·83–1·46)	1·35* (1·04–1·77)
Black smoke				
Lag 0	1·08 (0·85–1·38)	1·19* (1·01–1·40)	1·14 (0·92–1·40)	1·20 (0·99–1·45)
Lag 1	1·35* (1·11–1·64)	1·14 (0·99–1·32)	1·10 (0·92–1·32)	1·13 (0·95–1·35)
Lag 2	1·21* (1·00–1·47)	0·97 (0·84–1·11)	0·89 (0·74–1·06)	1·14 (0·97–1·33)
5-day mean	2·31* (1·66–3·23)	1·10 (0·85–1·43)	1·02 (0·76–1·36)	1·45* (1·11–1·90)
SO_2				
Lag 0	1·45* (1·13–1·85)	1·17 (0·99–1·38)	1·09 (0·89–1·34)	1·06 (0·86–1·30)
Lag 1	1·41* (1·09–1·82)	1·06 (0·90–1·25)	1·00 (0·81–1·23)	0·83 (0·68–1·02)
Lag 2	1·40• (1·10–1·79)	0·98 (0·84–1·15)	0·99 (0·81–1·21)	0·93 (0·76–1·13)
5-day mean	2·25* (1·42–3·55)	0·99 (0·71–1·39)	0·97 (0·69–1·37)	0·94 (0·67–1·33)
NO_2				
Lag 0	1·20* (1·03–1·39)	1·00 (0·90–1·10)	1·06 (0·95–1·20)	0·99 (0·88–1·12)
Lag 1	1·16* (1·01–1·33)	1·01 (0·92–1·11)	1·06 (0·95–1·18)	1·02 (0·91–1·13)
Lag 2	1·18* (1·03–1·35)	1·04 (0·95–1·14)	0·95 (0·85–1·06)	1·02 (0·92–1·14)
5-day mean	1·79• (1·39–2·30)	1·03 (0·84–1·27)	1·07 (0·88–1·30)	1·01 (0·83–1·22)

*p<0·05.

Table 3: **Estimated odds ratios (95% CI) for prevalence of respiratory symptoms and decrease in peak expiratory flow (PEF) with increase in air pollutants in children with BHR and relatively high serum total IgE (n=121)**

	Lower respiratory symptoms	Upper respiratory symptoms	⩾10% morning PEF decrease	⩾10% evening PEF decrease
Particulate matter <10 μm				
Lag 0	1·08 (0·75–1·57)	1·12 (0·99–1·28)	1·07 (0·93–1·23)	1·13 (0·98–1·30)
Lag 1	1·04 (0·70–1·53)	1·01 (0·89–1·15)	0·86* (0·75–0·99)	1·05 (0·91–1·21)
Lag 2	0·98 (0·69–1·39)	1·01 (0·89–1·15)	0·97 (0·85–1·11)	0·99 (0·87–1·14)
5-day mean	1·15 (0·61–2·15)	0·93 (0·67–1·28)	0·99 (0·79–1·23)	0·94 (0·75–1·17)
Black smoke				
Lag 0	1·30 (0·93–1·83)	1·04 (0·89–1·22)	0·97 (0·82–1·14)	1·19* (1·01–1·39)
Lag 1	0·79 (0·54–1·16)	1·03 (0·89–1·19)	1·02 (0·88–1·17)	1·06 (0·92–1·22)
Lag 2	0·93 (0·62–1·37)	0·95 (0·82–1·09)	0·99 (0·86–1·14)	1·08 (0·94–1·24)
5-day mean	1·16 (0·56–1·40)	1·01 (0·72–1·43)	1·06 (0·84–1·34)	0·95 (0·76–1·20)
SO_2				
Lag 0	1·12 (0·76–1·66)	1·01 (0·89–1·13)	1·02 (0·89–1·16)	1·10 (0·97–1·25)
Lag 1	0·61* (0·39–0·94)	1·08 (0·96–1·22)	1·00 (0·87–1·15)	1·06 (0·93–1·21)
Lag 2	1·11 (0·73–1·67)	1·01 (0·90–1·15)	0·96 (0·84–1·09)	0·94 (0·82–1·07)
5-day mean	0·68 (0·25–1·86)	0·99 (0·71–1·39)	0·92 (0·72–1·19)	0·88 (0·69–1·12)
NO_2				
Lag 0	1·13 (0·92–1·39)	1·03 (0·94–1·14)	1·06 (0·96–1·18)	1·16* (1·05–1·28)
Lag 1	0·90 (0·71–1·14)	0·92 (0·84–1·02)	0·99 (0·90–1·09)	1·03 (0·94–1·14)
Lag 2	0·98 (0·78–1·24)	1·08 (0·99–1·18)	1·02 (0·93–1·12)	1·00 (0·91–1·10)
5-day mean	0·86 (0·51–1·44)	1·01 (0·80–1·28)	1·10 (0·92–1·32)	0·97 (0·81–1·17)

