
CAN POPULATION CHARACTERISTICS ACCOUNT FOR THE VARIATION IN HEALTH IMPACTS OF AIR POLLUTION? A Meta-Analysis of PM₁₀-Mortality Studies.

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Abstract: In this paper a regression analysis is undertaken using the largest sample of air pollution mortality studies to date, from both developing and developed countries, in an attempt to further the understanding of the relationship between suspended particles and mortality. Applying Empirical Bayes meta-analysis, it is estimated that mortality rates on average increase by 6 per cent per 100- $\mu\text{g}/\text{m}^3$ increase in Particulate Matter (PM₁₀) concentrations, with greater effects in countries with high income inequality. We further find evidence that education and income have an influence on the effects of PM pollution.

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I. Introduction

As early as in 1952, during the air pollution disaster in London,¹ it was established that high levels of particulate-based smog could cause dramatic increases in daily mortality. The relationship between particulate matter and mortality has been analysed for some time now, and studies have reported evidence of increases in daily mortality also at much lower levels of particle concentrations. The variability among epidemiological findings, however, suggests that the connection between particulate matter and mortality is not well understood.

In this study we analyse the largest sample of short-term air pollution mortality studies to date, from the widest range of countries, in an attempt to further the understanding of the relationship between particles and mortality. In particular, our sample consists of time-series studies examining the effect of changes in daily (averaged) air pollution levels on daily mortality. The statistical relationship between particulate air pollution and mortality is addressed in epidemiologic studies, and the ensuing ‘dose-response functions’ tell us the impact on the mortality rate of a population of a certain dose of pollution.² Because the epidemiologic studies differ in a number of ways, the regression coefficient of the dose-response function is likely to vary both with the characteristics of the exposed population, other site-specific differences, as well as analytical decisions.

This study will focus on whether population characteristics can explain some of the differences in effect estimates, while through sample selection trying to minimise the potential for other underlying sources for differences.

The analysis involves isolating relevant *moderator* variables using meta-regression methods. A moderator variable is a variable that causes differences in the correlation between two other variables, in this case between mortality and ambient concentration of air pollution. If there is true variation in results across studies, then one or more moderator variables must exist that are able to account for the variance. The general underlying form is as follows:

$$b_j = \mathbf{b} + \sum \mathbf{a}_k Z_{jk} + u_j \quad (j = 1, 2 \dots L) \quad (k = 1, 2 \dots M) \quad (1)$$

here b_j is the reported dose-response estimate in the j th study from a total of L studies, \mathbf{b} is the summary value of b , Z_{jk} are the variables that could explain variations amongst the

¹ The London smog disaster (December 1952) established that high levels of air-borne particles and sulphur dioxide produced large increases in daily death rates (HMSO, 1954).

² For a review of the main study designs associated with epidemiologic studies refer to B. M. M. Gaarder (2002), chapter 5.

studies, \mathbf{a}_k are the coefficients of the M study characteristics that are controlled for, and u_j is the error term.³

The differences in results from individual studies imply that the quite commonly used procedure of transferring the regression coefficient unchanged to another population may lead to incorrect estimates of adverse health effects and the related costs. However, direct studies of the population in question may often not be feasible due to the quality of data, or to time and financial constraints. With the growing body of dose-response studies increasingly carried out also outside of the US, a second-best option is emerging. Rather than transferring the dose-response *coefficients* unaltered from one population to another, the existing studies can be used to estimate the coefficients on relevant moderator variables, and these in turn may enable us to transfer dose-response *functions*. We are then in the position to tailor-make the coefficient for local conditions.

The meta-analysis may hence serve three main purposes: it can increase our understanding of what affects the amount of deaths that are related to air pollution; it will help highlight areas where further studies may be needed; and finally, through the ensuing coefficients of the moderator variables, it may help transferring the dose-response coefficients to countries where empirical studies have not yet been feasible or to forecast the effects of policies targeting air pollution.

Section 2 introduces the concept of meta-analysis, as well as the various uses, strengths, and weaknesses of this type of analysis. In section 3 we then move on to presenting the moderator variables selected. Next, section 4 describes the criteria used in composing our sample of past studies, the data used to capture the moderator variables, as well as the model and estimation procedure. The main results are presented in section 5, together with a sensitivity analysis and a discussion of the findings. Main results and implications are summarised in chapter 6.

II. A Survey of Existing Meta-Analyses of the Mortality from Air Pollution

Meta-analysis involves the synthesising of previous empirical analyses. Before presenting a survey of what has been done in this field, a brief introduction to the original studies and study-designs upon which these meta-analyses are based is therefore required.

The majority of the original studies use time-series data to examine the effect of short term responses in mortality to changes in air pollution levels. The main advantage of time series studies over cross-sectional studies is that socio-economic and demographic characteristics of the population are unlikely to change and do therefore not require explicit

³ Button and Nijkamp (1997).

modelling. The studies usually assume that the daily death counts (Y_i) are Poisson-distributed⁴ with:

$$\log (E(Y_i)) = X_i \mathbf{b}$$

where X_i is the vector of covariates on day i , \mathbf{b} is the vector of regression coefficients, and E denotes expected value. The unit of analysis in these studies is the day, and hence the potential confounders that must be controlled for are those that vary over time, possibly in coincidence with air pollution. Based on this logic, the vector of explanatory variables typically contains terms corresponding to a measure of air particulate, as well as meteorological covariates (e.g. ambient temperature and relative humidity), long-term and seasonal trend components, disease epidemics (e.g. influenza episodes), and day of the week and holidays.

It is important to point out that the dose-response function technique, as presented above, is mechanistic, incorporating no model of how individuals behave. The dose-response coefficient relies on the socio-economic and demographic characteristics remaining unchanged. Although it can quite accurately describe the effect of a change in air pollution on mortality in a certain population, demographically different groups and groups subject to different economic constraints may respond differently to exposure to air pollution. This is why, when we compare results from studies carried out at different sites, we need to take such differences into account. That is the role of the moderator variables in the meta-analyses.

Early meta-analyses were mainly concerned with finding the average effect across studies, implicitly assuming that the estimated effect in each study is an estimate of an effect size common for the whole population of studies. More recently, meta-analytic work has started to focus upon discovering and explaining the variations in effect sizes (Raudenbush and Bryk (1985)).

Button and Nijkamp (1997) discuss a number of issue areas within environmental policy evaluation which could benefit from the use of the meta-analysis techniques. In evaluating environmental costs, the meta-analysis can be used to look for indicators of central tendency in previous case studies or, alternatively, to explain why the studies

⁴ Only a small portion of a population dies on any given day. The number that die is a count; i.e. it can only take on values limited to the non-negative integers. This suggests that a Poisson process is the underlying mechanism modelled, since in a Poisson process a homogeneous risk to the underlying population is assumed. Given that underlying risk, the probability of Y deaths occurring on a given day is given by:

$$\text{prob}(y/\lambda) = \frac{e^{-\lambda} \lambda^y}{y!}$$

where λ is the expected number of deaths on any day (i.e. $E(Y)$) (Schwartz et al. (1996b)).

generate differing results. Furthermore, meta-analysis can be used in connection with the assessment of the effectiveness of alternative policy instruments in containing environmental damage, the assessment of political acceptability of alternative environmental instruments by decision makers, exploration of the appropriate political level of intervention to contain environmental damage, and finally in forecasting the effects of environmental policies.

Rosenthal (1991) distinguished three purposes of meta-analyses. First, to summarise for a set of studies what the overall relationship is between two variables investigated in each study. Second, to look at the factors associated with variations in the nature of relationships between two variables over a range of studies. Finally, to look at the aggregate data for each study and correlate this with other characteristics of the study (Bergh et al. (1997)).

There is a wide range of problems involved in employing meta-analysis in economic research. Broadly, we can divide the problems into two categories. The first category has to do with the objectivity with which the information is collected and reported, whereas the second deals with comparability between studies and how well the studies are designed for the particular question they want to address. There is a possible bias resulting from the nature of the studies that are included or excluded. First, the researchers use various inclusion-selection rules for the analysis (e.g. including only published studies) which are inherently subjective. Second, there is the tendency to publish only positive results. As for comparability, a number of challenges exist. Studies often use diverse units of output measures and, furthermore, diverse methods of obtaining these outputs (e.g. diverse regression methods, different sets of control variables). A degree of subjectivity is introduced into many of these studies and thereby into the meta-analysis because the reported results were based on what, in the authors' opinions were the best coefficient estimates obtained. In particular, some studies reported coefficients obtained using same day level of pollution, others used one-day lags, and others again used moving averages of different lengths.

Estimates can differ partly due to the fact that the studies use different samples of the total population and partly due to the differing conditions under which the research takes place. Fixed effects models assume the existence of a common effect size in all the studies, whereas random effects models assume a different real effect in each study. In the latter case, combining effect sizes from empirical studies means assessing the average size of the real effect.

If we reject the hypothesis of equal real effect sizes, the next question is then whether we can find moderator variables that explain the variations between the empirically estimated effect sizes. If a linear combination of variables exists that completely explains the variations in the real effect sizes, then the effect size is fixed and not random (although the real effect sizes are different in each study). This is, however, a rare case. In most cases it is more realistic to use a model that takes account of the imperfections of the explanatory model.

Before we review the literature of meta-analyses on studies of mortality from particulate matter it is useful to understand the various models and assumptions underlying the different approaches.

Let us assume that the estimated effect size d_i of study i is equivalent to a true effect size \mathbf{d}_i plus an error of estimate e_i , where the errors are assumed to be independent and normally distributed with a variance v_i :

$$d_i = \mathbf{d}_i + e_i, \quad i = 1, \dots, k, \quad e_i \sim N(0, v_i) \quad (2)$$

The random effects model assumes that the effect size parameters \mathbf{d}_i can be decomposed into a mean population effect \mathbf{q} and a between-study variability term u_i , where the errors are assumed to be independent and normally distributed:

$$\mathbf{d}_i = \mathbf{q} + u_i, \quad i = 1, \dots, k, \quad u_i \sim N(0, \mathbf{t}^2) \quad (3)$$

The mixed effects model (which is equivalent to a random effects model incorporating study characteristics) assumes that the effect size parameter is a function of known study characteristics and random error:

$$\mathbf{d}_i = W_i' \mathbf{g} + u_i, \quad i = 1, \dots, k, \quad u_i \sim N(0, \mathbf{t}^2) \quad (4)$$

The fixed effect model implicitly assumes no between-study variability in either of the equations above, i.e. $u_i = 0$.

Combining equations (3) and (1) we obtain:

$$d_i = W_i' \mathbf{g} + u_i + e_i \quad (5)$$

Therefore, assuming that the error terms are independent, we can express the marginal distribution of d_i from the mixed effect model as:

$$d_i \sim N(W_i' \mathbf{g}, v_i + \mathbf{t}^2) \quad (6a)$$

The distribution of d_i from the random effect model, the simple fixed effect model, and the fixed effect model with study characteristics can be expressed respectively as:

$$(6b) d_i \sim N(\mathbf{q}, v_i + \mathbf{t}^2), \quad (6c) d_i \sim N(\mathbf{q}, v_i), \quad \text{and} \quad (6d) d_i \sim N(W_i' \mathbf{g}, v_i)$$

Based on the epidemiological literature dealing with the relationship between air pollution and mortality, to our knowledge seven meta-analyses have been carried out: Ostro (1993), Schwartz (1994), Lipfert and Wyzga (1995), Environmental Protection Agency (1996), Levy et al. (2000), Institute for Environmental Studies (2000), and Maddison and Gaarder (2001).⁵

After converting the results of the different studies into a common metric, Ostro's meta-analysis derived the unweighted average of central estimates and found that the mean effect of a 10 $\mu\text{g}/\text{m}^3$ (micrograms per cubic meter) change in PM_{10} on the percentage change in mortality varied between 0.64 and 1.49 per cent. Lipfert and Wyzga, on the other hand, calculated the variance weighted average of air-pollution-mortality elasticities and found that the mean overall elasticity as obtained from time-series studies for mortality with respect to various air pollutants entered jointly was approximately 0.048 (0.01 – 0.12). The elasticity obtained for population-based cross-sectional studies was of similar magnitude. The models used in both of these meta-analyses implicitly assume that each coefficient estimate, \mathbf{b} , is a random sample from a single underlying distribution with a distribution as in expression (6c). Ostro's study in addition implicitly assumes equal estimation errors in all of the studies.

Joel Schwartz (1994c) carried out a meta-analysis on a set consisting of studies from the US, London, and Athens. The main aim of the analysis was to compare the results found in different studies to the levels of potential confounders and the correlation between particulate matter and potential confounders in the individual studies to assess the likelihood that the results are driven by inadequate control for those factors. It then combines the studies in a meta-analysis and computes the average percentage increase in mortality per unit of pollution. Three approaches to calculating this average were used; unweighted, variance weighted, and quality weighted. The latter weights were based on the possibility in each study that the true effect sizes vary at least in part as a function of multiple identifiable sources, or confounding variables, and that if these have not been taken properly into account in the regression model used in a particular study the random error term will be larger for these studies. The central concern in the study was of confounding by some other pollutant, by weather and season, and an additional concern was the quality of the exposure assessment. Studies were given a higher weight the more they controlled for confounding factors (the highest weight was 4 and the lowest 2). The unweighted meta-analysis, as well as the analyses using the various weighting options all

⁵ For a review of how the present study relates to the Maddison and Gaarder study, please refer to appendix A.

gave a relative risk of 1.06 for a 100 $\mu\text{g}/\text{m}^3$ increase in total suspended particulate mass, which implies that the relationship is highly unlikely to be due to confounding factors.⁶ By introducing the quality weights, Schwartz is allowing for the idea that there is no single common underlying effect size. However, the size of the weights was provided by the researcher based on his subjective opinion of the quality of control for confounding. This subjective weighting may influence the results and is a weakness of the above meta-analysis.

In its meta-analysis the U.S. Environmental Protection Agency (EPA (1996)) criteria document used a random effects model to estimate PM mortality, where the distribution of the effect parameter is assumed to be given by expression (6b). The relative risk for mortality from PM_{10} exposure averaged over 2 days or less was in this study estimated as 1.031 per 50 $\mu\text{g}/\text{m}^3$ PM_{10} (CI: 1.025 – 1.038), whereas for a longer averaging time of between 3 and 5 days the relative risk was estimated as 1.064 (CI: 1.047 – 1.082). When potential confounding pollutants were included in the model the relative risk estimate decreased (1.018, CI: 1.007 – 1.029). Although the random effects model can quantify the amount of residual variance that can be explained by study characteristics, it does not attempt to identify what these characteristics are or how they influence the effect estimates.

In the most recent meta-analysis carried out by the Institute for Environmental Studies (IVM) (2000), the purpose was to obtain a single pooled estimate of the health effects reported from the selected studies in order to use this for evaluating the benefits gained from improving air quality in Mexico City. A weighted average was computed, giving more emphasis to studies with lower error in estimating their regression coefficient, as well as studies carried out in Mexico city ('articles with estimates based on Mexico City were given double the weight of international cases, because they are more likely to reflect the socio-demographic and susceptibility characteristics of the Mexico City population' (p.27, IVM (2000))). The pooled estimate of the effect of PM_{10} on total mortality was 0.79 per cent change per 10 $\mu\text{g}/\text{m}^3$ daily average PM_{10} (CI: 0.06 – 1.68). There is a certain inconsistency/contradiction in the method they have used. By weighting the estimates according to the inverse of their variance, the study is assuming that the variability in reported effects is attributable solely to sampling error. On the other hand, giving higher weights to the studies carried out in Mexico City implies that the

⁶ The information concerning the effect from exposure to air pollution on the risk of mortality uncovered by regression analysis can be expressed in a number of alternative ways. The findings are often expressed in terms of relative risk. The relative risk indicates the ratio of the probability of occurrence of a given effect between

authors assume that these studies are capturing some local characteristics and are hence more relevant for the purpose of policy-evaluation in Mexico City. This is a rather indirect way of controlling for confounding factors and may weaken the reliability of the pooled estimates. Furthermore, as was the objection to Schwartz' study, the size of the weights was provided by the researcher on a rather ad hoc basis and may influence the results.

