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Exposure-response functions for health effects of ambient air pollution applicable for China – a meta-analysis

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Abstract

Assessing the benefits of projects and policies to reduce air pollution requires quantitative knowledge about the relationship between exposure to air pollution and public health. This article proposes exposure-response functions for health effects of PM_{10} and SO_2 pollution in China. The functions are based on Chinese epidemiological studies, and cover mortality, hospital admissions, and chronic respiratory symptoms and diseases. We derive the following coefficients for acute effects: a 0.03% (S.E. 0.01) and a 0.04% (S.E. 0.01) increase in all-cause mortality per $\mu g/m^3$ PM_{10} and SO_2, respectively, a 0.04% (S.E. 0.01) increase in cardiovascular deaths per $\mu g/m^3$ for both PM_{10} and SO_2, and a 0.06% (S.E. 0.02) and a 0.10% (S.E. 0.02) increase in respiratory deaths per $\mu g/m^3$ PM_{10} and SO_2, respectively. For hospital admissions due to cardiovascular diseases the obtained coefficients are 0.07% (S.E. 0.02) and 0.19% (S.E. 0.03) for PM_{10} and SO_2, respectively, whereas the coefficients for hospital admissions due to respiratory diseases are 0.12% (S.E. 0.02) and 0.15% (S.E. 0.03) for PM_{10} and SO_2, respectively. Exposure-response functions for the impact of long-term PM_{10} levels on the prevalence of chronic respiratory symptoms and diseases are derived from the results of cross-sectional questionnaire surveys, and indicate a 0.31% (S.E. 0.01) increase per $\mu g/m^3$ in adults and 0.44% (S.E. 0.02) per $\mu g/m^3$ in children. With some exceptions, Chinese studies report somewhat lower exposure-response coefficients as compared to studies in Europe and USA.

Keywords: Exposure-response; Health effects; Air pollution; Meta-analysis; China; Epidemiology

1. Introduction

Air pollution and its impact on people's health and the environment is a matter of great concern in China. Heavy reliance on coal in power production and a rapidly growing car fleet, usually in combination with outdated technologies and poor maintenance, has led to a concentration of air pollutants far exceeding the limits of both national air quality standards and the air quality guidelines recommended by the World Health Organization (WHO, 2000). Indoor air pollution is a particular concern for a large number of people who depend on coal and biomass for cooking and heating (Wang and Smith, 1999).

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Quantifying the relationship between exposure to air pollution and the resulting effects on health makes it possible not only to analyze the cost-effectiveness of pollution abatement strategies and to set air quality standards, but also to assess the health benefits of energy conservation and other measures that typically reduce emissions of greenhouse gases (GHG). The costs of such measures in a country like China – where air pollution is ubiquitous – may be seriously overestimated if the benefits of any simultaneous reduction of air pollution are not taken into account (Wang and Smith, 1999; Cifuentes et al., 2001; Seip et al., 2001; Ho et al., 2002; Aunan et al., 2003; O'Connor et al., 2003).

Coherent associations between air pollution and various health end points have already been observed in a number of Western studies. See, for example, reviews by Dockery and Pope, 1994; Brunekreef et al., 1995; Pope et al., 1995a; EC, 1998; Brunekreef and Holgate, 2002; US-EPA, 1997, 2003. The findings include increased mortality, especially cardiovascular and respiratory mortality, increased incidence rate and duration of acute respiratory symptoms, exacerbation of asthma, decline in lung function, increased hospitalization (especially for respiratory and cardiovascular diseases), and increased prevalence of chronic diseases (e.g. chronic obstructive pulmonary disease (COPD) which includes diseases as chronic bronchitis and emphysema). Epidemiologic evidence also suggest that exposure to outdoor air pollution is associated with an increased rate of lung cancer (Katsouyanni and Pershagen, 1997; Cohen et al., 1997; Pope et al., 2002).

A large share of the epidemiological studies from the USA report that particles, or some exposure index related to particulate air pollution, have the greatest explanatory power in exposure-response functions for several health end-points, although other air pollutants may also be associated with the effects. The evidence of particles playing a central role is especially strong in studies of mortality, for which an association have been reported over a wide range of concentrations, and in a variety of communities with varying climates and mixtures of pollutants. European studies tend to report associations also for other air pollutants than particles, especially for SO_2 (e.g. Sunyer et al., 1996; Vigotti et al., 1996; Samoli et al., 2003) and for NO_2 (e.g. Pönkä, 1991), and in the USA, sulfate has been reported to be associated with mortality and morbidity (Lippmann and Thurston, 1996). Moreover, there is evidence of health impacts of O_3, mainly in the lower respiratory system, independent from simultaneous exposure to particles (US-EPA, 1996).