p<0·05.

Table 4: **Estimated odds ratios (95% CI) for prevalence of respiratory symptoms and decrease in peak expiratory flow (PEF) with increase in air pollutants in children without BHR and with relatively low serum total IgE (n=167)**

100 μm/m³ increase in particulate matter and 40 μm/m³ increase in black smoke, SO_2, and NO_2. This variation in air pollution represents typical variation in normal winters in the study areas.

Results

632 children were included in the study. Data were missing for 173 children on BHR, serum total IgE, or both, but these children were similar to the group of 459 children with complete data in terms of sex ratio and percentage of predicted forced expiratory volume in 1 s. Of the 459 children with complete data, 121 (26%) had BHR and relatively high (above median) serum total IgE concentration, 167 (36%) had neither BHR nor relatively high serum total IgE concentration, 67 (15%) had BHR and relatively low (below median) serum total IgE concentration, and 104 (23%) had a relatively high serum total IgE concentration but no BHR (table 1).

Table 2 shows 24 h mean and median air-pollution concentrations in the study areas. Data on lower respiratory symptoms, upper respiratory symptoms, decreases of 10% or more in peak expiratory flow, and air pollutants for the 121 children with BHR and relatively high serum total IgE (table 3) show that the prevalence of lower respiratory symptoms increased significantly by between 32% and 139% for every 100 μg/m³ increase in particulate matter smaller than 10 μm (lag 0 through 5-day mean), and by between 16% and 31% for every 40 μg/m³ increases in black smoke, SO_2, or NO_2 (lag 0 through 5-day mean). We found no significant association between the prevalence of upper respiratory symptoms or morning and evening decreases in peak expiratory flow of 10% or more, and increased concentrations of the different air pollutants in that group, although the prevalence of upper respiratory symptoms tended to increase with raised air pollution on the same day (lag 0), and evening decrease in peak expiratory flow of 10% or more was associated with increased amounts of airborne particulate matter and black smoke.

In the group with no BHR and relatively low serum total IgE, we found no consistent positive or negative association between respiratory symptoms, decreased peak expiratory flow, and increased air pollution (lag 0 through 5-day mean, table 4). There was also no consistent