Rather than providing pooled effect estimates, the meta-analysis by Levy et al. (2000) addresses between-study variability potentially associated with analytical models, pollution patterns, and exposed populations. They use the mixed effects Empirical Bayes (EB) model derived by Raudenbush and Bryk (1985), assuming that variability is due partly to sampling errors (or intra-study variability) and partly to between-study variability. This method is used in the present study as well, and the details of the method are set out in section 4.3. With a sample of 29 observations, 19 from the United States and 10 from outside of the United States, they investigate whether the ratio of $PM_{2.5}$ to PM_{10} , other pollutants, climate, season, prevalence of gas stoves and/or central air conditioning, percentage of elderly, percentage in poverty, and the rate of mortality can explain some of the differences in effect estimates. When analysing the 19 PM studies from the U.S. for which more confounding variables were available, the mortality rate was estimated to increase by 0.7 per cent per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations, with greater effects at sites with higher $PM_{2.5}/PM_{10}$ ratios, supporting the hypothesised role of fine particles. When all of the 29 studies were included, but only a subset of the predictors were available (PM_{10} concentration, averaging time and lag time, percentage of the population older than 65 years of age, baseline mortality rate, heating and cooling degree days, and dummy variables for PM_{10}/TSP and U.S./non-U.S. studies) only baseline mortality rate was significant. The grand mean estimate was about the same as for the 19-studies sample.

Finally, the meta-analysis by Maddison and Gaarder (2001) investigates whether, in a sample of 13 European and developing country studies, some of the between-study variability can be associated with pollution levels, the percentage of the population over 65 years of age, average income level, and the level of income inequality at a certain average income level. By weighting the effect estimates according to their estimated variances, we implicitly assumed a fixed effect model with study characteristics for which the distribution was given in expression (6d). The study found that the effect estimates were

significantly affected by the percentage of the population over 65 years of age, as well as income distribution. Based on the data used in our study, a model without predictors (i.e. fixed effect model) gives an estimate of the effect of PM₁₀ on total mortality of 0.3 per cent change per 10 µg/m³. An implicit assumption of our analysis, which seems unlikely and therefore weakens the results of this study, is that all the variance among the study effects other than sampling variance can be explained as a function of the study characteristics we chose to include.

In addition to the meta-analyses discussed above, a number of review articles have relied on qualitative discussions of the credibility of the evidence related to potential confounding factors (e.g. climate, correlated pollutants). Some of the authors of these studies conclude that a causal relationship clearly exists (Brunekreef et al. (1995), Pope et al. (1995a), Pope et al. (1995b), Thurston (1996)), whereas others (Gamble and Lewis (1996), Moolgavkar and Luebeck (1996)) argue that the relationship is spurious. The lack of quantitative base, however, makes these review studies more vulnerable to the set of studies chosen and the points the authors wish to argue. (Levy et al. (2000)).

III. The Moderator Variables

As mentioned in section 2, the original studies do not explicitly model the demographic and socio-economic characteristics of the population studied. The reason for this is that for a factor to confound the relationship between pollution and daily mortality it must be correlated with both pollution and mortality. Therefore, characteristics such as baseline health, age, and income cannot induce an association between today's mortality count and yesterday's air pollution, since they are not correlated with air pollution and do not vary on a daily basis.⁷ For cross-sectional mortality studies, on the other hand, personal characteristics and habits are important potential confounders, whereas short-term weather changes are not (Schwartz (1994c)). In our meta-analysis the aim is to combine time-series studies cross-sectionally, and to explain the variation in the dose-response coefficients using moderator variables (also known as effect modifiers). These moderators will hence need to address cross-sectional differences, rather than factors changing over time.

When deciding on which study-characteristics to include as potential predictors or moderator variables three factors guided the selection; theoretical plausibility, availability of characteristic-data, and novelty. This led to the following moderator variables; mean

⁷ The most important confounders for the relationship between air pollution and daily mortality are weather and infectious disease epidemics, according to Schwartz (1994c).

particle levels, amount of elderly people in the population, income level, income distribution, education, baseline health, and health services.⁸ The reasons why we believe these factors (or characteristics) to be potential moderators are discussed below. Other study characteristics, such as the lag and averaging times, the levels of other pollutants, the ratio of fine particles to overall particle concentration, and the type of mortality considered, although potentially interesting predictors, were either not considered due to lack of information in many of the studies or were investigated through sensitivity analysis.

Most dose-response analyses have implicitly assumed a log-linear relationship between the mortality count and pollution, however, it has been argued that this may not be accurate. As the exact shape of the relationship is not yet known, we argue that it may be interesting to include pollution as a moderator variable. By regressing the estimated pollution-mortality association on pollution (i.e. second order partial derivative), we pick up any non-linearities in the relationship.

A variety of advanced disease states, as well as generally lower baseline health levels, may predispose individuals to heightened susceptibility to premature death due to exposure to air pollution. This implies that the death rate due to a certain amount of particle exposure may increase more among elderly and individuals with lower baseline health as compared to the younger and those with better health, and that death rates due to respiratory and cardiovascular failure increase more than the total rate. However, as exposure tends to be approximated by air pollution concentration measurements from central monitoring stations, it is possible that the individual exposure for a certain amount of pollution concentration also varies with baseline health levels and age (i.e. the optimal amount of averting activities may be affected by age and health level). Furthermore, the heightened susceptibility to exposure may influence the amount of mitigating activities chosen by elderly individuals and individuals with low levels of baseline health. In other words, both age and baseline health levels may well influence the amount of mitigating and averting activities undertaken, and hence affect health indirectly. On the one hand, it is possible that an individual with low health levels will be more inclined towards trying to prevent further adverse health effects (both due to personal experience with bad health and due to decreasing utility at an increasing rate). On the other hand, the individual may be used to being in bad health and expect to live for a shorter time, and therefore less inclined to invest in health. It is theoretically not clear what net effect baseline health and

⁸ The first four were also used in the Maddison and Gaarder meta-analysis.

age will have on the concentration-response coefficient, but both characteristics may certainly play a role and should therefore be included as moderator variables.

Empirical dose-response studies have found that mortality among the elderly is more responsive to changes in particulate pollution than is mortality for the entire population or mortality among the younger generation (Ostro et al. (1996), Schwartz and Dockery (1992a)). Evidence further suggests that air pollution has its greatest adverse effects on people with pre-existing chronic conditions such as asthma, bronchitis, and emphysema (Ostro (1987)).

Age and baseline health will tend to be closely associated when looking at entire populations. In particular, if a population has a large percentage of elderly people it indicates that the baseline health of that population is rather high, enabling so many to live to an old age. Hence, if the baseline health variable is omitted from the regression analysis the age-variable, which is supposed to pick up the part of the population that is most at risk from high air pollution, will also proxy for the average health level of the population. These are two offsetting effects, and age will hence tend to be biased downwards. Baseline health levels will tend to be associated with level of income, although the association will probably be highly sensitive to the measure used for baseline health. Low-income individuals may have worse baseline health levels if low income and little education have given rise to wrong and/or insufficient nutrition and other health investments in the past.⁹ On the other hand, people who have a history of chronic obstructive pulmonary disease or cardio-pulmonary problems are also thought to be particularly vulnerable, and these types of health problems are more pronounced in high-income groups and countries.

There are several reasons why one would expect the increase in mortality due to ambient particles to vary with income.¹⁰ Firstly, for a certain increase in ambient concentration of air pollution we argued that lower income groups were likely to experience a larger increase in exposure than were the higher income groups because the former are not being able to afford much averting activities (e.g. sealing houses to reduce the penetration of outdoor pollutants, using less-polluting heating and cooking fuels, spending less time in traffic). Secondly, for a certain amount of exposure and its anticipated health effect we suggested that the behavioural response (e.g. visiting a doctor, taking medication) will typically be influenced by income level. These mitigating measures imply costs which poor people may not be able to afford, or willing to pay

⁹ Refer to B.M.M. Gaarder (2002), section 5.4.3 in chapter 5.

¹⁰ Refer to chapter 5 in B.M.M. Gaarder (2002) for a more detailed discussion.

given their budget constraints. Finally, we argued that there may be differences between low and high income groups, and even more so between low and high-income countries, in the extent to which official mortality statistics reflect actual mortality. It is not unlikely that deaths among the poor will be underrepresented or unavailable in official statistics. Although this latter point may imply an under-representation of the increase in mortality due to air pollution in lower income groups or countries, we suggest that the overall measured adverse health effects of an increase in air pollution will tend to be larger in low-income countries than in higher-income countries. Income should therefore be included as a mediator variable.

There are additional reasons why exposure may differ between developed and developing countries, and why an increase in exposure may lead to a larger increase in mortality in low than high-income countries that are not necessarily due to income levels, although income may be part of the underlying explanation for these factors. Firstly, the effect of an increase in pollution on exposure may be larger in low than high-income countries due to the fact that many low-income countries are situated in warm climates and the residents in these climates are therefore likely to spend a greater portion of their time outdoors (Ostro (1994)). Other differences between low and high-income countries may also influence the amount of time spent outdoors, such as crime rates, indoor air pollution, and social interaction traditions. Furthermore, the pollution level locally at the work place may be higher in less developed countries due both to the cost of abatement and less strict work place regulation. Finally, an increase in exposure may lead to a larger increase in mortality in low than high-income countries due to the quality and availability of health care. In addition to the often very restricted availability of health care, the quality of health care in developing countries is often poor, something which may affect the efficiency of mitigating measures. Hence, the risk of dying from the health effect of air pollution may be influenced by own behaviour or by the facilities available, and could be higher for lower income groups or cities. Due to the lack of reliable data on availability and quality of health care, time spent outdoors, work place pollution etc., such variables have in general not been included as moderator variables. By excluding these from our analysis, we implicitly allow income to proxy for their effects.

There are at least three reasons to believe that the income distribution in a country, i.e. relative income, is important for the difference in health effects. First, unless the effect of income on the dose-response coefficient is linear, using an average income variable will not capture correctly the sum of the effects of each individual's income on his or her adverse health. In particular, there are probably decreasing returns to averting

and mitigating activities which would imply a tendency for higher inequality to be associated with higher mortality rates.¹¹ Second, the location at which people live within a city will affect the amount of air pollution they are exposed to. Although individuals can move between cities, it seems likely that for most cities housing prices are determined by within-city demand. Hence, it is not so much the income level as the position within the income distribution that determines where an individual lives. If individuals with relatively low income tend to live in the most polluted areas, as evidence suggests, and if the adverse effect of air pollution is larger on lower income individuals (due to lower baseline health, less education etc.), then this would once again imply a larger PM₁₀-mortality in cities with large income-inequality.¹² Third, there is a line of research that implicates the biochemical effects of psychological stress as a risk factor, and relates this stress to social status (Deaton and Paxson (1999)). Social status can then be modelled as income relative to the average income. If mortality is associated with stress, and stress is related to social status (income relative to the average income), then this is a third reason why higher income inequality may lead to a larger mortality rate from air pollution. GINI may be proxying for inequalities in baseline health or for the quality and availability of health care, if satisfactory measures for these two variables are not available.

It may also be of interest to consider whether the effect of income inequality on the mortality rate from air pollution varies according to the average level of income at which the inequality takes place. On the one hand, one could speculate that high income inequality in a low-income country would imply a large amount of people not being able to undertake any averting and mitigating activities whatsoever (demand-side), and that only a small increase in income and health investment for these population groups therefore would have a large effect in reducing mortality. On the other hand, the range of averting and mitigating measures available to the public (the supply-side) and the information about the effects of pollution and how to minimise these may well be larger in high-income countries, implying that the way in which income is distributed may play a more significant role in determining the amount of deaths caused by particulates. Furthermore, high income-inequality in high-income countries may arguably lead to

¹¹ There are several reasons why we find decreasing returns to health investment likely. First, it is reasonable to assume that the most cost-efficient measures are undertaken first. Second, it is not unlikely that a similar health investment measure has a larger positive effect at high levels of exposure and low levels of baseline health than at lower levels of exposure and better health levels, and the two latter characteristics tend arguably to be associated with higher income groups.

¹² A cautionary remark is in order: if the effect of air particles on mortality were to be increasing at a decreasing rate, then the above finding would not necessarily hold. Empirical evidence so far, however,

more psychological stress than in low-income countries. For the above reasons, we suggest considering both the effect of a relative income-inequality measure, as well as an income distribution measure that takes the average level of income into account as moderator variables in the regression analysis.

The income variable we ideally would like to have is the average income in the location in question (be it a city or otherwise), or even more precisely, the average income of the vulnerable population within the relevant location. We were not able to obtain this information, however, and had to settle for a second-best option, namely the average income in the country in question. If the average level of income is similar in the study location as it is in the country as a whole, this will be a satisfactory approach. However, it is not unlikely that for the study locations, most of which are relatively large cities, this will not be the case. If the average income level in large cities differs in a consistent manner from the country average, then it is possible that the best way of capturing the average city-income is a composite of the average country income and the income distribution in the country.

The level of education may affect the knowledge people have about health, health production, and the connection between air pollution and health, and hence affect the level of baseline health, as well as the amount of averting and mitigating expenditures undertaken and the efficiency of these expenditures. Although there is conflicting evidence as to whether little knowledge/education leads to over or under-investment in health, we suggest that education should be included as a moderator variable. Since schooling is closely associated with income, the income coefficient will probably proxy for this variable if it is not included as a moderator variable.

Finally, the health services provided in a country are likely to influence the amount and severity of adverse health incidences. Health services are likely to be highly positively correlated with baseline health and the amount of people over the age of 65, and negatively with income inequality.

The expected signs of the moderator variables presented and discussed in this section are summarised in table 1 below.

does not support this, rather, a linear or even a convex function are usually assumed.

Table 1: Summary of the expected signs of the moderator variables in a table

<i>Expected Signs of Moderator Variables</i>	
Moderator variable	Sign
Air Pollution	+
Baseline Health	-
Age	+
Income	-
Income Inequality	+
Composite Variable (Interaction Variable) of Income and Income Distribution	?
Education	-
Health Services	-

All of the above mentioned variables are potentially important moderator variables, especially when transferring estimates to cities in developing countries which may take substantially different values on all of these. The level of air pollution is in general significantly higher in many developing countries than in the developed countries that generated most of the literature. Furthermore, an important difference between developed and developing countries is that the former tend to have an ageing population, whereas the latter have a majority of young people (higher birth-rate and lower life-expectancy), and we therefore find it potentially interesting to include this moderator variable. A crucial difference between developed and developing countries is the lower average income level in the latter. In addition, income in developing countries tends to be more unequally distributed (the average GINI-coefficient for the low-income countries in the World Development Report 1998/99 is 0.41, and for the high-income countries it is 0.30). As for health levels and education, these are both closely associated with income and thus typically on average much lower in developing countries than in their richer counterparts.