The composition of air pollution differs significantly between China and most Western states, however, and the exposure-response coefficients found in the Western studies cannot simply be transferred to a Chinese context. Moreover, demographic factors such as age-distribution and health status may influence the impact that air pollution has on public health. The aim of this article then is to perform a meta-analysis of Chinese epidemiological studies to derive a set of exposure-response functions for health effects of air pollution that can be used in evaluating air pollution measures in China. Previous attempts to estimate impacts on health of air pollution in China have applied a combination of Chinese and Western studies to derive exposure-response functions (e.g. Kan and Chen, 2003a; Aunan et al., 2004; Li et al., 2004; Kan et al., 2004). No systematic review of Chinese epidemiological studies has, however, been carried out to our knowledge.

We look specifically at the relationships between particulate matter (PM_{10}) and sulfur dioxide (SO_2) and mortality, hospital admissions, and chronic respiratory symptoms and diseases. We express the exposure-response functions in terms of percentage change (per unit of exposure) rather than as absolute numbers. The European ExternE program concluded that this type of estimate is more reliably transferable between locations, as it ensures that the calculated reduction in health damage from a reduction in the population exposure is a function also of the actual frequency before abatement takes place (EC, 1995). By taking a percentage change approach we suggest the functions may also be transferable to other developing countries characterized by similar air pollution conditions as China.

2. Materials and methods

Epidemiological studies of effects of PM_{10} and SO_2 in China published in English and Chinese were collected through a systematic literature search. Studies considered relevant were those in which significant ($P<0.1$) relative risk estimates for health outcomes were given for a reported change or difference in air pollution level. Subclinical effects, such as effects on lung function, were not included. The exposure-response coefficients were estimated by means of a simple meta-analysis technique (inverse variance method), in which the overall coefficient is a weighted average of the individual study coefficients (e.g. Schlesselman and Collins, 2003). The weights used in the calculation are the inverse of the study variance. We used the upper 95% confidence interval to calculate the standard error (S.E.) in case it was not given explicitly. The S.E. of a combined coefficient is the inverse of the square root of the sum of weights. Heterogeneity was assessed by means of the Q statistic, which is the sum over all studies of the study weight multiplied with the square of the difference between the study coefficient and the weighted average coefficient. The Q statistic is referred to a chi-square distribution with $n-1$ degrees of freedom, where n is the number of studies that are pooled.

The health effects of particles are usually related to the inhalable fraction, PM_{10}, and for some endpoints perhaps a fraction of even smaller particles (see, e.g. Maynard and Howard, 1999). We applied a PM_{10}/TSP-ratio of 0.6 to estimate coefficients for PM_{10} when the study reported TSP (particle measure in studies are indicated in Table 1). This is based on studies in various cities across China. A study in Shenyang reports a PM_{10}/TSP ratio of 0.7 (Xu et al., 2000); the World Bank (2001) reports a ratio of approximately 0.4 in three cities located in Hebei and Guangdong; and Wei et al. (1999) report a ratio of 0.65 in the cities Lanzhou, Wuhan, Chongqing, and Guangzhou. Conversion from TSP to PM_{10} adds to the uncertainty in the proposed coefficients, but no attempt was made to quantify this uncertainty.

In the functions proposed here, the estimated effect on health is attributed to one pollution component. This implies that we used results where the regression models behind the functions were fitted separately for the individual components or, with respect to the end-point chronic respiratory illness, we attribute the reported enhanced relative risk to the reported range of particulate pollution. This does not imply that there is no effect of other air pollution components, but simply that each one of the components is treated as an indicator of the health damaging agent(s) in the pollution mixture. Thus, when calculating the health benefit from a certain reduction in air pollution, one should not add the estimates that are obtained by applying the PM_{10} and SO_2 functions, respectively, because each of these estimates may represent the effect of the air pollution mixture as such. In case a certain reduction is obtained both for PM_{10} and SO_2, we suggest applying the function that gives the highest benefit, because in the opposite case the effect is likely to be understated. This is not a fully satisfactory procedure, but as long as independent functions in most cases are not available (and perhaps will never be due to synergistic effects between air pollutants) it is in our view justified. Concerning SO_2 we suggest not calculating health damage if the annual average is below the threshold of 50 $\mu g/m^3$, which is the WHO air quality guideline (WHO, 2000). For particles, there is according to WHO no evident threshold for effects on morbidity and mortality and we suggest to use the estimated natural background concentration as a lower impact threshold.