	Lower respiratory symptoms	Upper respiratory symptoms	⩾10% morning PEF decrease	⩾10% evening PEF decrease
Particulate matter <10 μm				
Lag 0	0·77 (0·48–1·24)	1·13 (0·92–1·40)	1·04 (0·78–1·38)	1·07 (0·82–1·41)
Lag 1	1·34 (0·94–1·93)	0·98 (0·79–1·22)	0·86 (0·66–1·12)	0·98 (0·76–1·26)
Lag 2	1·24 (0·86–1·81)	0·97 (0·79–1·20)	0·91 (0·71–1·17)	0·93 (0·73–1·19)
5-day mean	1·92 (0·84–4·41)	0·83 (0·54–1·25)	0·78 (0·51–1·20)	0·83 (0·55–1·26)
Black smoke				
Lag 0	1·00 (0·51–1·96)	0·95 (0·75–1·20)	0·82 (0·61–1·11)	1·04 (0·78–1·39)
Lag 1	1·39 (0·81–2·37)	0·91 (0·75–1·11)	0·96 (0·74–1·24)	0·99 (0·77–1·26)
Lag 2	1·10 (0·67–1·81)	0·94 (0·77–1·14)	0·88 (0·68–1·13)	0·94 (0·75–1·18)
5-day mean	0·97 (0·38–2·50)	0·86 (0·56–1·30)	0·73 (0·47–1·13)	0·78 (0·52–1·18)
SO_2				
Lag 0	0·72 (0·41–1·28)	0·82 (0·62–1·09)	0·74 (0·51–1·07)	1·23 (0·88–1·73)
Lag 1	1·03 (0·56–1·91)	0·84 (0·64–1·12)	0·96 (0·67–1·37)	1·32 (0·96–1·82)
Lag 2	0·96 (0·58–1·60)	1·12 (0·86–1·45)	1·18 (0·83–1·66)	1·14 (0·83–1·56)
5-day mean	1·83 (0·50–6·69)	0·72 (0·40–1·30)	0·79 (0·43–1·45)	1·21 (0·68–2·14)
NO_2				
Lag 0	0·72* (0·53–0·99)	0·92 (0·80–1·06)	0·79*(0·66–0·94)	0·93 (0·79–1·10)
Lag 1	0·97 (0·72–1·31)	0·95 (0·83–1·09)	0·89 (0·75–1·04)	0·91 (0·78–1·07)
Lag 2	0·99 (0·73–1·34)	1·03 (0·91–1·17)	0·94 (0·80–1·11)	0·95 (0·82–1·11)
5-day mean	1·18 (0·60–2·35)	0·98 (0·74–1·30)	0·71* (0·53–0·96)	0·75* (0·57–0·98)

p<0·05.

Table 5: **Estimated odds ratios (95% CI) for prevalence of respiratory symptoms and decrease in peak expiratory flow (PEF) with increase in air pollutants in children with BHR and relatively low serum total IgE (n=67)**

	Lower respiratory symptoms	Upper respiratory symptoms	≥10% morning PEF decrease	≥10% evening PEF decrease
Particulate matter <10 μm				
Lag 0	1·04 (0·80–1·35)	1·01 (0·85–1·20)	0·97 (0·80–1·17)	1·02 (0·85–1·22)
Lag 1	1·21 (0·98–1·51)	0·95 (0·81–1·12)	1·09 (0·91–1·30)	1·06 (0·90–1·25)
Lag 2	1·18 (0·96–1·45)	0·93 (0·80–1·09)	1·02 (0·85–1·21)	1·08 (0·93–1·27)
5-day mean	1·35 (0·89–2·04)	0·93 (0·69–1·25)	0·95 (0·71–1·28)	1·04 (0·80–1·34)
Black smoke				
Lag 0	1·06 (0·80–1·41)	1·04 (0·86–1·26)	0·85 (0·69–1·04)	0·96 (0·79–1·17)
Lag 1	1·28* (1·00–1·65)	1·01 (0·85–1·20)	1·00 (0·82–1·20)	1·04 (0·87–1·23)
Lag 2	1·07 (0·84–1·35)	0·92 (0·78–1·09)	0·99 (0·83–1·19)	0·98 (0·84–1·16)
5-day mean	1·49 (0·98–2·25)	0·99 (0·72–1·34)	0·77 (0·56–1·06)	0·99 (0·76–1·30)
SO_2				
Lag 0	1·44* (1·17–1·77)	0·98 (0·84–1·14)	0·92 (0·79–1·08)	1·00 (0·85–1·17)
Lag 1	1·28* (1·00–1·64)	1·01 (0·87–1·18)	1·03 (0·89–1·21)	1·05 (0·90–1·23)
Lag 2	1·38* (1·08–1·77)	0·89 (0·77–1·03)	0·99 (0·85–1·15)	1·16* (1·00–1·35)
5-day mean	2·49* (1·54–4·04)	0·83 (0·60–1·15)	0·88 (0·64–1·19)	1·24 (0·94–1·62)
NO_2				
Lag 0	1·04 (0·85–1·27)	1·09 (0·97–1·22)	0·98 (0·87–1·11)	1·00 (0·89–1·12)
Lag 1	0·85 (0·69–1·05)	0·99 (0·88–1·11)	1·00 (0·88–1·13)	0·92 (0·83–1·03)
Lag 2	0·99 (0·81–1·22)	0·90 (0·80–1·01)	1·04 (0·92–1·17)	0·98 (0·88–1·10)
5-day mean	0·83 (0·53–1·32)	1·03 (0·80–1·34)	0·92 (0·73–1·15)	0·96 (0·78–1·18)