If the original studies have not satisfactorily controlled for confounding variables such as other pollutants, the ratio of fine particles to overall particle concentration, temperature, season, and humidity, and if these are correlated with the measured ambient particles, the resulting dose-response coefficients may be biased. However, assuming that the original studies have (linearly) controlled for various confounding factors, these may still have an impact on the measured effect size of air pollution on mortality. As information on these variables was missing in many of the studies in our sample such moderator variables were left out. However, a meta-analysis focussing on such confounding variables was carried out by Levy et al. (2000).

IV. Sample Selection, Data, and Methodology

4.1 Sample

The sample on which we perform the meta-analysis is composed of time-series studies gathered from previously published meta-analyses or review articles (Maddison and Gaarder (2001), Levy et al. (2000), Institute for Environmental Studies (2000)), as well as from PubMed.¹³

The selection of the wider sample is based on the following criteria for inclusion:

1. papers including the quantification of either Total Suspended Particles (TSP), Black Smoke (BS), or Particulate Matter (PM) larger than 2.5 μm in diameter;
2. published papers evaluating the association between exposure to particles and total mortality;
3. mortality figures modelled using Poisson regression analysis;
4. studies carried out on a representative sample of the population (e.g. excluding studies carried out on particular age groups); and
5. analysis controlling the confounding effect due to meteorology and temporal effects.

Papers not presenting information on the variance, standard error, or confidence intervals of the estimated coefficient were excluded. Furthermore, papers reanalysing the same site and time period (either by the same or different authors) were excluded on the grounds of double counting. Instead of restricting the sample to APHEA and any available developing country studies, as we did in the Maddison and Gaarder study (2001),¹⁴ all available studies were included. In total, 70 estimates from 56 studies and 21 countries were selected.

A number of factors potentially influencing the estimated dose-response coefficients were not used as criteria for inclusion or exclusion, but were rather the subjects of sensitivity analyses. In the case of the total mortality measure, we found it interesting to investigate whether inclusion of studies looking at all-cause mortality rather than just non-accidental mortality had a significant effect on the regression results. Similarly, testing the sensitivity of our findings to the air particle measurements used, as well as the lag structure, could potentially yield new insights into the underlying relationship between air pollution and mortality.

¹³ PubMed, a service of the National Library of Medicine, provides access to over 11 million citations from MEDLINE and additional life science journals.

¹⁴ This was mainly based on 7 time-series studies (10 observations) resulting from the APHEA project for European cities (see Katsouyanni (1997) for an overview). The sample was supplemented with studies from Chile (Ostro et al. (1996)), Sao Paolo (Saldiva et al. (1995) and Delhi (Cropper et al. (1997)).

A further factor likely to affect the estimated association between exposure and health in low and high-income countries differently is the way in which exposure has been measured. As adequate information on indoor air pollution in different countries was not available this factor could not be subjected to a sensitivity analysis, however, it will be important to keep in mind when interpreting our results. Ambient pollution at central monitoring stations may be particularly ill-suited to capture particulates exposure in low-income countries. Studies have found that indoor air pollution levels are as high if not higher than outdoor levels in several developing countries due to lack of air conditioning and some indoor sources present (e.g. Chestnut et al. (1998), Baek et al. (1997)). If it is vulnerable people (low baseline health levels, or of higher age) who tend to die from air pollution, and if indoor air pollution does not strongly covary with outdoor air pollution from day to day, then the exposure-response association may be much larger but not be captured by studies that use readings from central monitoring stations to measure exposure. In other words, those who are vulnerable to outdoor air pollution may already have died from indoor air pollution.¹⁵

As for the amount of pollutants included in the regression model, it could be used neither as inclusion/exclusion criteria, nor as a subject for sensitivity analysis. The main reason for this is that many studies were unclear as to whether the final results they reported for the particulate mortality coefficient were actually based on single, dual, or multiple pollution models. From the studies that did express clearly the amount of pollutants involved in their regressions we know, however, that a large majority of the time-series studies included in our sample feature single-pollutant rather than multi-pollutant regressions. The potential drawbacks of both single and multiple pollutant regressions are discussed briefly below.

Some epidemiologists are uneasy with the reliance on single pollutant regressions because different pollutants tend to be highly correlated (Moolgavkar et al. (1995)). They argue that it is premature to single out one of them as being responsible for the observed correlation between air pollution and mortality. Furthermore, the use of single pollutant models renders the interpretation of the available evidence difficult, since it is not known if the deaths attributed to the different air pollutants are additive or not. Finally, choosing

¹⁵ Studies (e.g. Baek et al. (1997), Chestnut et al. (1998), Janssen et al. (1998)) comparing indoor and outdoor concentrations of air pollution found the difference to be attributable in part to human indoor activities (e.g. type of stove used for cooking and heating, ventilation, tobacco smoke). Clearly, the more indoor air pollution is attributable to indoor activities, the less indoor air pollution will covary with outdoor air pollution.

one pollutant as a marker for air pollution can lead to under-estimation of the problem if in fact several air pollutants are responsible.

The use of single pollutant regressions has been defended in the literature by Schwarz et al. (1996b). They argue that given the correlation between the pollutant variables and the relatively low explanatory power of air pollution for mortality, including multiple pollutants in the regression risks letting the noise in the data choose the pollutant.

We will assume that the studies selected on the basis of our selection criteria were independent samples from a random distribution of the conceivable population of studies. In section 4 we will return to this issue and discuss why this assumption may be difficult to support.

4.2 Data

A number of airborne particulate measurement methods have been used in exposure-response studies. Gravimetric (weight) measurements of collected particles yield direct measurements of airborne particle mass. The high-volume sampler collects and measures the mass of total suspended particulates (TSP), whereas more recent samplers include devices to selectively collect and measure the mass of various size fractions of PM (e.g. PM₁₀, PM₁₃, PM_{2.5}). Two optical, and thus indirect, methods of measuring the mass of collected particles have also been frequently used. The black smoke (BS) method is based on light reflectance from particle stains on sample collection filters, whereas the coefficient of haze (COH) method is based on light transmission through the filter stain. According to the EPA, credible estimates of particle concentrations (in $\mu\text{g}/\text{m}^3$) can only be made via site-specific calibration against mass measurements from collocated gravimetric sampling devices. (EPA (1996), Vol. I, 1-6). The correlation between the different particle measures may have seasonal, meteorological, and geographical variations, and the fact that various particle mass measures are employed in different studies therefore complicates using any particular particle measure as indicator of airborne particulates. Some measurement error is necessarily induced by using common converters.

Each study in the meta-analysis supplied mean values of daily data over the study period (often from several monitoring sites) for either TSP, BS, or PM. TSP and PM₁₃ were converted to PM₁₀ using the factors of 0.55 and 0.77, respectively, and black smoke was considered equal to PM₁₀. Note that this implied dividing the estimated coefficients in studies using the TSP and PM₁₃ measures by 0.55 and 0.77, respectively, in order to convert these into being PM₁₀ or BS effects. When converting TSP to PM₁₀ we relied on the estimate

of EPA,¹⁶ which suggested that PM₁₀ is between 0.5 and 0.6. of TSP. We chose the mean of 0.55 as our conversion factor. As for BS, data from co-located BS and TSP monitors¹⁷ suggest an average ratio of BS/TSP of 0.55, and it is therefore assumed BS is roughly equivalent to PM₁₀. The conversion factor for PM₁₃ to PM₁₀ was simply obtained by dividing 10 by 13. A few studies used both BS and TSP as particle measures, and in these cases we chose the TSP measure, a gravimetric measure and therefore more straightforward to convert to PM₁₀. Particles in ambient air are usually divided into two groups according to size: fine (diameter less than 2.5 μm) and coarse (diameter larger than 2.5 μm). The two size fractions tend to have different origins, composition, and health effects and this makes conversions from fine particle measures to coarse problematic. PM_{2.5} and COH are essentially fine particle measures, and studies using these measures have been excluded from the present analysis.

The proportion of population over 65 (OVER65) was used as a measure of the segment of the population that empirically has been found to be most at risk from the acute effects of air pollution. These data were obtained on a country-level from the World Bank (SIMA).¹⁸ The SIMA data-base provided yearly observations on the percentage of the population over 65 years of age for all the study countries and all the required years. The OVER65-measure used in our regression analysis is hence the average for the relevant study period. Studies carried out in the same country may therefore have different OVER65-measures because they were carried out in different time periods. Three cautionary remarks are in order. First the studies are carried out in specific geographical entities within a country that do not necessarily have the same age distribution in their population as the country overall and this may therefore introduce some degree of measurement error into our regression analysis. Second, the impacts of air pollution on deaths by age group may be very different in low-income than in high-income countries. Cropper et al (1997) found that in Delhi peak effects occurred in the 15 to 44 age group, whereas in the US peak effects occur among people 65 and older. Finally, certain studies have also found that young children may be more susceptible than the average population to high levels of air pollution. A large proportion of people over 65 will tend to be negatively correlated with the proportion of young children, and this may thus bias the OVER65 variable downwards.

GNP per capita at purchasing power parity (PPP) is used as a measure of average

¹⁶ See EPA (1982).

¹⁷ See Cummings and Waller (1967).

income in the regression analysis. PPP GNP is gross national product converted to international dollars using purchasing power parity rates.¹⁹ An international dollar has the same purchasing power over GNP as a U.S. dollar has in the United States (i.e. the same amounts of goods and services can be purchased in the domestic market as a U.S. dollar can in the United States). Estimates on PPP GNP were obtained from the World Bank (SIMA). The SIMA data-base provided yearly observations for most of the study countries from 1975 onwards. The income measure used in our regression analysis is hence an average for the relevant study period. Main weakness of the measure is the fact that the income level in the location where the study was carried out may differ significantly from the overall income level of the country.

The GINI-coefficient was used to measure inequality in the income distribution of a country. The Gini-coefficient measures the extent to which the distribution of income among individuals or households within an economy deviates from an equal distribution. A Lorenz curve plots the cumulative percentages of total income received against the cumulative number of recipients, starting with the poorest household. The Gini-coefficient measures the area between the Lorenz curve and the line of absolute equality, expressed as a percentage of the maximum area under the line. Hence, a Gini-coefficient of zero represents perfect equality, and an index of 100 implies perfect inequality (World Development Indicators 2000). Estimates of the Gini-coefficients were obtained from the World Bank (SIMA). It is important to note, however, that the number of observations over time is very limited for most countries, and furthermore that national data differ greatly in terms of how data are collected and expressed (e.g are the coefficients calculated for income or consumption, gross income or taxable income, household income or individual income?). Furthermore, the income distribution of the cities in the meta-studies are not necessarily the same as the overall income distribution of their respective countries. The GINI-coefficients will therefore most probably measure income distribution with some degree of error.

The interaction term between the GINI-coefficient and GNP per capita, DIST, will reveal whether the effect of the distribution of income on the slope of the dose-response function differs between low- and high-income countries.

¹⁸ SIMA is the World Bank's internal database system containing more than 40 databases from the Bank and other international institutions.

¹⁹ Purchasing power parity conversion factor is the number of units of a country's currency required to buy the same amounts of goods and services in the domestic market as U.S. dollar would buy in the United States. Purchasing power parity conversion factors are estimates by World Bank staff based on data collected by the International Comparison Programme (World Development Indicators 2000).

Several measures of education were considered; enrolment ratios (education participation), expected years of schooling and illiteracy rates (education outcomes), as well as indicators for education efficiency. Out of these indicators only data on net and gross enrolment ratios were available for a large number of countries (and all of the countries included in the analysis). The gross enrolment ratio is the ratio of total enrolment, regardless of age, to the population of the age group corresponding to the relevant level of education, whereas the net enrolment ratio is the ratio of the number of children of official school age actually enrolled in school to the population of the corresponding official school age. Because the gross enrolment ratio necessarily also includes repeaters, a high ratio does not necessarily indicate a successful education system. For this reason we have chosen net enrolment as the preferred education/knowledge indicator. A drawback of the latter indicator is that children who start school at an age earlier or later than the official school age will not be included in this ratio. More generally, enrolment does not reflect actual attendance, and there may be reasons for overstating enrolments if for example teacher pay is related to student enrolment. Two net enrolment ratios were available; one for primary and one for secondary education. Net enrolment in secondary education was chosen as our education indicator (EDUC) because the majority of the countries in our sample had a net primary enrolment ratio of 100 percent, rendering the latter indicator powerless as a moderator variable. Observations on net secondary enrolment ratios were available for all the countries in the analysis back to 1980. The data for net secondary enrolment ratio was once again obtained from SIMA, and were available from 1980 onwards. They were averaged over the relevant study period. A measurement error may have been introduced due to the fact that enrolment ratios locally may differ from country-level ratios.

Two principal approaches are used to provide summary measures of population health. Disability-Adjusted Life Expectancy (DALE) summarises the expected number of years to be lived in the equivalent of 'full health', i.e. adjusted to take account of time lived with a disability or illness. Disability-Adjusted Life Years (DALYs), on the other hand, are a gap measure; they measure the gap between a population's actual health and some defined goal (a long life free of illness and disability). The relationship between life-expectancy at birth (LEAB), DALE, and DALYs can easily be shown with the help of a graph depicting survival curves (figure 1). The survivorship curve (bold line in figure 1) indicates, for each age along the x-axis, the proportion of an initial birth cohort that will remain alive at that age. Life expectancy at birth is equal to the total area under the survivorship curve (i.e. it equals areas A+B). Area A is time lived in full health, whereas

area B is time lived in a health state that is less than full. Disability-adjusted life expectancy weighs the time spent in B by the severity of the health states that B represents before adding it to the area below the full-health-survivorship curve (i.e. area A). Finally, disability adjusted life years quantify the difference between the actual health of a population and some stated goal for population health (in figure 1 the health goal is to live in ideal health until the death-day). DALYs weigh the time spent in B by the severity of the health states that B represents before adding it to the area above the full-health-survivorship curve, i.e. area C. (Mathers et al. (2000)).

DALE is estimated using information on the fraction of the population surviving to each age (calculated from birth and death rates), the prevalence of each type of disability at each age, and the weight assigned to each type of disability. Survival at each age is adjusted downward by the sum of all the disability effects, each of which is the product of a weight and the complement of a prevalence (the share of the population not suffering that disability). The adjusted survival shares are then divided by the initial population to give the average number of equivalent healthy life years that a new-born can expect. If we enumerate health states, S , using a discrete index h , DALE can be calculated as follows:

$$DALE_x = \sum_h \int_x^L w_h(u) \times S_h(u) du$$

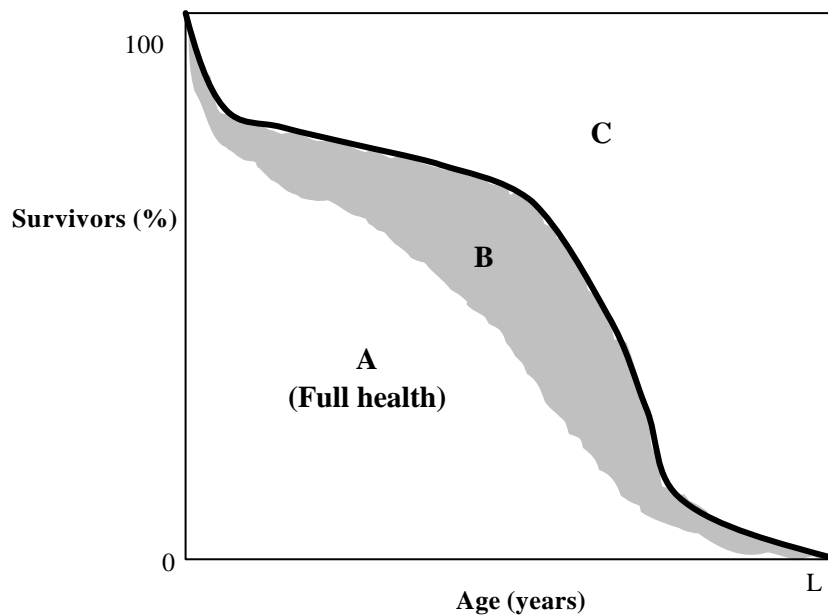
where w_h is weight, u represents age, and the integral is over ages from x onwards (L represents the end of the life-time).