For long-term mortality and infant mortality, there were no Chinese studies available. In evaluating air pollution reductions the omission of long-term mortality and infant mortality altogether will result in a far greater inaccuracy than the inclusion of these endpoints based on studies from Western studies. Thus we suggest using estimates from Western countries for these endpoints. This will be described in more detail below.

3. Results

3.1. Mortality

We found six studies on daily mortality carried out in Chinese cities (Table 1), in Beijing (Xu et

Table 1
Results from epidemiological studies in China

End-point (pollutant)	Coefficient (%)	S.E.	Reference (particle measure in original study)
All-cause mortality			
PM_{10}	0.046	0.017	Jin et al., 1999 (TSP)
	0.028	0.009	Xu et al., 2000 (TSP)
	0.038	0.017	Cropper et al., 1997 (TSP)[a]
	0.030	0.010	Kan and Chen, 2003 (PM_{10})
SO_2	0.191	0.060	Xu et al., 1994
	0.161	0.059	Wong et al., 2001
	0.024	0.009	Xu et al., 2000
	0.039	0.024	Venners et al., 2003
	0.159	0.025	Kan and Chen, 2003
Mortality due to cardiovascular diseases			
PM_{10}	0.128	0.053	Jin et al., 1999 (TSP)
	0.040	0.015	Kan and Chen, 2003 (PM_{10})
	0.036	0.013	Xu et al., 2000 (TSP)
	0.072	0.055	Cropper et al., 1997 (TSP)[a]
SO_2	0.169	0.045	Kan and Chen, 2003
	0.182	0.041	Venners et al., 2003
	0.018	0.012	Xu et al., 2000
Mortality due to respiratory diseases			
PM_{10}	0.359	0.127	Jin et al., 1999 (TSP)
	0.052	0.143	Cropper et al., 1997 (TSP)
	0.094	0.053	Wong et al., 2001 (PM_{10})
	0.060	0.035	Kan and Chen, 2003 (PM_{10})
	0.302	0.199	Xu et al., 1994 (TSP)
	0.043	0.027	Xu et al., 2000 (TSP)
SO_2	0.104	0.048	Venners et al., 2003
	0.325	0.078	Kan and Chen, 2003
	0.736	0.744	Xu et al., 1994
	0.074	0.025	Xu et al., 2000
Hospital admissions for respiratory diseases			
PM_{10}	0.159	0.030	Wong et al., 1999 (PM_{10})
	0.100	0.025	Wong et al., 2002 (PM_{10})
SO_2	0.129	0.040	Wong et al., 1999
	0.178	0.040	Wong et al., 2002
Hospital admissions for cardiovascular diseases			
PM_{10}	0.060	0.025	Wong et al., 1999 (PM_{10})
	0.070	0.020	Wong et al., 2002 (PM_{10})
SO_2	0.159	0.050	Wong et al., 1999
	0.208	0.035	Wong et al., 2002
Chronic respiratory illness in adults			
PM_{10}	0.299	0.014	Zhang et al., 1999 (TSP)
	0.648	0.129	Xu and Wang, 1993 (TSP)
	0.461	0.050	Jin et al., 2000 (TSP)
	0.725	0.370	Xiao et al., 1990 (TSP)
Chronic respiratory illness in children			
PM_{10}	0.362	0.017	Qian et al., 2000 (TSP)
	0.969	0.045	Qian et al., 2004 (PM_{10})

Table 1 *(Continued)*

End-point (pollutant)	Coefficient (%)	S.E.	Reference (particle measure in original study)
	4.756	0.815	Yu et al., 2001 (PM_{10})
	0.447	0.054	Zhang et al., 2002 (PM_{10} and TSP)

For mortality and hospital admissions the coefficients refer to percentage change in number of cases per person per $\mu g/m^3$ change in daily ambient concentration. For chronic respiratory illness they refer to percentage change in prevalence rates per $\mu g/m^3$ change in long-term concentration. S.E.: Standard Error.

[a] Carried out in Delhi and thus not included in the meta-analysis. Shown here because TSP levels in Delhi are similar to, e.g. Beijing and use of coal is widespread.

al., 1994), Benxi (Jin et al., 1999), Shenyang (Xu et al., 2000), Hong Kong (Wong et al., 2001), Shanghai (Kan and Chen, 2003b), and Chongqing (Venners et al., 2003). All the studies were time-series studies applying Poisson regression, except Kan and Chen (2003b), which used a case-cross-over design and logistic regression and Jin et al. (1999), which used linear regression to estimate the odd ratios associated with increased TSP levels.