p<0·05.

Table 6: **Estimated odds ratios for prevalence of respiratory symptoms and decrease in peak expiratory flow (PEF) with increase in air pollutants in children without BHR but with relatively high serum total IgE (n=104)**

association in the group with BHR but relatively low serum total IgE (table 5). In the group without BHR but with relatively high serum total IgE, the prevalence of lower respiratory symptoms increased significantly by between 28% and 149% for every 40 μg/m³ increase in SO_2 (lag 0 through 5-day mean), and by 28% per 40 μg/m³ increases in black smoke on the preceding day (lag 1, table 6).

Discussion

We have shown that children with BHR and relatively high concentrations of serum total IgE are susceptible to air pollution. This susceptibility is expressed as an acute and subacute effect (lag 0 through 5-day mean) of increased amounts of air pollution on the prevalence of lower respiratory symptoms. We also showed trends towards an acute and subacute effect of air pollution on the prevalence of evening decrease in peak expiratory flow of 10% or more, and acute effects (lag 0) of air pollution on the prevalence of upper respiratory symptoms.

Dockery and Pope[1] analysed the combined data of several epidemiological studies of children in the USA and Netherlands. They estimated a mean increase of 3% in the prevalence of lower respiratory symptoms with each 10 μg/m³ increase in daily mean concentrations of airbourne particulate matter, and a 0·7% increase in the prevalence of upper respiratory symptoms for each 10 μg/m³ increase in airborne particulate matter smaller than 10 μm. Although that combined study used different ranges of increase in particulate matter, and reported estimates on single lags, the effect of such particulate matter on the prevalence of lower and upper respiratory symptoms seems to be slightly greater in the children with BHR and relatively high serum total IgE concentration in our study. This finding might be explained by the fact that the airways of such children are more susceptible to irritants than those of children with chronic respiratory symptoms. Another explanation might be that that group of children had the same susceptibility to air pollution as children with chronic respiratory symptoms, but that our selection criteria based on the clinical characteristics of BHR and serum IgE concentration is less prone to error than selection criteria based on self-reported chronic respiratory symptoms used in other studies.

We studied the effect of several types of single air pollutants on respiratory health. This approach does not take into account that air pollution is in fact a mixture of air pollutants, which may interact and modify each others' effects on respiratory health. However, our research strategy is judged appropriate and effective for identification of at-risk groups and for public-health purposes.[15] Therefore, investigation of the effect of single air pollutants on respiratory health seems appropriate.

To our knowledge, our study is the first to compare the effects of air pollution in children with BHR and relatively high serum concentrations of total IgE with the effects of air pollution in a control group. The effects of air pollution on the group of children with BHR and relatively high serum total IgE were comparable with those previously described in children with chronic respiratory symptoms.[1,2] We conclude that with BHR and high serum concentrations of total IgE should be targeted by public health-improvement strategies. Medical characterisation of subgroups of study populations seems worthwhile in future research into air pollution. Although odds ratios were rather low (range 1·16–2·39), the effect of air pollution on public health is likely to be substantial since these odds ratios refer to large numbers of people.