The DALE estimate for the population of each country was found in the World Health Report, Annex Table 5, of the World Health Organisation. As this is a relatively newly developed health indicator, estimates were available for 1999 only. Although this is an indicator that may not be changing rapidly, it will nevertheless be an unprecise measure of baseline health, especially in the older studies.

Although an individual with low health levels is more likely on average to die relatively early compared to an individual with higher health levels, life-expectancy at birth (LEAB) is an inaccurate measure of population health since it does not take illness and disability into account. The advantage of this measure is that it was available in SIMA, and has been calculated for the countries in our sample with irregular intervals since the 1970's. LEAB therefore offers the possibility, although imperfect, of adjusting our health measure to reflect the period in which a particular study was carried out.

Other health indicators are either focusing on specific population groups (e.g. infant mortality), specific adverse health occurrences (e.g. per cent of population with HIV), or are at most indirect measures of population health by measuring expenditure on health or health facilities per capita (e.g. amount of inhabitants per hospital or per doctor), and are therefore not interesting for the present purposes.

Figure 1: Survivorship function for a population



Source: Mathers et al. (2000).

Finally, for the purpose of testing the sensitivity of some of our results we wanted to include a measure of the countries' health services. We rejected the use of health expenditure data as a measure of availability and quality of health services, on the ground of being a measure of input that would "reward" inefficient health service systems. From the health service indicators and health utilisation indicators supplied in SIMA, only the former (physicians and hospital beds per 1000 people) were available for all of the countries in our sample. The number of physicians per 1000 people was chosen as measure of the health service in a country. Data were available in SIMA, and have been calculated for the countries in our sample with irregular intervals since the 1970's, hence approximated averages could be calculated for the study periods. The main weakness of this measure is that it does not reveal anything about the distribution of these physicians in various regions or income-classes. In addition, some countries incorrectly included retired physicians or those working outside the health sector.

The data can be found in a table in appendix D.

4.3 *Methodology*

In this section we will briefly compare two alternative regression methods, derive the log likelihood function for the mixed effect Empirical Bayes model, as well as describe the tests for homogeneity and for outliers.

In order to obtain the coefficients of the moderator variables two alternative regression methods will be described and briefly compared. In Variance-Weighted Least Squares regressions (VWLS), the concentration-response functions are weighted according to the statistical precision of the studies using the inverse of the variance of each study. This is the method used by Maddison and Gaarder (2001). VWLS differs from Ordinary Least Square (OLS) in that homogeneity of variance is not assumed – the conditional variance of the dependent variable is estimated prior to the regression. VWLS treats the estimated variance as if it were the true variance when it computes standard errors. This method implicitly assumes that all the variance among the study effects other than sampling variance can be explained as a function of known study characteristics (i.e. there is no unexplained between-study variability). We consider this an unrealistic assumption, and note that when available knowledge is insufficient to account for the between-study variation, the model is misspecified. The Empirical Bayes method offers a way of dealing with the insufficiency of knowledge, in particular; it allows us to model the variation among the effect sizes as a function of study characteristics plus error. Empirical Bayes is therefore the main method used in this paper.

According to Raudenbush and Bryk (1985), the Empirical Bayes meta-analysis can be considered a special case of a two-stage hierarchical linear model. The first stage consists of estimating a within-study model separately for each study, and at the second stage a between-study model explains variation in the within-unit parameters as a function of differences between units. This distribution of the true effect size consists of a vector of known constants representing differences between the studies, a vector of between-study parameters, and a random error term, and it is referred to as the prior distribution of the true effect size. Empirical Bayes methods provide a general strategy for estimation when many parameters must be estimated and the parameters themselves constitute realisations from a prior probability distribution.

Estimates can differ partly due to the fact that the studies use different samples of the total population and partly due to the differing conditions under which the research takes place. Fixed effects models assume the existence of a common effect size in all the studies, whereas random effects models assume a different real effect in each study. In the latter

case, combining effect sizes from empirical studies means assessing the average size of the real effect. The common or average effect can be found by calculating the variance weighted average of the effect sizes found, and will be called \mathbf{b}_w . In order to choose whether the fixed or the random effects model is the most appropriate, we can perform a homogeneity test using Cochran's Q-statistic defined as:

$$Q = \sum_{i=1}^k \frac{(\mathbf{b}_i - \mathbf{b}_w)^2}{v_i} \quad (9)$$

where v_i is the variance of the reported effect from study i , \mathbf{b}_i . If the sample size is large in each study, Q asymptotically has a X^2 -distribution with $k-1$ degrees of freedom. The hypothesis of homogeneity will be rejected if the value of Q is large.

If we reject the hypothesis of equal real effect sizes, the next question is then whether we can find moderator variables that explain the variations between the empirically estimated effect sizes. If a linear combination of variables fully explains the variations in the real effect sizes, then the effect size is fixed and not random (although the real effect sizes are different in each study). This is, however, a rare case. In most cases it is more realistic to use a model that takes into account the imperfections of the explanatory model.

Let us briefly recapitulate the main equations for the mixed effect model already presented in section 2. We assumed that the estimated effect size d_i of study i is a function of known study characteristics W_i , random errors u_i (inter-study variability) and errors of estimate e_i (intra-study variability):

$$d_i = W_i' \mathbf{g} + u_i + e_i$$

Assuming that the error terms are independent, the marginal distribution of d_i is:

$$d_i \sim N(W_i' \mathbf{g}, v_i + \mathbf{t}^2)$$

Raudenbush and Bryk (1985) use maximum likelihood techniques to derive empirical Bayes estimates "because these techniques are more widely understood than Bayesian methods". If we assume that the estimate of v_i from each study is approximately equivalent to its true value, we can find the likelihood of the data as a function of \mathbf{t}^2 alone, and thereby find the likelihood estimate of \mathbf{t}^2 .

Following Raudenbush and Bryk, \mathbf{t}^2 is determined by maximum likelihood method, where the log of the likelihood is proportional to:

$$-\sum \log(v_i + \mathbf{t}^2) - \log \left| \sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' \right| - \sum (v_i + \mathbf{t}^2)^{-1} (d_i - W_i' \mathbf{g}^*)^2 \quad (10)$$

Furthermore, \mathbf{g}^* is the maximum likelihood estimate for the vector of derived coefficients, and is given by the following expression:

$$\mathbf{g}^* = \left(\sum \mathbf{I}_i W_i W_i' \right) \sum \mathbf{I}_i W_i \mathbf{b}_i \text{ where } \mathbf{I}_i = \mathbf{t}^2 / (v_i + \mathbf{t}^2)$$

The mathematical derivation of these results is presented in appendix B.

We developed a new programme in STATA (version 6) in order to maximise the above likelihood function, which can be found in appendix C.

There are three key issues in identifying model sensitivity to individual observations, and these are known as residuals, leverage, and influence. The residuals reveal the distance between the value of the i th dependent variable, Y_i , and the fitted value, Y' , and an outlier is identified by a large residual. The leverage, on the other hand, reveals the distance between the value of the independent variable for the i th observation, X_i , and the mean of all the X values, \bar{X} . Having a large leverage can hence also identify an outlier. However, points with large residuals may, but need not, have a large effect on the results, and points with small residuals may still have a large effect, and similarly for the leverage. ‘Influential’ is therefore defined with respect to an index that is affected by the size of the residuals and the size of the leverage. Two outlier tests were performed on our sample. The first test, suggested by Belsley, Kuh, and Welsch (1980), requires that DFITS values greater than $2\sqrt{k/n}$ are subjected to further investigation. The DFITS can be written as follows:

$$DFITS_i = r_i \sqrt{\frac{h_i}{1-h_i}}$$

where r_i are the residuals, h_i is the i th leverage, k is the number of explanatory variables (including the constant), and n is the number of observations. DFITS is an attempt to summarise the information in the leverage versus residual-squared-plot into a single statistic. The second test, known as Welsch’s Distance W_i , is defined as follows:

$$W_i = DFITS_i \sqrt{\frac{n-1}{1-h_i}}$$

The cutoff for Welsch Distance is $3\sqrt{k}$.

V. Results

5.1 Main Findings

In the sample the coefficients reported were used no matter what lag structure was used and whether additional pollutants were included in the model or not. If several coefficients were reported we used the one favoured by the researcher, and if no preference was mentioned we chose the most significant coefficient. In the 8 studies reporting results

both for single and multiple pollutants we used the preferred single pollutant results, since the large majority of studies only reported single pollutant results. The Apeha group decided to search for the best relationship of each pollutant with mortality with a lag of up to 3 days, and the best cumulative effect of several days including the same day and up to 3 previous days, and the EMECAM project in Spain followed the methodology developed by the Apeha group. In practice, this led to a sample largely consisting of single pollutant models.

We used Cochran's Q -statistic on the pooled random effects model to assess homogeneity of the studies, and found significant heterogeneity in effect estimates. The value of the test-statistic is 329, exceeding the 99th percentile point of the Chi-square distribution with 69 degrees of freedom. Applying the two outlier-tests described in section 4.3 to the pooled random effects model, we found that no observation in the sample failed the Welsch's Distance test. Three observations (Huelva, Pamplona, and Basel) failed the DFITS cutoff point; however, the heterogeneity in effect estimates remained even after these were excluded from the sample (the value of Cochran's Q -statistics was 301).

When we apply the EB model to the full sample of 70 observations, a model without predictors yields an estimated grand mean of the regression coefficients of 0.00060, which is equivalent to a 6.0 percentage (CI: 5.1 – 6.9 per cent) increase in daily mortality associated with a 100- $\mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations. This pooled estimate is similar to the ones found by Levy et al. (2000), Borja-Aburto et al. (2000), and Ostro (1993), although slightly lower.

When moderator variables were entered one at a time into the model which included only a constant, the income inequality variable, GINI, had the greatest significance from the range of moderator variables available ($z=3.76$). When this moderator variable was added to the model the t^2 decreased from 1.83e-07 to 1.61e-07 (for the other moderator variables the following t^2 values were obtained: DIST $t^2 = 1.68\text{e-}07$ ($z=3.27$); GNP $t^2 = 1.77\text{e-}07$ ($z=2.34$); POLL $t^2 = 1.87\text{e-}07$ ($z=-1.30$); OVER65 $t^2 = 1.87\text{e-}07$ ($z=-0.13$); EDUC $t^2 = 1.87\text{e-}07$ ($z=0.39$); DALE $t^2 = 1.86\text{e-}07$ ($z=0.82$); LEAB $t^2 = 1.85\text{e-}07$ ($z=1.20$)).

By comparing equations 0a and 2a and 0b and 2b in table 2, it is clear that practically the same results are obtained whether we use DALE or LEAB as the baseline health measure. We therefore choose DALE as baseline health indicator from this point onwards in our analysis because it is the theoretically preferred indicator. Our main finding is the positive and significant association of the GINI coefficient and its interaction term with GNP, DIST, with the dose-response coefficient. In particular, we

find that a four-point increase in the GINI-coefficient leads to an increase in the mortality rate from a $100\text{-}\mu\text{g}/\text{m}^3$ increase in PM_{10} of almost 1 percent. When DIST is included in the model (equations 0a and 2a) we furthermore observe that the coefficient on GNP takes a negative sign but is insignificant, whereas when GINI is included instead (equations 0b and 2b) the GNP-coefficient becomes positive and significant at the 0.05 level. To simplify matters we will from here onwards refer to the former model-specification as the DIST-model, and the latter as the GINI-model. If we do not include any variable picking up income distribution in our regression (equation 1), the average level of income, GNP, is found to have a positive and significant effect on the dose-response association. This seems to indicate that the direct effect of GNP on the dose-response coefficient is negligible, whereas its main effect is by interacting with income inequality. In particular, high income-inequality appears to have a larger (increasing) effect on the dose-response coefficient in high-income countries.

The education term is consistently taking a negative coefficient, but its z -statistic indicates that it is slightly below a level that can be termed significant. As for baseline health, measured either by standard or disability adjusted life expectancy, its coefficient varies but it is insignificant, as are both the constant and the level of average ambient pollution. Somewhat surprisingly, the percentage of the population aged 65 or older is consistently negative, however it is only significant in the case where income is included without any measure of distribution present in the regression (equation 1).

Finally, we included the number of physicians per 1000 people, PHYS, as a moderator variable in equations 4a and 4b. The effect of this variable was found to be negative but insignificant, and the only implication of including it in our regression model worth mentioning was a slight decrease in significance for both GINI and DIST. Both, however, remained significant.

Note that our findings would have been entirely different had we used the OLS or VWLS regression methods. This confirms how important it is to understand the assumptions underlying each method and to choose the correct one. See appendix E for the findings applying the main regression equations on the full sample using the methods of OLS and VWLS.

Table 2: Summary of regression results using EB-methodology on full sample (70).

Equation Method	0a EB	0b EB	1 EB	2a EB	2b EB
Dep. var.	<i>b</i>	<i>b</i>	<i>b</i>	<i>b</i>	<i>b</i>
OBS	70	70	70	70	70
CONST	-0.2417	-0.10689	-0.43615	-0.3296	0.3686
se	(1.3268)	(1.3091)	(1.329)	(1.8351)	(1.8300)
<i>z</i>	<i>(-0.18)</i>	<i>(-0.08)</i>	<i>(-0.33)</i>	<i>(-0.18)</i>	<i>(0.20)</i>
POLL	-0.00084	-0.00123	-0.00068	-0.000783	-0.00134
se	(0.00142)	(0.00141)	(0.00142)	(0.00145)	(0.00144)
<i>z</i>	<i>(-0.59)</i>	<i>(-0.87)</i>	<i>(-0.48)</i>	<i>(-0.54)</i>	<i>(-0.93)</i>
OVER65	-0.03609	-0.01917	-0.06204*	-0.02909	-0.01271
se	(0.02745)	(0.02908)	(0.02465)	(0.02604)	(0.02727)
<i>z</i>	<i>(-1.32)</i>	<i>(-0.66)</i>	<i>(-2.52)</i>	<i>(-1.12)</i>	<i>(-0.47)</i>
GNP	-0.000024	0.000028*	0.000041**	-0.000027	0.000028*
se	(0.000033)	(0.000014)	(0.000013)	(0.000033)	(0.000014)
<i>z</i>	<i>(-0.72)</i>	<i>(2.08)</i>	<i>(3.16)</i>	<i>(-0.82)</i>	<i>(2.07)</i>
GINI		0.02265**			0.02377**
se		(0.0085)			(0.00850)
<i>z</i>		<i>(2.67)</i>			<i>(2.80)</i>
DIST	1.52e-06*			1.54e-06*	
se	(7.14e-07)			(7.17e-07)	
<i>z</i>	<i>(2.13)</i>			<i>(2.15)</i>	
LEAB				0.02259	-0.00360
se				(0.02680)	(0.02844)
<i>z</i>				<i>(0.84)</i>	<i>(-0.13)</i>
DALE	0.0248	0.00508	0.02884		
se	(0.0218)	(0.0232)	(0.0218)		
<i>z</i>	<i>(1.14)</i>	<i>(0.22)</i>	<i>(1.32)</i>		
PHYS					
se					
<i>z</i>					
EDUC	-0.00958	-0.00627	-0.00891	-0.00855	-0.00581
se	(0.00586)	(0.00584)	(0.00587)	(0.00574)	(0.00566)
<i>z</i>	<i>(-1.63)</i>	<i>(-1.07)</i>	<i>(-1.52)</i>	<i>(-1.49)</i>	<i>(-1.03)</i>
Tau2	0.177***	0.170***	0.179***	0.178***	0.170***
<i>z</i>	<i>(4.80)</i>	<i>(4.76)</i>	<i>(4.80)</i>	<i>(4.82)</i>	<i>(4.76)</i>
Log like.	-84.39	-64.34	-59.04	-84.27	-63.94
Wald chi2	19.27	22.20	14.69	18.61	22.19

Source: Own regression results.