Table 2
Exposure-response coefficients resulting from meta-analyses

End point (pollutant)	Coefficient	S.E.	Q-stat.	d.f.
All-cause mortality				
PM_{10}	0.03	0.01	1.0	2
SO_2	0.04	0.01	36.4	4
Mortality due to cardiovascular diseases				
PM_{10}	0.04	0.01	2.8	2
SO_2	0.04	0.01	23.5	2
Mortality due to respiratory diseases				
PM_{10}	0.06	0.02	7.8	4
SO_2	0.10	0.02	10.0	3
Hospital admissions for cardiovascular diseases				
PM_{10}	0.07	0.02	0.1	1
SO_2	0.19	0.03	0.6	1
Hospital admissions for respiratory diseases				
PM_{10}	0.12	0.02	2.3	1
SO_2	0.15	0.03	0.8	1
Chronic respiratory illness in adults				
PM_{10}	0.31	0.01	18	3
Chronic respiratory illness in children				
PM_{10}	0.44	0.02	>100	3

For mortality and hospital admissions the coefficients refer to percentage change in number of cases per person per $\mu g/m^3$ change in daily ambient concentration. For chronic respiratory illness they refer to percentage change in prevalence rates per $\mu g/m^3$ change in long-term concentration. S.E.: Standard Error, d.f.: Degrees of freedom.

The Beijing study (Xu et al., 1994) reports a positive and significant association between all-cause mortality and SO_2, whereas the corresponding association for TSP is positive, but not significant and thus excluded in the meta-analysis. Log-linear functions were fitted to the data in the Beijing study and the coefficient of a linearized exposure-response function is 0.19% (S.E. 0.03) per $\mu g/m^3$ SO_2, when linearized over a range of 10–400 $\mu g/m^3$, which was the lower and upper 5% percentiles values of SO_2. A study in Delhi (Cropper et al., 1997), where TSP concentrations are at the same level as for instance in Beijing and the use of coal is widespread, report coefficients for all-cause mortality and cardiovascular mortality within the range of the Chinese studies (inclusion of these coefficients will not change the results in Table 2).

For PM_{10}, the pooled coefficient (0.03%, S.E. 0.01) (Table 2) is in the lower range of results from larger time-series studies in Europe and the USA. In the APHEA2 project (Air Pollution and Health – a European Approach, second phase), Katsouyanni et al. (2001) report an increase in daily mortality rates in the range 0.02–0.08% per $\mu g/m^3$ PM_{10} based on data from 29 cities. Heterogeneity in the effect parameters between cities was explained by effect modifiers, as for instance the level of NO_2 and climate. The overall point estimate of 0.06% per $\mu g/m^3$ PM_{10} is somewhat reduced in Katsouyanni et al. (2003), where the data were reanalyzed using adjusted parameters in the statistical software and alternative models to control seasonality and meteorologic variables (see also Dominici et al., 2002; Colburn and Johnson, 2003 for a description of why data were reanalyzed). The revised analysis indicates a coefficient of 0.04–0.06% per μg/m^3 PM_{10} depending on the model. Whereas several studies in the USA previously have indicated a higher coefficient, approximately 0.10% per μg/m^3 PM_{10} (see reviews, e.g. in EC, 1995; Aunan, 1996), a recent comprehensive study across 90 cities, the NMMAPS (National Morbidity, Mortality, and Air Pollution Study), when reanalyzed for the same reason as above, indicates a 0.02–0.03% increase in all-cause mortality per μg/m^3 PM_{10} (HEI, 2003). A somewhat higher coefficient was reported from the Harvard Six Cities project: approximately 0.07% per μg/m^3 PM_{10} in the reanalysis by Klemm and Mason (2003).

Regarding SO_2, the pooled coefficient (0.04%, S.E. 0.01) (Table 2) is in line with values reported in studies in European countries. Reanalysis of data in APHEA cities (Samoli et al., 2003) indicates a pooled estimate for all cities in the range 0.03–0.06% per μg/m^3 SO_2 depending on model specifications. Lower coefficients were estimated for Eastern European countries as compared to Western, 0.01%–0.04% and 0.03%–0.06%, respectively. The large studies in USA, as NMMAPS, report no evidence of an association between short-term SO_2 and daily mortality.

Steeper coefficients for mortality due to cardiovascular (CVD), respiratory (RD), and chronic obstructive pulmonary (COPD) diseases were reported in the Chinese studies. We pool the significant coefficients from the studies into two groups: CVD and RD (COPD being a subgroup of RD). The obtained estimates are a 0.04% (S.E. 0.01) change in CVD deaths per μg/m^3 for both PM_{10} and SO_2. For RD deaths the coefficients are 0.06% (S.E. 0.02) per μg/m^3 PM_{10} and 0.10% (S.E. 0.02) for SO_2 (Table 2).