Contributors

H Marike Boezen and Saskia C van der Zee coordinated population recruitment, medical characterisation, and diary measurements, and undertook data management. H Marike Boezen, Gerard Hoek, and Judith M Vonk contributed to data analysis. Dirkje S Postma, Jorrit Gerritsen, and Bert Rijcken contributed to study design. Bert Brunekreef was the principle investigator in a larger study from which we took our population sample. Jan P Schouten coordinated medical characterisations, and took part in study design and data analysis. All investigators contributed to writing the paper.

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Mortality after all major types of osteoporotic fracture in men and women: an observational study

Jacqueline R Center, Tuan V Nguyen, Diane Schneider, Philip N Sambrook, John A Eisman

Summary

Background Mortality increases after hip fractures in women and more so in men. Little is known, however, about mortality after other fractures. We investigated the mortality associated with all fracture types in elderly women and men.

Methods We did a 5-year prospective cohort study in the semi-urban city of Dubbo, Australia, of all residents aged 60 years and older (2413 women and 1898 men). Low-trauma osteoporotic fractures that occurred between 1989 and 1994, confirmed by radiography and personal interview, were classified as proximal femur, vertebral, and groupings of other major and minor fractures. We calculated standardised mortality rates from death certificates for people with fractures compared with the Dubbo population.

Findings 356 women and 137 men had low-trauma fractures. In women and men, mortality was increased in the first year after all major fractures. In women, age-standardised mortality ratios were 2·18 (95% CI 2·03–2·32) for proximal femur, 1·66 (1·51–1·80) for vertebral, 1·92 (1·70–2·14) for other major, and 0·75 (0·66–0·84) for minor fractures. In men, these ratios were 3·17 (2·90–3·44) for proximal femur, 2·38 (2·17–2·59) for vertebral, 2·22 (1·91–2·52) for other major, and 1·45 (1·25–1·65) for minor fractures. There were excess deaths (excluding minor fractures in women) in all age-groups.

Interpretation All major fractures were associated with increased mortality, especially in men. The loss of potential years of life in the younger age-group shows that preventative strategies for fracture should not focus on older patients at the expense of younger women and of men.

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Bone and Mineral Research Division, Garvan Institute of Medical Research, St Vincent's Hospital, Sydney, New South Wales 2010, Australia (J R Center MB, T V Nguyen PhD, Prof J A Eisman MB); **University of California, San Diego, La Jolla, California, USA** (D Schneider MD); **and University of Sydney, Royal North Shore Hospital, Sydney** (Prof P N Sambrook MB)

Correspondence to: Dr Jacqueline R Center (e-mail: j.center@garvan.unsw.edu.au)

Introduction

Although osteoporosis and related fractures are well-recognised public-health concerns, their impact on mortality remains unclear. Increased mortality after hip fracture is generally accepted, but there are few data on the outcomes of other fracture types. Excess mortality varies after hip fracture, with 12-month rates ranging from 12% to 35%.[1] The variation in these mortality estimates may relate to differences in demography, age of people studied, study size, and completeness and length of follow-up. Most excess mortality occurs within the first 3–12 months after fracture,[2–5] and increases with age.[5–11]

In women, low bone-mineral density has been associated with increased mortality independent of fracture.[12,13] At least some of the fracture-mortality association may, therefore, reflect the underlying health of the individual. Although comorbid health status has been associated with mortality after hip fracture in most studies,[3,5–7,14–19] this finding has not been universal.[20]

If osteoporosis is independently associated with mortality, increased mortality could be associated with other types of osteoporotic fractures. In one population-based study, women with vertebral fractures had increased 5-year mortality.[21] These were clinically diagnosed vertebral fractures that may represent only a third of all vertebral deformities. By contrast, the survival of women with forearm fractures,[21–23] foot, or ankle fractures[23] has been reported to be no different from that of the general community.