Note: The dose-response coefficient, **b**, and its standard error have both been multiplied by 1000 in order to make the numbers more readable. The standard error is given in parenthesis below the estimated coefficients, and the *z*-statistic is in italics and parenthesis. Tau2 is the inter-study variation.

Table 2 continued: Summary of regression results using EB-methodology (for full sample and sample excluding outlier (69)).

Equation Method	3a	3b	4a	4b
Dep. var.	EB	EB	EB	EB
OBS	<i>b</i>	<i>b</i>	<i>b</i>	<i>b</i>
	69	69	70 (+ phys)	70 (+ phys)
CONST	-0.2846	-0.12835	-0.44796	-0.2047
se	(1.2352)	(1.2173)	(1.4429)	(1.4295)
z	<i>(-0.23)</i>	<i>(-0.11)</i>	<i>(-0.31)</i>	<i>(-0.14)</i>
POLL	-0.000409	-0.00084	-0.00064	-0.00114
se	(0.001323)	(0.00131)	(0.00153)	(0.00154)
z	<i>(-0.31)</i>	<i>(-0.64)</i>	<i>(-0.42)</i>	<i>(-0.74)</i>
OVER65	-0.03758	-0.01903	-0.02997	-0.01651
se	(0.02571)	(0.02724)	(0.03151)	(0.03223)
z	<i>(-1.46)</i>	<i>(-0.70)</i>	<i>(-0.95)</i>	<i>(-0.51)</i>
GNP	-0.000032	0.000020	-0.000020	0.000028*
se	(0.000031)	(0.000013)	(0.000034)	(0.000014)
z	<i>(-1.04)</i>	<i>(1.54)</i>	<i>(-0.59)</i>	<i>(2.05)</i>
GINI		0.02356**		0.02213*
se		(0.00793)		(0.0091)
z		<i>(2.97)</i>		<i>(2.42)</i>
DIST	1.54e-06*		1.42e-06'	
se	(6.70e-07)		(7.68e-07)	
z	<i>(2.30)</i>		<i>(1.85)</i>	
LEAB				
se				
z				
DALE	0.01928	-0.00144	0.02756	0.00685
se	(0.02034)	(0.0216)	(0.02315)	(0.02527)
z	<i>(0.95)</i>	<i>(-0.07)</i>	<i>(1.19)</i>	<i>(0.27)</i>
PHYS			-0.03089	-0.01437
se			(0.07765)	(0.07688)
z			<i>(-0.40)</i>	<i>(-0.19)</i>
EDUC	-0.00385	-0.00046	-0.00947	-0.00634
se	(0.00560)	(0.00557)	(0.00594)	(0.00590)
z	<i>(-0.69)</i>	<i>(-1.03)</i>	<i>(-1.59)</i>	<i>(-1.07)</i>
Tau2	0.148***	0.141***	0.182***	0.176***
z	<i>(4.80)</i>	<i>(4.77)</i>	<i>(4.80)</i>	<i>(4.75)</i>
Log like.	-73.96	-53.43	-88.74	-68.77
Wald chi2	17.79	21.73	19.10	21.84

Source: Own regression results.

Note: The dose-response coefficient, **b**, and its standard error have both been multiplied by 1000 in order to make the numbers more readable. The standard error is given in parenthesis below the estimated coefficients, and the *z*-statistic is in italics and parenthesis. Tau2 is the inter-study variation.

Table 3: Correlations between predictors considered in the full meta-analysis (n=70). Values greater than 0.5 are in italics.

	over65	educ	Dale	Leab	gnp	gini	dist	poll	phys	se
over65	1									
educ	<i>0.60</i>	1								
dale	<i>0.82</i>	<i>0.65</i>	1							
leab	<i>0.80</i>	<i>0.63</i>	<i>0.94</i>	1						
gnp	<i>0.62</i>	<i>0.66</i>	<i>0.59</i>	<i>0.67</i>	1					
gini	-0.40	-0.21	-0.17	-0.12	-0.01	1				
dist	<i>0.45</i>	<i>0.59</i>	<i>0.46</i>	<i>0.55</i>	<i>0.94</i>	<i>0.28</i>	1			
poll	<i>-0.53</i>	<i>-0.63</i>	<i>-0.58</i>	<i>-0.63</i>	<i>-0.61</i>	<i>0.16</i>	<i>-0.54</i>	1		
phys	<i>0.77</i>	<i>0.41</i>	<i>0.67</i>	<i>0.66</i>	<i>0.32</i>	<i>-0.46</i>	<i>0.14</i>	<i>-0.22</i>	1	
se	<i>0.30</i>	<i>0.21</i>	<i>0.27</i>	<i>0.29</i>	<i>0.20</i>	<i>-0.04</i>	<i>0.17</i>	<i>-0.26</i>	<i>0.34</i>	1

Source: Own calculations.

5.2 *Sensitivity Analysis*

There are several reasons why one should be very cautious when generalising the results. First, the full sample is still relatively small, and single observations may affect the results unduly. Second, the conversion factors used between different measures of air pollution are approximations and may have affected the outcome. Also, there is a possible difference in the ‘positive results’-bias between various countries. Furthermore, both the mortality measures and the lag structure considered and/or reported vary between the studies and may have induced errors. Finally, the fact that some of the countries, as well as cities, in the study enter with multiple observations whereas others have only one may be problematic and requires investigation. Given these drawbacks of our sample, a sensitivity analysis is necessary before drawing conclusions about potential causal predictors of the PM₁₀-mortality relationship. Special attention is paid below to outliers, mortality measure, negative results, lag structure, pollution measurement, and the number of observations per location.

First, we undertook a search for outliers, i.e. subsets of the observations which, if deleted, would change the results markedly, and tested the sensitivity of our results to exclusion of these observations. When performing the DFITS-test (described in section 4.3) on our sample only Basel was singled out. From table 2 (equations 3a and 3b) we observe two main changes in estimated effects when excluding Basel from the sample. First, there is an increase in effect and significance of GINI and DIST in equations 3a and 3b, respectively. Second, a decrease in the positive effect of GNP occurs in equation 3b, and an increase in the negative effect of GNP occurs in equation 3a. In both cases GNP is insignificant. No observation in the sample failed Welsch’s Distance test.

Out of the sample of 70 pollution-mortality studies, 9 were not excluding mortality due to external causes from their mortality measure. If externally caused mortality is independent of air pollution, then including these studies in the full sample should not bias the results – they would at most bring more noise into the results. However, to our knowledge no study specifically investigates the association between accidents and homicides etc. and air pollution.

When excluding studies measuring all-cause mortality, the central effect estimate is 5.9 per cent (CI: 5.0 – 6.8 per cent), whereas only looking at all-cause mortality studies gives a mean of 6.5 per cent (CI: 0.13 – 1.18 per cent). Next, we tested whether the inclusion of studies measuring all-cause mortality induced estimation errors using the two main moderator effect models. In table 4 equation 5a we see that the main significant effect

of this exclusion was to increase the negative effect of GNP on the dose-response coefficient – this coefficient becomes weakly significant at the 0.1 level – and increase the size and significance of the interaction term, DIST. As for the other terms, their coefficients were similar in magnitude and direction to the optimum model and remain insignificant. When DIST is replaced by GINI (equation 5b), we observe that the increasing effect of GNP on the dose-response coefficient found in the full sample regression decreased and became insignificant through the exclusion of the 9 studies. The increasing effect of GINI on the particle-mortality association, on the other hand, increased further. The remaining coefficients remained insignificant, as compared to the full-sample model, and except for CONSTANT and DALE the coefficients of the additional terms had the same sign.

Out of the pollution-mortality studies, 6 reported negative results, and 4 out of these originated from the EMECAM program studies (i.e. Spain). If all confounding variables were controlled for, the model correctly specified, and the data measured without error, it seems unlikely that we would find negative estimates of the dose-response coefficient, i.e. indicating that air pollution decreases mortality. Hence, we could view the studies reporting negative coefficients as noise in our meta-analysis and therefore leave these out of the analysis.²⁰

The main objection against exclusion of negative results has to do with researcher-induced sample-selection bias. The negative studies may to some extent counteract the noise in positive studies, studies that we would not discard because they have the “right” sign. Furthermore, it is highly likely that if all results had an equal chance of getting published (i.e. without prejudices against negative results) we would in fact have had more negative coefficients in our meta-analysis.

If we exclude the negative coefficients from our sample (hence, sample size 64), the grand mean is 6.7 per cent (CI: 5.9 – 7.5 per cent). The findings from estimating the two main models using the sample consisting only of positive results are similar to those we obtained when we excluded all-cause mortality studies from the sample (equations 6a and 6b).

²⁰ An additional, but rather tentative, reason for excluding the negative results is that 4 out of the 6 originated from the EMECAM program studies (i.e. Spain). If all the countries in our analysis had an equal probability of reporting and publishing negative results, then including these studies in the full sample should not affect the relative significance of the moderator variables. However, were this not to be the case, then the ‘positive results’-bias may influence our conclusions. Since Spain and the United States receive the most weight in the meta-analysis due to multiple observations for each country, it is worth noting that the US studies were all published in international and competitive journals, whereas the Spanish studies were both carried out due to a government grant and published in a government-supported journal. The ‘positive results’-bias may therefore well have been larger in the US than in Spain, and for comparability of results one may hence argue that the negative results should be excluded.

As mentioned in section 4.1, the correlation between the different particle measures may have seasonal, meteorological, and geographical variations, and using fixed and common conversion factors will most likely induce some measurement error.

The central effect estimates when stratifying by pollution measure were 5.0 per cent (CI: 3.4 – 6.7 per cent), 6.6 per cent (CI: 5.1 – 8.2 per cent), and 6.4 per cent (CI: 4.9 – 7.8 per cent), for BS, TSP, and PM₁₀, respectively.²¹ We also tested the sensitivity of our findings from the main models to the air particle measurements used, by considering the coefficients derived using TSP, BS, and PM₁₀ separately. A main cautionary remark is called for before entering into the more specific challenges we are faced with when analysing the samples stratified by pollution measurement. The sample sizes for each pollution measurement is relatively small (24 observations for TSP, 33 in the case of PM₁₀, and 16 for the BS measurement), and any results have to be taken with the utmost caution. As most of the moderator variables are country based, rather than city based, and some (GINI and DALE) are available for a certain year only and not in time-series, the regression model developed above is only meaningful when a variety of countries are included in the sample. Furthermore, the larger the amount of studies in the sample originating from the same country, the more likely it is that the variables that do vary from one study to the next proxy for those that do not. Keeping this in mind, and given that 50 per cent of the studies using BS to measure air pollution are from Spain and approximately 50 per cent of those using PM₁₀ have been carried out in the US, we argue that the results from these two samples will not be very meaningful. In the case of the sample of 24 TSP based coefficients, 13 countries are represented and none with more than 5 coefficients. Equations 7a and 7b of table 4 give the results of the main model for the TSP-sample. When we compare the results with those of the full sample for the model which includes DIST, we observe a strengthening of the negative effect of GNP on the dose-response coefficient, although it remains insignificant. Furthermore, the size and significance of the interaction term increases. The remaining coefficients once more remain insignificant, however only EDUC which was close to being significant in the full sample retains the direction of its coefficient. With GINI replacing DIST (equation 7b), we observe that the positive effect of GNP on the dose-response coefficient found in the full sample regression decreases and becomes insignificant. The positive effect of GINI on the particle-mortality association, on the other hand, increases further. The remaining coefficients once more remain insignificant,

²¹ The sample sizes for the three air pollution measures estimates were 16, 24, and 33 for BS, TSP and PM₁₀ (including two studies using PM₁₃), respectively. The sample adds up to more than our full sample because 3 of the studies reported estimates in two of the measures.

and only the coefficients for POLL and EDUC do not change direction. For the sake of completeness the results of the main model for the PM₁₀ and BS-sample are given in equations 8 and 9 (table 4), respectively.

Next, we investigated the sensitivity of our findings to the choice of lag structure. In particular, we ran our favoured model on samples of the pollution-mortality coefficients that were obtained for average air particle levels of the same day (table 5, equations 10a and 10b), previous day (table 5, equations 11a and 11b), and of two days previously (lag 2 – equations 12a and 12b) in turn. Before proceeding, however, the same warning has to be made as was made in connection with the pollution measurements analysis on the previous page. The sample sizes for each lag is relatively small (28 observations for lag 1, 23 in the case of lag 1, and 12 for lag 2), and any results have to be taken with the utmost caution, and in particular in the case of the smaller samples is likely to drive the results. Interestingly, the results varied widely. For same day air pollution, the GINI coefficient was once again found to have a strongly significant²² positive effect on the dose-response coefficient, whereas GNP took a negative sign but was insignificant. POLL has a significantly negative effect on the relationship. As for baseline health, it is found to have an increasing and significant effect, whereas education has a decreasing and significant effect. For particle pollution lagged one day, none of the moderator terms have significant coefficients. In the case of two-day lagged pollution, however, GINI is once again weakly significant,²³ with the usual positive sign, and the coefficient for education is significant and this time takes a positive sign. The estimates of central effect were 5.3 per cent (CI: 4.3 – 6.2 per cent), 5.5 per cent (CI: 4.1 – 6.9 per cent), and 3.7 percentage (CI: 1.6 – 5.7 per cent) for lag 0, lag 1, and lag 2, respectively.²⁴

Furthermore, we tested the sensitivity of our findings to the decision to include several studies from the same city, in order to detect the potential effects of double counting. Only 5 cities have been entered twice in the sample, and we tested several combinations when only one of these studies was considered (sample size was then 65). As expected, the findings did not change significantly from those made for the full sample.

If we had obtained information on the values of the moderator variables at the city level, rather than at the country level, the fact that some countries enter with several observations whereas others only with one would not have been a cause for concern. However, this is not the case – only the average particulate measure was based on the exact

²² The null-hypothesis of no effect is rejected at the 0.1 per cent level.

²³ The null-hypothesis of no effect rejected at the 10 per cent level, but not at the 5 per cent level.

²⁴ The sample sizes for lag 0, 1, and 2 were 28, 23, and 12, respectively.

study location. Our final sensitivity test therefore consisted of including only one observation for each country. This can be done in three alternative ways. First, the variance weighted averages of both the dependent and independent variables, as well as of the standard error, are calculated for those countries with more than one observation, and the resulting averages can then be entered as observations for those countries. The second procedure is an extension of the first; the only difference is that we take the time period in which the studies were carried out into account, since we have time series data for most of the moderator variables. All studies from one country carried out mainly in the period 1975-85 were averaged, as were those for the periods 1985-95, and 1995-, implying that we had two “observations” each for Spain and Germany, and three for the US, and thus a sample of 25. Finally, we can consider only one (real) study per city, and a number of potential study combinations.