Air pollution may have both long-term and short-term impacts, and solely using daily time-series studies to estimate mortality impacts of pollution reductions most likely results in severe underestimates (Künzli et al., 2001; Dominici et al., 2003). In recent years increasing attention has been paid to the long-term impact that air pollution may have on the mortality rates and life expectancy in a population that is continuously exposed to pollution. Four studies from the USA provide evidence of long-term effects of PM_{10} and/or $PM_{2.5}$: Dockery et al., 1993; Pope et al., 1995b, 2002; Abbey et al., 1999. The exposure-response coefficients for particles and mortality reported in these long-term cohort studies are 5–10 times higher than the ones found in the studies of short-term effect of pollution on daily mortality rates: 0.8% per μg/m^3 in the Six Cities Study (Dockery et al., 1993) and 0.4% in the American Cancer Society (ACS) study (Pope et al., 1995b) (assuming a $PM_{2.5}$/PM_{10}-ratio of 0.6). These coefficients were reproduced in a comprehensive reanalysis (HEI, 2000). Results from the ACS study with extended follow-up time indicated a coefficient of 0.2–0.3% per μg/m^3 PM_{10}, depending on period (Pope et al., 2002). Time-series studies in Europe and the USA have demonstrated how the exposure-response coefficient rises substantially when moving from daily to monthly patterns, consistent with results from these cohort studies (Schwartz, 2000; Dominici et al., 2003; Zanobetti et al., 2003).

In China, evidence of air pollution having long-term impacts on mortality rates is provided by a cross-sectional study in Beijing (Zhang et al., 2000), but no long-term cohort studies of mortality rates have been carried out. Given that there are no cohort studies available and the short-term effects that can be calculated from time-series studies most likely constitute only a limited share of the total effect, the question arises how to estimate the total effect of air pollution on mortality in China. Taking into account that short-term mortality studies tend to report lower coefficients in China than in most Western country studies, and that large differences in demographic and socio–economic characteristics between China and USA prevail, it may seem questionable to transfer results from the USA to Chinese conditions. In the absence of long-term cohort studies in high pollution areas, we suggest, however, that estimates from US studies may be used in applied studies in China. In doing so, it should be recognized that the results are likely to be on the high side of the probability distribution and should be interpreted with caution.

Both short-term and long-term impacts on mortality can be estimated in terms of excess premature deaths. For policy makers evaluating impacts

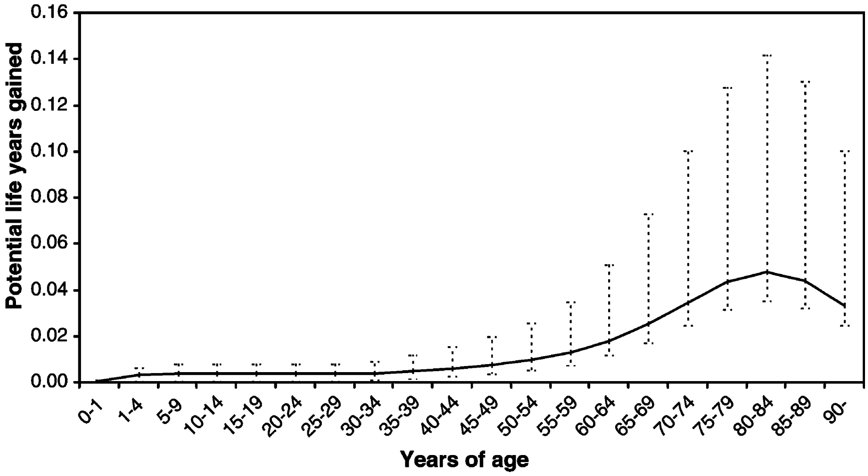

Fig. 1. Potential number of life years gained per person in one cohort at different age, estimated for a 10 $\mu g/m^3$ reduction in PM_{10} exposure (using the Chinese life table for 2003 and including an effect in infants). The integral under the curve, i.e. Δ life expectancy at birth, is 0.31 year. (Error bars refer to 95% Confidence Interval in exposure-response coefficients only.)

of air quality improvements, however, a more relevant metric may be one that addresses the question of how premature these excess deaths are and how life expectancy is affected in the population. The relationship between particulate air pollution and changes in life expectancy at birth and changes in life years lived have previously been estimated for specific populations from adjusting their 'life table' with exposure-response coefficients from long-term cohort studies (Brunekreef, 1997; Krewitt et al., 1999; Aunan et al., 2004). As an illustration, we apply the lower coefficient from Pope et al. (2002) (derived from the period when particulate air pollution was higher) – 0.24% (S.E. 0.12) – to a life table constructed using 2003 census data for the Chinese population (NBS, 2003). We assume that the death risk in people older than 30 years is affected by air pollution (in the Pope et al. studies enrolment was restricted to persons who were at least 30 years of age), and apply the coefficient uniformly to 5-year age groups between the ages of 30 and 90 in the life table. The estimated increase in life expectancy at birth from a 10 $\mu g/m^3$ reduction in the long-term level of PM_{10} is 0.26 year (see Aunan et al., 2004, and references therein for a description of methodology). The largest impact is found in the elderly due to their higher baseline mortality rates (see Fig. 1). Hence, a main effect seems to be that the elderly live longer and (probably) experience better health. For very large PM_{10} reductions, the estimated relationship (Fig. 2) gives unrealistically large increases in life expectancy and cannot be used.

Any impact on infant mortality markedly affects life expectancy, and even more if the baseline infant mortality rate is high, as it still is in many Chinese provinces. Six studies to our knowledge have reported an exposure-response function for particulate air pollution and all-cause infant mortality in different parts of the world (Bobak and Leon, 1992; Woodruff et al., 1997; Bobak and Leon, 1999; Loomis et al., 1999; Chay and Greenstone, 2003; Eun-Hee et al., 2003). The strongest association between infant mortality and air pollution is reported for mortality resulting from respiratory ailments. The pooled coefficient for all-cause infant mortality from these six studies (using the inverse variance method) is 0.39% (S.E. 0.04). When we apply this in the life table estimation, the change in life expectancy at birth from a 10 $\mu g/m^3$ reduction in the long-term PM_{10} level increases to 0.31 year. Although the air pollution levels were lower in the infant mortality studies

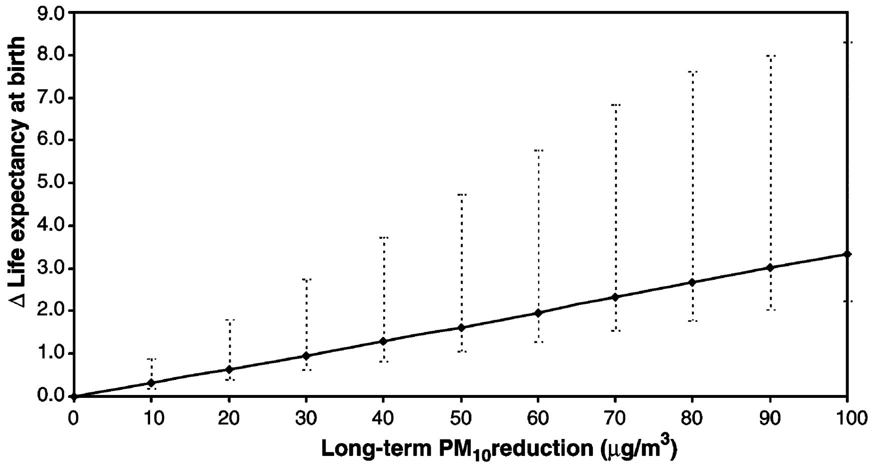

Fig. 2. Estimated increased life expectancy at birth (ΔLE) as a function of reduced long-term PM_{10} exposure (using the Chinese life table for 2003 and including an effect in infants). (Error bars refer to 95% Confidence Interval in exposure-response coefficients only.)

than in most Chinese cities at present, we would recommend application of the coefficient in China, until Chinese studies are available. Uncertainties related to transferring need, however, to be acknowledged.

3.2. Hospital admissions

We found three studies addressing the association between hospital admissions (HA) and air pollution in China, one in Chongqing (Zhou et al., 1997) and two in Hong Kong (Wong et al., 1999, 2002). We decided to exclude the first due to the methodology used and the low level of significance in the results (multiple linear regression analysis, with $\alpha = 0.15$). For an average temperature of 20 °C (T was an independent variable in the regression) the results from Chongqing indicate a 0.21% change in hospital admission due to chronic obstructive pulmonary disease (HA-COPD) per $\mu g/m^3$ SO_2 (in the range 50–150 $\mu g/m^3$). The coefficients in Table 2 are based on the two studies in Hong Kong, in which hospital admissions due to all respiratory (HA-RD) and all cardiovascular diseases (HA-CVD) were the end-points. The statistical approach (Poisson regression) in the two studies was reported to closely follow the ones adopted in APHEA1 and APHEA2 in Europe.