The two main regressions were performed on the simple country-averaged sample of 21 observations (table 6, equations 13a and 13b) and on the period sensitive country-averaged sample of 25 observations (table 6, equations 14a and 14b). The results from the two samples were similar to each other, and significantly different from our previous results. Once again, however, a cautionary remark is in order when interpreting the results, due to the sample sizes. Both education (EDUC) and the amount of older people in the population (OVER65) were found to have significantly negative effects on the dose-response coefficient, i.e. higher levels of these variables are associated with lower PM₁₀-mortality coefficients. As for the two income distribution terms, they were both insignificant when entered in turn. When entered together with the relative income inequality measure, GINI, the average income-term takes a positive coefficient and is highly significant. As for the income coefficient when the interaction term, DIST, is included in the model, GNP has a significantly positive coefficient in the sample of 21 observations, but is insignificant in the sample of 25. The estimates of the central effect is 4.9 per cent (CI: 3.9 – 5.9 per cent) in the sample with 21 observations, and 5.0 per cent (CI: 4.1 – 6.0 per cent) in the sample consisting of 25.²⁵

With over 2 million potential study combinations, a complete combination analysis was not deemed feasible. However, by randomly selecting 24 combinations and performing the two main regressions, we propose that we can gain some insights from which we can generalise.²⁶ Focussing first on the model including the GINI term, we found that GNP entered the regression significantly for 54 per cent of the study combinations, with a

²⁵ When including only one observation for each country, the sample size is 21.

²⁶ Refer to appendix F for the regression results.

consistently positive sign. Education entered with a consistently negative and significant sign for 50 per cent of the study combinations. In addition, DALE, OVER65, and GINI entered in descending order of frequency, although their frequencies were well below 25 per cent – GINI was in fact only significant in one study combination and pollution in none. Turning our attention to the model that included the DIST term instead of GINI, we found that GNP was now only significantly positive in 8 per cent of the study combinations. As for education, it now entered negatively and significantly in 71 per cent of the study combinations. DALE and OVER65 both again appeared in study combinations at frequencies below 25 per cent.

Table 4: Summary of regression results using EB-methodology (sample excluding all-cause mortality studies (61), sample excluding negative coefficients (64), sample of TSP studies (24), PM₁₀ studies (33), and BS studies (16)).

Equation Method Dep. var.	5a EB <i>b</i>	5b EB <i>b</i>	6a EB <i>b</i>	6b EB <i>b</i>	7a EB <i>b</i>	7b EB <i>b</i>	8 EB <i>b</i>	9 EB <i>b</i>
OBS	61 (- accid)	61 (-accid)	64 (- neg)	64 (-neg)	24 (TSP)	24 (TSP)	33 (PM ₁₀)	16 (BS)
CONSTANT	-0.0101	0.2070	-0.4408	-0.2518	0.7019	0.2659	-1.2173	-4.6017
(se)	(1.2239)	(1.1940)	(1.1066)	(1.0770)	(2.8333)	(2.9177)	(3.1341)	(3.1584)
	(-0.01)	(0.17)	(-0.40)	(-0.23)	(0.25)	(0.09)	(-0.39)	(-1.46)
POLL	-0.00090	-0.00149	-0.00114	-0.00162	0.00146	-0.000275	-0.00119	-0.00490'
(se)	(0.00131)	(0.00129)	(0.00120)	(0.00118)	(0.00459)	(0.00473)	(0.00357)	(0.00267)
	(-0.69)	(-1.16)	(-0.95)	(-1.38)	(0.32)	(-0.06)	(-0.33)	(-1.83)
OVER65	-0.02539	-0.00437	-0.01352	0.00865	0.00495	0.01425	-0.16550***	-0.08868
(se)	(0.02654)	(0.02766)	(0.02493)	(0.02612)	(0.05099)	(0.05487)	(0.04911)	(0.20868)
	(-0.96)	(-0.16)	(-0.54)	(0.33)	(0.10)	(0.26)	(-3.37)	(-0.43)
GNP	-0.0000559'	0.0000149	-0.0000395'	0.0000135	-0.000075	0.000052	0.000079***	0.000178
(se)	(0.0000317)	(0.0000129)	(0.0000287)	(0.0000117)	(0.000062)	(0.000046)	(0.000021)	(0.000116)
	(-1.76)	(1.15)	(-1.38)	(1.15)	(-1.21)	(1.14)	(3.75)	(1.54)
GINI		0.03044***		0.02539***		0.03857*	0.01531	-0.15899
(se)		(0.0081)		(0.00723)		(0.01787)	(0.01568)	(0.10366)
		(3.76)		(3.51)		(2.16)	(0.98)	(-1.53)
DIST	2.08e-06**		1.60e-06**		4.24e-06**			
(se)	(6.91e-07)		(6.25e-07)		(1.56e-06)			
	(3.00)		(2.56)		(2.72)			
DALE	0.01558	-0.01075	0.02333	0.00064	-0.00991	-0.02397	0.0393	0.17084
(se)	(0.02062)	(0.02172)	(0.01817)	(0.01911)	(0.03397)	(0.03906)	(0.05042)	(0.11149)
	(0.76)	(0.50)	(1.28)	(0.03)	(-0.29)	(-0.61)	(0.78)	(1.53)
EDUC	-0.00437	-0.000197	-0.00604	-0.00247	-0.00366	-0.000474	-0.00912	-0.02883
(se)	(0.00598)	(0.00585)	(0.00511)	(0.00499)	(0.0149)	(0.0156)	(0.00931)	(0.03062)
	(-0.73)	(-0.03)	(-1.18)	(-0.50)	(-0.25)	(-0.03)	(-0.98)	(-0.94)
Tau2	0.136***	0.127***	0.110***	0.101***	0.257**	0.278**	0.158**	2.10e-13
	(4.52)	(4.44)	(4.45)	(4.39)	(2.80)	(2.78)	(2.84)	(0.00)
Log like.	-67.41	-46.28	-59.21	-37.68	-78.23	-60.82	-56.92	-28.95
Wald chi2	20.84	26.57	21.16	27.52	11.95	9.09	29.86	64.83

'P<0.1, *P<0.05, **P<0.01, ***P<0.001, β and se have both been multiplied by 1000.

Table 5: Summary of regression results using EB-methodology (sample of lag 0 coefficients (28), lag 1 coefficients (23), and lag 2 coefficients (12)).

Equation Method Dep. var. OBS	10a EB <i>b</i> 28 (lag 0)	10b EB <i>b</i> 28 (lag 0)	11a EB <i>b</i> 23 (lag 1)	11b EB <i>b</i> 23 (lag 1)	12a EB <i>b</i> 12 (lag 2)	12b EB <i>b</i> 12 (lag 2)
CONSTANT (se)	-3.0395 [*] (1.5626) (-1.95)	-2.8248 [*] (1.2782) (-2.21)	-3.2315 (3.7424) (-0.86)	-3.0477 (3.7276) (-0.82)	6.8799 (5.8035) (1.19)	3.4584 (4.7874) (0.72)
POLL (se)	-0.00426 ^{**} (0.00159) (-2.69)	-0.00475 ^{***} (0.00136) (-3.50)	0.00011 (0.00392) (0.03)	0.00055 (0.00399) (0.14)	-0.00871 (0.00573) (-1.52)	-0.00795 (0.00533) (-1.49)
OVER65 (se)	-0.00072 (0.03982) (-0.02)	0.01920 (0.03066) (0.63)	0.00888 (0.07217) (0.12)	0.02318 (0.07606) (0.31)	0.00673 (0.18719) (0.04)	-0.04446 (0.16149) (-0.28)
GNP (se)	-0.000108 [*] (0.000045) (-2.38)	-1.63e-06 (0.000011)	1.64e-07 (0.000053) (0.00)	-8.55e-06 (0.000027) (-0.32)	-0.000199 (0.000153) (-1.31)	-0.000035 (0.000056) (-0.63)
GINI (se)		0.035780 ^{***} (0.00685) (5.23)		0.00545 (0.01677) (0.33)		0.0760 [*] (0.04355) (1.75)
DIST (se)	2.83e-06 ^{**} (9.05e-07) (3.13)		-1.64e-07 (1.21e-06) (-0.14)		5.23e-06 (3.19e-06) (1.64)	
DALE (se)	0.07652 ^{**} (0.02880) (2.66)	0.04667 [*] (0.02205) (2.12)	0.04342 (0.07168) (0.61)	0.03390 (0.07431) (0.46)	-0.10986 (0.10606) (-1.04)	-0.11412 (0.10456) (-1.09)
EDUC (se)	-0.01663 [*] (0.00737) (-2.26)	-0.01331 [*] (0.00565) (-2.36)	0.00784 (0.01033) (0.76)	0.00944 (0.01113) (0.85)	0.0220 (0.01724) (1.28)	0.04462 [*] (0.02219) (2.01)
Tau2	0.1625 (1.08)	9.42e-16 (0.00)	0.1558 ^{**} (2.58)	0.1580 ^{**} (2.59)	0.191 (1.29)	0.179 (1.28)
Log like.	-61.71	-37.71	-68.44	-49.34	-69.91	-50.62
Wald chi2	27.00	51.13	8.94	8.94	10.91	11.67

^{*}P<0.1, ^{*}P<0.05, ^{**}P<0.01, ^{***}P<0.001, β and se have both been multiplied by 1000.

Table 6: Summary of regression results using EB-methodology (sample consisting of one coefficient (averaged or actual) per country (21), and taking time-period into account (25)).

Equation Method Dep. var.	13a EB b	13b EB b	14a EB b	14b EB b
OBS	21 (country)	21 (country)	25 (country)	25 (country)
CONSTANT	-0.3806	-0.3244	-0.5841	-0.5168
(se)	(1.2077)	(1.2440)	(1.1836)	(1.1926)
	(-0.32)	(-0.26)	(-0.49)	(-0.43)
POLL	0.00129	0.00149	0.00065	0.00052
(se)	(0.00158)	(0.00156)	(0.00144)	(0.00145)
	(0.82)	(0.95)	(0.45)	(0.36)
OVER65	-0.0654*	-0.0598*	-0.0570*	-0.0518*
(se)	(0.0293)	(0.0335)	(0.02832)	0.0313)
	(-2.23)	(-1.79)	(-2.02)	(-1.65)
GNP	0.000072*	0.000059**	0.000028	0.000042**
(se)	(0.000033)	(0.000022)	(0.000033)	(0.000016)
	(2.18)	(2.64)	(0.85)	(2.62)
GINI		-0.000072		0.00583
(se)		(0.00860)		(0.00874)
		(-0.01)		(0.67)
DIST	-4.00e-07		4.15e-07	
(se)	(7.56e-07)		(7.50e-07)	
	(-0.53)		(0.55)	
DALE	0.03015	0.02779	0.03107	0.02508
(se)	(0.02062)	(0.02426)	(0.01988)	(0.02312)
	(1.46)	(1.09)	(1.56)	(1.09)
EDUC	-0.01579**	-0.01549*	-0.01191*	-0.01076*
(se)	(0.00601)	(0.00641)	(0.00585)	(0.00618)
	(-2.62)	(-2.42)	(-2.04)	(-1.74)
Tau2	0.0666*	0.0698*	0.0667**	0.0662**
	(2.37)	(2.47)	(2.75)	(2.75)
Log like.	-59.52	-41.00	-56.47	-37.66
Wald chi2	14.66	14.02	14.33	14.51

5.3 Discussion

Given the sensitivity of the findings to the sample choice and the measures used, interpretation is a challenging task. In this section we will first discuss the general findings from regressions carried out on the full sample (sample size 70), the sample excluding the 9 all-cause mortality studies (sample size 61), as well as on the sample consisting of coefficients derived using the TSP measure of particles (sample size 24). We will then compare and contrast these results with those obtained with the samples stratified according to the particle measure and the lag structure, as well as with those that include only one observation (an actual observation or an averaged one) for each country.

Among the moderator variables tested, GINI and the interaction term DIST are the strongest and most stable predictors of the dose-response relationship. The positive coefficients indicate that higher income inequality in general, as measured by the GINI-coefficient, and in high-income countries in particular, as captured by the interaction term DIST, are associated with higher mortality rates from air particles.

The GINI-model yielded a positive coefficient for the GNP term, however, it is only significant in the full sample. In the case of the DIST-model, the GNP term takes a negative coefficient that is weakly significant only in the case of the sample excluding the all-cause mortality studies. This main tendency suggests that the negative direct effect of income is smaller than the indirect increasing effect via its interaction with income inequality. In other words, the effect income has in increasing the positive effect of the inequality in income distribution on the dose-response coefficient is larger than the negative (decreasing) effect of average income on the PM_{10} -mortality association.

In section 3 we mentioned four possible reasons for income inequality to have an increasing effect on the mortality rate from air pollution. First, decreasing returns to averting and mitigating activities would imply a tendency for higher inequality to be associated with higher mortality rates from changes in air pollution levels. Second, if individuals with relatively low income live in the most polluted areas, and if the adverse effect of air pollution is larger on lower income individuals, then this would once again imply larger PM_{10} -mortality in cities with large income inequality. Third, if mortality is associated with stress, and stress is related to social status, then higher income inequality may lead to a larger mortality rate from air pollution. Finally, GINI may have been proxying for inequalities in baseline health or for the quality and availability of health care. We did include health services as a moderator variable in one of our regression models, and found a slight decrease in the significance for both GINI and DIST, however,

both remained significant. Since our measure of health services was relatively poor, however, this does not necessarily imply that GINI is not proxying for some aspect of health services.

Our findings provide empirical support for the idea that income inequality has an increasing effect on the mortality rate from air pollution. However, with the level of aggregation of our data there is little we can do in terms of choosing among the above explanations.

One implication of the regression results for income level and inequality (as captured in the variable DIST) is rather surprising: inequality in income has a larger positive effect on the PM₁₀-mortality association in high-income countries. At the risk of 'over-interpreting', two explanations may be given for this phenomenon. First of all, there may be more mitigating and averting activities available in high-income countries to the ones who can afford it. The idea is that the supply side of health investments is more developed in high-income countries, e.g. the existence of low pollution neighbourhoods, well-isolated houses, air filtering systems, medicines, medical advice, high-quality medical procedures. In addition, and related to this, comes the fact that the awareness of the general population, irrespective of income level, about the health risks from air pollution and how to minimise them probably is higher in high-income countries. The second possible explanation for the positive sign of the GNP and GINI interaction term is that high income inequality leads to more psychological stress in a high-income country than in a country with a lower level of average income, or alternatively that the stress induced by inequality is more fatal. First, the higher level of stress may be related to various media incessantly informing and reminding people of the differences. Second, different life-styles (diet, exercise etc.) may mean that stress has a more fatal outcome in high-income countries. Furthermore, high-income countries also tend to have a higher degree of social mobility, something which possibly affects the stress-level experienced by 'under-achievers'. Finally, there is possibly a stronger focus on material status in high-income countries.

Another slightly surprising finding is the very small and insignificant direct negative effect of income on the dose-response coefficient. It is possible that income does not affect behaviour (averting and mitigating activities) to such an extent that it shows up very significantly in the regression results. Income level is likely to affect the level of baseline health and education, however these are measured by other moderator variables. A very likely, and perhaps more plausible explanation, however, of the lack of significance of the coefficient for the direct effect of income, is that it is highly correlated

with education, disability-adjusted life expectancy, the percentage of the population over 65 years of age, and the ambient level of pollution (see table 2). As long as the measures used are not perfect, income may be picking up parts of these effects, some of which work in opposing directions. Alternatively, the other measures may have captured part of the effect that should have been captured by income.

We should also remind the reader that the income variable we ideally would like to have is the average income in the location in question (and preferably of the population particularly vulnerable to air pollution). Since we were not able to obtain this information, we had to use the average income in the relevant country instead. Not only may this imply that our income measure is fairly poor, but in addition it may imply that the GINI coefficient partly proxies for the city income-level.

As for the additional moderator variables POLL, OVER65, EDUC, DALE, and the constant, none was significant in any of the three samples mentioned. The education term was the most consistent of these variables, taking a negative coefficient in all of the samples. The sign suggests that the amount of knowledge or education people have may play a role in avoiding fatal effects from air pollution.

The effect of the average ambient level of pollution (POLL) on the PM₁₀-mortality coefficient varies in sign and is insignificant. This finding suggests that non-linearities in the effect of particles on mortality are not very significant. The positive but insignificant coefficient for ambient PM₁₀ concentration found in Levy et al. (2000) is in line with our finding.

Similarly, the coefficient for baseline health (DALE) varies in sign from one sample and regression model to another, but it is consistently insignificant. Again, this *could* indicate that basic health level does not affect significantly the rate at which people die from air pollution. In principle, it is possible that a healthy person could die suddenly from high exposure to air pollution, just as well as an older person or a person suffering from some illness, however, this does not make sense intuitively. The insignificance of the coefficient is most likely due to DALE not being a satisfactory measure of baseline health.

The coefficient for percentage of the population over 65 years of age also varies in sign according to the sample we are analysing, but is consistently insignificant. Only when we excluded any measure of income inequality from our regression equation did OVER65 become significantly negative, something which suggests that it was proxying for income inequality (from table 3 we observe that the correlation is -0.40). Levy et al. (2000) found a negative and weakly significant coefficient for population over 65 years

of age (per cent) in their regression analysis on a sample of 19 U.S. studies. The difference in results between the two studies may possibly be explained by the fact that this latter study did not include income inequality or baseline health as moderator variables in their regressions.

In section 4 we argued that we would expect elderly people to be most at risk from the acute effects of air pollution, and therefore a positive coefficient for this moderator variable. So how do we explain these findings? We suggest that since baseline health is such a multi-faceted concept, no single moderator variable will adequately be able to measure the desired dimension. It is therefore possible that, although we have included DALE as a measure of baseline health in our regression model, OVER65 is still picking up part of the effect of higher baseline health on the PM₁₀-mortality association. The reason for this is that individuals tend to survive to a high age if they are in good health, so a society with a large proportion of elderly people may also be a “healthy” society. This will then induce a downward bias in the estimator, which may have cancelled out the expected positive coefficient for OVER65.

The inclusion of a number of moderator variables is required in order to distinguish among potential predictors. However, the lack of independence can pose problems. The findings discussed above of insignificant coefficients, with varying signs, for DALE, OVER65, and POLL may be a function of these correlations. In addition, these moderator variables may not be measuring the desired dimensions or may not represent the actual characteristics of the site. Both DALE and OVER65 are based on country data, rather than data from the site in question. In addition, DALE exists for one year only, and may therefore be very misleading especially in the case of older studies. Although pollution data are available for the particular city we are studying, potentially significant information e.g. on pollutant mixture and particle size was missing.

Interestingly, we find that the estimate of the central effect of PM₁₀ on mortality is larger when studies of all-cause mortality are included rather than excluded from the sample. As the three types of particulate pollution measures are all represented in the 9 studies investigating all-cause mortality, the type of particulate measure used is unlikely to have caused this difference in results. Although the 9 studies may differ from the rest of the sample in some other unidentified way, it is also possible that externally caused mortality is not entirely independent of air pollution. In particular, we suggest that air pollution may affect individuals' level of concentration, stress, as well as visibility and that therefore the amount of externally induced mortality incidences increases with higher levels of air

pollution. Furthermore, air pollution will tend to be correlated with the amount of traffic, and thereby possibly with traffic-accidents.

By stratifying the sample according to the particle measure, we observed that the particle measure used affected the findings on the PM_{10} -mortality association. Although erroneous conversion factors can explain the differences in central effect estimates according to the pollution measure used, they cannot explain the extent to which the estimated moderator-effects and their significance differ. The fact that most of the moderators are country based means that regressing the observed dose-response coefficients on these variables only makes sense when a variety of countries are included in the sample. Since half of the selected studies using BS to measure air pollution are from Spain and half of those using PM_{10} have been carried out in the US, we argued that the results from these two samples would not be meaningful. For the record, it is worth mentioning that even with samples where such a moderator-effect analysis is meaningful, differences in moderator effects could nevertheless be expected. The studies using BS are likely to differ from those employing PM_{10} in several important respects. First of all, BS is a measure based on older technology and is therefore typically used in earlier air pollution studies. Second, PM_{10} became a commonly used measure of air particles in the US before most other countries, and there is therefore a disproportionate amount of US studies in our sample that use this measure. US studies, as well as newer studies may differ in several respects from older studies and studies carried out in other countries (e.g. in terms of pollutant mixtures, and population characteristics), and this could well be reflected in the moderator coefficients.

Two of the sensitivity tests undertaken in this paper stand out from the rest because of the very different results from those discussed above; the test for sensitivity to the lag structure, and that for the sensitivity to multiple observations for some countries. We will discuss the main findings from these analyses in turn and consider whether they are seriously challenging the results from the rest of the regression analysis.

The results from regressing the dose-response coefficients on the moderator variables varied widely according to the time lag of the pollution measurement used in the original study. For the association between same-day air pollution and mortality we found that the GNP coefficient is significantly negative and the DIST-coefficient significantly positive. When GINI is included instead of DIST, GNP retains the negative sign but is insignificant. These results are in line with our previous findings. However, for air pollution lagged one day there is no significant effect from income, income inequality, or their interaction term. In the case of a two-day lag, the effects are similar to

the same-day effects for the income-related variables but the significance levels are lower, and only GINI is weakly significant.

The widely varying results according to the lag structure are most likely due to the small sample sizes. The sample for lag zero consists of 28 observations, for lag one 23, and in the case of lag two, 12. One has to be extremely cautious with results based on small sample sizes, and in particular in the case of the smaller samples, i.e. lag one and two, the size of the samples is likely to drive the results. The smaller the size, the wider the confidence interval, reflecting an amount of uncertainty about the parameter being estimated, which becomes undesirable at some point. Everything else equal, the results from the analysis upon the largest sample are more likely to have some meaning, although caution is still required. These results were nevertheless found to be in line with our previous findings.

If we have included the main explanatory variables in our regression model, then the representativeness of the sample would not have been an issue. The reason for this is that the requirement for a sample to be representative is based on the idea that there may be underlying factors not known or fully understood by the researcher that influence the relationship under investigation. If, however, the moderators cannot adequately account for the discrepancies between study findings, then the fact that the sample may be unrepresentative constitutes a challenge. How serious this challenge is depends to some extent on what we would like to do with our findings. In particular, the challenge will be present if we would like to generalise our results in order to tailor-make dose-response coefficients to different circumstances. If, on the other hand, the purpose of the meta-analysis is first and foremost the wish to investigate *whether* various variables are significantly affecting the PM₁₀-mortality ratio, rather than the actual size of the effect, the unrepresentativeness of the sample may not be such a problem.

It is, however, not straightforward to determine what would have been a representative sample – ‘the concept of representativeness has always been a somewhat ambiguous idea, but the concept is useful in helping to illuminate potential problems in drawing inferences from samples’ (Cooper and Hedges (1994), p. 35). Like Levy et al. (2000), we assume that the studies are independent samples from a random distribution of the conceivable population of studies. The latter being all the potential studies that could have been carried out given our inclusion and exclusion criteria. This assumption is clearly difficult to support, mainly due to the overweighing of certain countries and cities, but also due to the sample entailing multiple studies conducted by the same author(s) and using the same methodology. On a more general note, there are two main reasons why

exhaustiveness of sampling does not necessarily yield a representative sample of the universe. First, some types of studies in the intended universe may not have been conducted (e.g. because of unreliable data), and second, although studies may have been conducted, they may not have been reported in journals or other forums accessible to the researcher.

To determine whether the results were sensitive to study selection, and in particular to the inclusion of multiple observations from certain countries, we therefore conducted analyses where each country entered with only one observation, either an averaged one or from one single study (randomly chosen). Averaging the data over several studies, and using the average as if it were an observation is a very dubious method, and is likely to create correlations between variables where these do not exist. These problems are worsened in the case where the averaged data came from studies covering very different time periods. Including single studies is therefore a preferred method when testing the sensitivity to multiple country observations. The main objections to this latter method are, first, that the sample becomes smaller, second, that we are ignoring/rejecting information that we actually have, third, that the observation that happen to be included may be unrepresentative for the country in question, and finally, the resulting sample may not necessarily be more representative than the full sample. Compared to the results from the full study, this country-weighted sensitivity analysis found a more significant effect of education in decreasing the particle-mortality effect. Furthermore, no significant role was found for either the GINI-coefficient or its interaction term with GNP. The role of income appears relatively unchanged, with a positive and significant effect in the GINI-model, and no significant effect in the DIST-model. These findings are similar to those obtained on the sample where data for the same country, of the same time-period, were averaged.

When comparing the result using the full sample with those using the ‘one-country-one-observation’ sample, the differences in results are of degree rather than of tendency/direction. The coefficient for education is consistently negative, for income inequality it is consistently positive, and as for income, it tends to have a direct effect which is either negative or zero, and an indirect effect via income inequality which is positive. Significance varies between the samples.

We have seen that there are weaknesses related to both samples. We nevertheless propose that the results based on the full sample are the preferred ones, since the sample with one observation for each country excluded existing information without necessarily

becoming more representative of the universe of potential studies (given our selection criteria).

Since the inter-study variability remained significant, it is quite likely that we omitted relevant moderators that could have explained at least part of it. It is also possible that omitted relevant moderators can explain the significance of some of the associations we found, e.g. of income inequality. Possible omitted relevant explanatory variables are smoking habits, the ratio of fine to coarse particles, other pollutants, central air conditioning prevalence, and cooking and heating mechanisms used. Furthermore, the data used to capture the moderator variables may have been poorly suited for that purpose. Most of the data were given on a country-wide level, whereas the studies were carried out for a specific region or city within that country which may well differ significantly from the country-average with respect to that particular characteristics. In addition, some of the moderator concepts were too complicated and vague to be captured by one type of indicators, e.g. health services.

Even if the above weaknesses could be solved, the problem of selection bias remains. Although we included all the available studies that fulfilled the selection criteria set out in section 4, there is no way of avoiding possible “publication-bias” or “submission-bias” (i.e. studies that do not find a significant relationship between PM_{10} and mortality, or that find a negative association, are not published or submitted for publication). Nevertheless, the file-drawer bias is highly unlikely to change the finding of a significant PM_{10} -mortality link. With the assembled sample (70) of largely positive estimates, 70 unpublished estimates with central estimates of -0.5 per cent and the mean variance of our 70 study estimates would be required to yield a statistically insignificant grand mean estimate. A further source of selection bias lies in the representativeness of our study locations. As argued earlier, it is possible that the cities in our analysis are not representative of the true range of conceivable studies. As argued by Levy et al. (2000), it is possible that a subset of cities “that were more or less prone to PM health effects were chosen for epidemiologic analyses” (p.115). The main problem with this challenge to our sample is to know the range of conceivable studies. Nevertheless, the cities and countries included in our sample represent a wide range of population characteristics and pollution concentration levels.

One particular and potentially important weakness of the original studies may have influenced the various findings from our meta-analysis. As briefly mentioned towards the end of section 4.1, measuring PM_{10} exposure with readings of ambient pollution from central monitoring stations may be highly problematic, especially in the

case of developing countries. If indoor air pollution in developing countries, caused by the use of highly polluting heating and cooking fuels and little ventilation, is a larger health risk than outdoor air pollution, then the exposure-response association may be much larger but not be revealed by studies using exposure measurements obtained from central monitoring stations. The individuals vulnerable to outdoor air pollution may already have died from indoor air pollution, and the coefficient will not capture these deaths adequately as long as indoor and outdoor air pollution do not strongly covary. A possible implication of this measurement error is that those moderator variables that tend to take a significantly different value in the case of developing than developed countries, i.e. income per capita, education, and disability-adjusted life expectancy may have lost significance.

A number of more general weaknesses and limitations to our meta-analysis are necessary to point out. First of all, it is important to note that as the meta-analysis uses a correlational design for its moderator variables, no causal inference can appropriately be made. Secondly, when the moderator variables cannot adequately account for the discrepancies between study findings, as is the case for our full sample, it becomes unclear whether the various research operations represent a common underlying construct, and uncertainty in the empirical effects of the moderators emerges. Furthermore, unresolved inconsistencies between studies may compromise the ability to detect true relations among variables. 'In such cases' states Cooper and Hedges (1994) 'meta-analytic findings should be accorded relatively low certainty' (p.490). Thirdly, even if the moderator variables successfully account for the empirical variability in study outcomes, the proper interpretation of the relation may remain an issue, especially when the moderators are identified on a between-study basis. The moderator is then an attribute of an entire study, and individuals in the primary research have not been randomly assigned to levels of the moderator variable. Such moderator variables may be confounded with other variables that covary with it.

Our first study-objective was to increase our understanding of what affects the amount of deaths that are related to air pollution. From our regression results we have seen that several of the population characteristics included as moderator variables may play a significant role in the association between particle pollution and mortality. In particular, income inequality may be associated with a larger effect of a change in pollution on the mortality rate, especially in high-income countries. The direct effect of income may be slightly decreasing the effect of pollution on mortality, and a similar finding was made for education.

Second study-objective was to help highlight areas where further studies may be needed. Indeed, we have pointed out that the discrepancies between study effects remain significant, that some of the data are not very well-suited to capture the moderator effects in question, that no causal inferences can appropriately be made for the moderator variables, that the sample of studies may not be representative of the hypothetical universe of studies, and that there may be serious measurement errors in the original studies. Subsequent research will need to address all of these concerns. The first point can be addressed by adding other possible moderator variables that have been discussed in the literature (e.g. the ratio of fine to coarse particles, other pollutants, gas stove prevalence, central air conditioning prevalence, warm air heating prevalence, smoking habits) to the ones discussed in the present paper. This may not only eliminate the discrepancies between study-effects, but may also alter the findings on the already included moderators. The second point can be approached by for example trying to obtain city- or location-specific information on the relevant moderator variables, and by more generally encouraging the primary studies to gather and publish this information. Meta-analysts should also be on the lookout for improved indicators of the moderator effects. In order to be able to make causal inferences, the third point, two criteria in addition to covariance have to be fulfilled; temporal ordering (i.e. that the cause should temporally precede the effect) and isolation (that the effect was not caused by something else). This suggests that future PM₁₀-mortality research could be directed towards panel-studies gathering individualised time-series data on the relevant moderator variables. As for the latter two points, future primary research would benefit from being directed towards the understudied populations; in particular, more studies from poor countries are required, and would also benefit from improved exposure measurements; in particular, including indoor air pollution exposure.