The PM_{10} coefficients obtained – 0.07% (S.E. 0.02) for HA-CVD and 0.12% (S.E. 0.02) for HA-RD – are more or less in line with estimates from Europe, whereas the SO_2 coefficients – 0.19% (S.E. 0.03) for HA-CVD and 0.15% (S.E. 0.03) for HA-RD – are higher. Pooled coefficients from APHEA2 studies in Europe indicate a 0.04–0.05% increase in HA-CVD for PM_{10} and approximately 0.07% for SO_2 (Le Tertre et al., 2002; Sunyer et al., 2003a). For admissions in people over 65, APHEA2 indicate a 0.09% increase in HA-RD for PM_{10} and 0.05% for SO_2 (Atkinson, 2001; Sunyer et al., 2003b), whereas Künzli (2000) in a meta-analysis of European studies obtain a coefficient of 0.13% per $\mu g/m^3$ PM_{10} for both HA-RD and HA-CVD. In USA results from NMMAPS indicate approximately 0.10% change in HA-CVD in people over 65 per $\mu g/m^3$ PM_{10} (Zanobetti and Schwartz, 2003), whereas excess risk estimates for HA-RH and PM_{10} are in the range 0.10–0.40% (US-EPA, 2003). Based on US studies from the early 1990s, WHO (1995) suggested an exposure-response function of a 0.20% (S.E. 0.09) change in HA-RD per $\mu g/m^3$ PM_{10}.

3.3. Chronic respiratory symptoms and diseases

We found eight studies addressing chronic respiratory symptoms and diseases in China, all of which were questionnaire surveys with a cross-sectional design. All applied multiple logistic regression to calculate odds ratios for the various symptoms and diseases, while controlling for a range of confounding factors and effect modifiers. Four of the studies investigate effects in adults, in Shenyang (Xiao et al., 1990), Beijing (Xu and Wang, 1993), Guangzhou, Wuhan and Lanzhou (Zhang et al., 1999) and Benxi (Jin et al., 2000), respectively. The other four investigate effects in children in Guangzhou, Wuhan and Lanzhou (Qian et al., 2000), Hong Kong (Yu et al., 2001), and Guangzhou, Wuhan, Chongqing and Lanzhou (Zhang et al., 2002; Qian et al., 2004). The studies by Jin et al. (2000) and Zhang et al. (2002) report odds ratios scaled to an explicitly given range of air pollutant. The others report odd ratios for clusters or areas for which the long-term average pollution level is given. For the latter, we used the significant odds ratios for prevalence rates of various chronic symptoms and diseases and the long term average levels of particulate air pollution to derive exposure-response coefficients to be included in the meta-analysis of what we denote chronic respiratory illness (CRI). The end-points ranged from milder symptoms as cough, phlegm and wheeze to more severe conditions as asthma, COPD in general and bronchitis in particular, and pneumonia. Pooling the coefficients for all end-points for adults we obtain 0.31% (S.E. 0.01) change in CRI per $\mu g/m^3$ PM_{10}. For children, the corresponding estimate is 0.44% (S.E. 0.02) (Table 2). These coefficients and standard errors apply whether we combine the individual coefficient within each study and subsequently combine the overall study coefficients, or we simply pool all coefficients derived from the different studies (25 and 29 coefficients for various CRI end-points in adults and children, respectively). Table 1 renders the study coefficients in combined form. For the specific end-point bronchitis, we estimated coefficients of 0.48% (S.E. 0.04) and 0.34% (S.E. 0.03) for adults and children, respectively. The heterogeneity in all estimates was high, reflected by very high Q-statistics and the arithmetic mean being rather different from the weighted mean [arithmetic means for all CRI was 0.53% (S.E. 0.10) in adults and 1.63% (S.E. 1.05) in children]. For CRI one reason for the heterogeneity may be that the coefficients included refer to varying end-points. However, there were no *systematic* differences in the magnitude of the coefficients for milder vs. more severe end-points. Moreover, the PM_{10} levels and gradient in Hong Kong are considerably lower than in the other cities, which may contribute to heterogeneity if the exposure-response relationship is non-linear (including estimates from Hong Kong did not change the estimated coefficient, however, due to low weights). Finally, the way the coefficients were derived may also have increased the uncertainty.

The coefficients for CRI and bronchitis estimated here for adults are considerably lower that what has been found in the USA. Two cross-sectional studies (Portnay and Mullahy, 1990; Schwartz, 1993) reported coefficients for chronic bronchitis of 1.09% and 1.27% per $\mu g/m^3$ PM_{10}. A longitudinal cohort study of new incidences of chronic bronchitis over a 10-year period by Abbey et al. (1993) indicated a 0.91% increase per $\mu g/m^3$ PM_{10} and thus confirmed the results of the generally assumed less reliable cross-sectional studies of prevalence rates. The coefficient for CRI in children is also considerably lower than reported from the USA, where the studies by Ware et al. (1986) and Dockery et al. (1989) both indicate approximately 2.5% increase in chronic bronchitis in children per $\mu g/m^3$ PM_{10}.

4. Discussion

Compared to studies in Europe and the USA, the Chinese epidemiological studies with some exceptions report lower coefficients in exposure-response functions for air pollution and health effects. European studies to some extent tend to report lower coefficients than studies in the USA. At least for particulate air pollution and mortality there is evidence from studies in Western countries to suggest the exposure-response relationship may become less steep as ambient concentration levels rise (Schwartz and Marcus, 1990; Lippmann and Ito, 1995; Samoli et al., 2001). Our observation that coefficients tend to be lower in China is consistent with this feature. Although a sigmoid shape of exposure-response functions is plausible, the lower coefficients reported in many Chinese studies may also result from other factors. A possible confounding with indoor air pollution is indicated in some of the Chinese studies. A misclassification of exposure will usually (but not necessarily) result in a downward bias of the observed association (Phillips and Smith, 1992).

In Western countries, studies typically report steeper exposure-response coefficients when cause-specific health end-points are addressed, of course given that the end-point concerned has a strong causal relationship with air pollution. This was also the case for the studies reviewed in this article. When it comes to the relative importance of particles vs. SO_2, Chinese and European studies tend to report associations between health effects and SO_2 to a larger extent than US studies. Our meta-analyses indicate, however, generally larger heterogeneity among SO_2 coefficients compared to PM_{10} coefficients, which can be interpreted as an indication of larger uncertainties in the SO_2 functions. The way S.E. of the combined coefficient is calculated does, however, not reflect this feature.

Heterogeneity in study coefficients was generally excessive for all end-points, and one may conclude that summary estimates are not justified. As long as studies were regarded relevant and valid, however, we deemed it not warranted to exclude any from the meta-analyses, even though some increase the heterogeneity more than others. The fact that few studies were available for each end-point and the different studies are carried out in different parts of the country and at different points of time implies that heterogeneity is expected. Assuming there is a fixed effect size of air pollution on health outcomes, which is not necessarily true, heterogeneity may imply that the studies have not been able to control adequately for confounders and effect modifiers. One may also question whether the standard errors of coefficients reported in the studies are representative for the uncertainties embedded. If a constant factor of error (percentage) is added to the standard errors reported in the different studies, the heterogeneity statistic becomes lower.

The epidemiological basis for establishing exposure-response functions is still thin in China, and the functions proposed in this article are based on only 16 studies altogether. Several more health end-points than those represented in the summary Table 2 are known to be associated with air pollution. Some of these may be important in a welfare perspective. For instance, we found no studies of the effect of daily ambient air pollution on the incidence of acute upper and lower respiratory symptoms. These symptoms may not be very serious for healthy parts of the population, but typically restrict activity and lead to work and school absenteeism. Probably more importantly, long-term cohort studies of impacts on mortality rates and studies of impacts in infant mortality have not been undertaken in China. We argue in this article that the absence of such studies in China justify transferring results from Western studies because omitting the end-points involved probably leads to the costs of air pollution being severely understated. One should, however, be careful to indicate the uncertainties that this approach entails. According to WHO (2000), extrapolation of health impacts slopes for particulate matter beyond 150 $\mu g/m^3$ PM_{10} must be done with extreme care due to the possible flattening of the curve.

Of course, transferring results from one part of China to another entails uncertainties in itself, for instance due to differences in effect modifying factors. Regarding the impact of air pollution on mortality rates, recent work has suggested that effects on health are not uniformly distributed. Factors such as education and antioxidant vitamin status may be important; thus disadvantaged population groups may be more susceptible (see Brunekreef and Holgate, 2002; O'Neill et al., 2003). A possible synergistic effect of air pollution and smoking (Xu, 1998) is of relevance in China, where smoking is common, particularly among men. Moreover, in China indoor air pollution resulting from the use of raw coal for cooking and heating poses large health risks to parts of the population. Generally, women and children are more prone to exposure to high levels of indoor air pollution, and thus suffer a disproportional share of the enhanced health risk (Pope and Xu, 1993; Smith, 1993; WHO, 2000; Zhang et al., 2001). More research into the likely distributional features of health damage due to outdoor and indoor air pollution in China is needed.

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