Final study-objective was, through the ensuing coefficients of the moderator variables, to help transferring the dose-response coefficients to countries where empirical studies have not yet been feasible or to forecast the effects of policies targeting air pollution. The coefficients derived from our meta-analysis could in theory help us tailor-make dose-response coefficients to local conditions. Indeed, for an example of how this can be done refer to Maddison and Gaarder (2001). However, as long as the between-study variation remains significant, we suggest proceeding with caution. Rather than using the exact coefficients derived in our meta-analysis, and which may change significantly once the whole variation has been explained by additional moderators, we propose using the findings to suggest relative differences. If we for example have estimated the dose-response

coefficient for one city, and would like to transfer it to another city that only differs in that it has a higher income inequality, we suggest that the empirical coefficient at hand probably will constitute a lower bound of the actual effect. If only the education level between two cities differed, we could again propose that the dose-response coefficient would probably be higher in the city with lower levels of education. A further note of caution is however in order. For any city or location with a value for one of the moderator variables outside the range covered in the meta-analysis, transfer will strictly speaking not be valid.

VI. Conclusion

In this paper we have applied the Empirical Bayes mixed-effect model to the largest sample of air-pollution mortality studies to date, from the widest range of countries, in order to determine whether the variability in effect estimates can be explained by a selected group of population characteristics. Theoretical plausibility, novelty, and availability of data guided the selection of study-characteristics included as potential moderator variables.

We estimated that a 100- $\mu\text{g}/\text{m}^3$ increase in PM_{10} concentrations was associated with a 6 per cent increase in daily mortality, which was in line with previous findings, although slightly lower. When including the moderator variables in our analysis we found that higher income inequality in general, as measured by the GINI-coefficient, and in high-income countries in particular, is associated with higher mortality rates from air particles. For our full sample, a four-point increase in the GINI-coefficient leads to an increase in the mortality rate from a 100- $\mu\text{g}/\text{m}^3$ increase in PM_{10} of almost 1 per cent (i.e. from 6 to 7 per cent). We further found some evidence that the PM_{10} -mortality relationship may be stronger in locations with lower education. Furthermore, there appears to be a positive (increasing) effect of income on the association between income inequality and the dose-response relationship and a negative direct effect of GNP per capita on the dose-response coefficient, with the former outweighing the latter. Our findings were quite sensitive to the samples and measures analysed in our sensitivity analyses.

Although our interpretations of these associations are highly tentative, we nevertheless find it useful to summarise potential explanations for the above findings. In addition to income inequality possibly proxying for the quality and availability of health services, or health inequalities, larger income inequalities may be associated with stress, and stress may in turn affect mortality from air pollution. Furthermore, decreasing returns to averting and mitigating activities would imply a tendency for higher inequality to be associated with higher mortality rates. The fact that income inequality increases the effect

of pollution on mortality more in high-income countries may, firstly, be due to higher availability of mitigating and averting activities in high-income countries to the ones who can afford it, and secondly to inequality creating relatively more psychological stress in high-income countries. The latter could plausibly be due to the role of media, higher social mobility, and stronger focus on material status in higher income countries. Alternatively, the stress induced by inequality may be more fatal in high-income countries due to different life-styles. Finally, it is not unlikely that the income level of the relevant population does not correspond to the per capita income level of the whole country – the combination of income and income inequality may then together proxy for this variable.

We argue that the level of education affects the knowledge people have about health, health production, and the connection between air pollution and health, and hence the amount of averting and mitigating expenditures undertaken and the efficiency of these expenditures. As for the generally small (and mostly insignificant) negative direct effect of income on the dose-response association, we mention the possibility that income does not affect behaviour to such an extent that it shows up very significantly in the regression results. Income level is likely to affect the level of baseline health and education, however these are measured by other moderator variables.

Although baseline health, the percentage of people over 65 years of age, and the level of ambient particle concentration were not found to play significant roles, this may be due to the quality and level of aggregation of available data, as well as the correlation between several of the moderator variables. Note also that even though we have invoked a number of moderator variables, and some of them are significant, substantial unexplained variation in the coefficients remains. This suggests that aspects of the relationship between air pollution and mortality remain unexplained or that they are due to any of a number of variables that we have mentioned but for which we did not have adequate data. Our findings on the included moderator variables must be qualified by these considerations.

We have also argued that several of the moderator variables (in particular income, education, and baseline health) may have lost significance due to measurement error in the original studies. Readings of ambient pollution from central monitoring stations may be particularly unsuited as measurements for PM_{10} exposure in developing countries. The individuals potentially vulnerable to outdoor air pollution may already have died from indoor air pollution, and the coefficient will not capture these deaths as long as indoor and outdoor air pollution do not covary.

Interestingly, we find a slight tendency for the effect of pollution to be larger in studies looking at all-cause mortality than those excluding deaths from ‘external’ causes, potentially implying that externally caused mortality is not entirely independent of air pollution. Furthermore, we find the estimated moderator effects to differ depending on the lag-structure, however this is most likely due to the small sample sizes.

The paper has introduced a variety of population characteristics not previously investigated, and uncovered that a number of these have a measurable influence on the magnitude of the PM-mortality relationship. The ensuing coefficients can in the first instance be used to suggest whether a dose-response coefficient transferred from one country to another is likely to under- or overestimate the mortality caused by ambient particles. When additional moderator variables are included that successfully explain the between-study variability, the ensuing coefficients can be used to tailor-make dose-response coefficients to local conditions. Subsequent EB analysis may benefit from adding other possible moderator variables to the ones discussed in the present paper. In addition, it should focus on obtaining more location-specific information on the relevant moderator variables and improved indicators of the moderator effects. Future meta-analyses would benefit from primary research being directed towards understudied areas and populations, such as in developing countries and rural areas, and from improving exposure measurements (e.g. increase the number of monitoring stations, include indoor air pollution exposure). We also recommend future PM₁₀-mortality studies to explore the possible causal inferences to be made. For these purposes, the primary research will need to be directed towards panel-studies gathering individualised time-series data on the relevant moderator variables.

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Appendix A: Relating the Present Study to the Maddison and Gaarder (2001) Study.

Dr. David Maddison, Senior Research Fellow at the Centre for Social and Economic Research on the Global Environment (CSERGE), took the initiative for undertaking a meta-analysis of air pollution mortality studies. In particular, his idea was to include moderator variables that potentially influence the slope of the dose-response function. The prime purpose of the paper was to quantify and value the health impacts of air pollution in densely populated cities of the developing world.

In the Maddison and Gaarder (MG) study we analysed a smaller sample (13 observations) than in the present paper (70 observations), using slightly different selection rules for the sample. In the present paper I furthermore argue that one of the observations in that sample should have been excluded. As for the selection of moderator variables, Dr. Maddison suggested including the level of ambient particle concentration and the percentage of the population over 65 years of age, whereas I suggested including income and income inequality. In the present paper, I additionally include baseline health, education, and health services as moderator variables in my analysis, and update all of the data used to capture the moderator variables. In the MG-study we used the method of variance-weighted least squares (VWLS), thereby implicitly assuming that all the variance among the study effects other than sampling variance can be explained as a function of the study characteristics we chose to include. In the present study I argue that that assumption is unrealistic, and propose to use Empirical Bayes method to take into account unexplained inter-study variability.

I show in this paper how the data, methodology, and sample corrections significantly change the findings from those presented in the MG-paper. Then I proceed by analysing the larger sample gathered specifically for the current paper, and discussing the findings.

In addition to the above differences, the present paper reviews the existing meta-analysis literature, carries out a thorough sensitivity-analysis, and discusses in detail the weaknesses attached to such a study.

Appendix B: Mathematical Derivation of the Equations in Section 4

This appendix is following closely the derivation in Raudenbush and Bryk (1985).

The maximum likelihood estimators \mathbf{d}_i and \mathbf{g}_i with known (or estimated) v_i and \mathbf{t}_i can be derived by the maximum likelihood method:

$$\begin{aligned} L(\mathbf{d}, \mathbf{g}; d) &= \prod_i^k f_i(d_i | \mathbf{d}_i) g_i(\mathbf{d}_i | W_i, \mathbf{g}) \\ &= \left[\prod_i^k (2pv_i) \right]^{1/2} \exp\left\{-\frac{1}{2} \sum (d_i - \mathbf{d}_i)^2 / v_i\right\} \times (2\mathbf{p}\mathbf{t}^2)^{-k/2} \exp\left\{-\frac{1}{2} \sum (d_i - W_i' \mathbf{g})^2 / \mathbf{t}^2\right\} \end{aligned}$$

Taking the derivatives of log L with respect to \mathbf{d}_i and setting it equal to 0 yields:

$$(d_i - \mathbf{d}_i) / v_i = (\mathbf{d}_i - W_i' \mathbf{g}) / \mathbf{t}^2$$

Solving this equation for \mathbf{d}_i we obtain

$$\mathbf{d}_i = [\mathbf{t}^2 / (v_i + \mathbf{t}^2)] d_i + [v_i / (v_i + \mathbf{t}^2)] W_i' \mathbf{g} = \mathbf{I}_i d_i + (1 - \mathbf{I}_i) W_i' \mathbf{g}$$

Setting $\partial \text{Log L} / \partial \mathbf{g} = 0$ yields

$$\sum \mathbf{d}_i W_i' = \sum W_i' \mathbf{g} W_i' = \mathbf{g}' \sum W_i W_i'$$

If we transpose both sides and substitute for \mathbf{d}_i from equation (FC) we obtain the following expression for \mathbf{g} :

$$\mathbf{g} = \left(\sum \mathbf{I}_i W_i W_i' \right)^{-1} \sum \mathbf{I}_i W_i' d_i$$

The approximate likelihood of the d_i is given by the marginal density of the vector $\mathbf{d} = (d_1, d_2, \dots, d_k)$:

$$\begin{aligned} L(\mathbf{g}, \mathbf{t}^2; d) &= \left\{ \prod_i^k [2p(v_i + \mathbf{t}^2)]^{-1/2} \right\} \exp\left\{-\frac{1}{2} \sum (v_i + \mathbf{t}^2)^{-1} (d_i - W_i' \mathbf{g})^2\right\} \\ &= \left\{ \prod_i^k [2p(v_i + \mathbf{t}^2)]^{-1/2} \right\} \exp\left\{-\frac{1}{2} Q\right\} \end{aligned}$$

where

$$\begin{aligned} Q &= \sum (v_i + \mathbf{t}^2)^{-1} (d_i - W_i' \mathbf{g})^2 = \sum (v_i + \mathbf{t}^2)^{-1} (d_i - W_i' \mathbf{g}^*)^2 + (\mathbf{g}^* - \mathbf{g})' \sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' (\mathbf{g}^* - \mathbf{g}) \\ &= Q_1 + Q_2 \end{aligned}$$

Under the distributional assumption of equation (10) we find that \mathbf{g}^* is multivariate normal

with mean vector γ and variance $\left[\sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' \right]^{-1}$.

The density of \mathbf{g}^* is hence;

$$f(\mathbf{g}^*; \mathbf{t}^2, \mathbf{g}) = (2\mathbf{p})^{-q/2} \left| \sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' \right|^{1/2} \exp\{-\frac{1}{2} Q_2\}$$

The density of d conditional on \mathbf{g}^* can now be expressed as

$$\begin{aligned} g(d|\mathbf{g}^*; \mathbf{t}^2) &= \frac{L(\mathbf{g}, \mathbf{t}^2; d)}{f(\mathbf{g}^*; \mathbf{t}^2, \mathbf{g})} = \frac{\left\{ \prod_i^k [2\mathbf{p}(v_i + \mathbf{t}^2)]^{-1/2} \right\} \exp\{-\frac{1}{2}(Q_1 + Q_2)\}}{(2\mathbf{p})^{-q/2} \left| \sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' \right|^{1/2} \exp\{-\frac{1}{2} Q_2\}} \\ &= (2\mathbf{p})^{-1/2(k-q)} \prod_i^k (v_i + \mathbf{t}^2)^{-1/2} \left| \sum (v_i + \mathbf{t}^2)^{-1} W_i W_i' \right|^{-1/2} \exp\{-\frac{1}{2} Q_1\} \end{aligned}$$

It follows from this expression that the log of the likelihood is proportional to expression (10).

Appendix C: Do-file in STATA to Maximise the Likelihood Function

The main difference between the two methods presented below is that method lf requires that the likelihood function meets the linear-form restrictions, whereas method d0 does not. Since our likelihood function does meet the linear-form restrictions, we can use either method. Note that the specifications written in italics were altered according to the exact model we wanted to run, and that the line-numbering does not belong to the do-file but was added in order to simplify this discussion. The program name in lines 1 and 14 (1 and 10 in method lf) must be changed whenever a change is made to the do-file. In addition, the variables entered into lines 8 and 14 of the do-file may be altered (lines 5 and 10 in method lf), as well as the sample size specified in line 11 (line 8 in method lf). The parenthesis at the end of line 14 (line 10 for method lf) indicates that if we want to restrict our sample, this can be done by adding 'if' and then the relevant restriction (the parenthesis is there to indicate that this is a choice feature – however, if the feature is used the parenthesis should be removed). Note that when restrictions are made on the sample, the sample size in line 11 (line 8 in method lf) must be adjusted to reflect this. Finally, it is optional to add 'difficult' to the maximize-command in line 15 (line 11 in method lf).

Method d0:

1. program define *d0prog*
2. version 6
3. args todo b lnf
4. tempvar t1 t2 detx lndetx
5. mlevel `t1' = `b'
6. mlevel `t2' = `b', eq(2)
7. quietly {
8. matrix accum X = *poll over65 gnp dist educ dale* [iweight=(se^2+`t2')^(-1)]
9. gen double `detx' = det(X)
10. gen double `lndetx' = ln(`detx')
11. mlsum `lnf' = -ln(se^2+`t2')-`lndetx'/70-((se^2+`t2')^(-1))*(\$ML_y1-`t1')^2
12. }
13. end
14. ml model d0 *d0prog* (*regco = poll over65 gnp dist educ dale*) / tau2 (if restriction)
15. ml maximize(, difficult)

Method lf:

1. program define *lfprog*
2. version 6
3. args lnf t1 t2
4. tempvar detx lndetx
5. matrix accum X = *poll over65 gnp dist educ dale* [iweight=(se^2+`t2')^(-1)]
6. quietly gen double `detx' = det(X)
7. quietly gen double `lndetx' = ln(`detx')
8. quietly replace `lnf' = -ln(se^2+`t2')-`lndetx'/70-((se^2+`t2')^(-1)*(\$ML_y1-`t1')^2)
9. end
10. ml model lf *lfprog* (*regco* = *poll over65 gnp dist educ dale*) / tau2 (if *restriction*)
11. ml maximize(, difficult)

Appendix D: Data

Table D1: Data based on information given in studies

OBS.	REFERENCE	PUBL. YEAR	COUNTRY	CITY	PERIOD	REGCO (Regression coefficient) ¹	SE (Standard error) ²	Change in daily mortality(%)/ 10mg/m ³ increase in PM ₁₀ ³	POLL (average PM ₁₀ level for period) ⁴
1	Touloumi et al.	1996	Greece	Athens	1987-1991	0.000480	0.000110	0.48	84.4
2	Sunyer et al.	1996	Spain	Barcelona	1985-1991	0.000677	0.000198	0.68	42.4
3	Bacharova et al.	1996	Slovak Rep.	Bratislava	1987-1991	0.000022	0.000364	0.02	49.2
4	Wojtyniak et al.	1996	Poland	Cracow	1977-1989	0.000173	0.000083	0.17	73.3
5	Wojtyniak et al.	1996	Poland	Lodz	1977-1990	0.000213	0.000085	0.21	57.3
6	Wojtyniak et al.	1996	Poland	Poznan	1983-1990	0.000175	0.000159	0.18	34.0
7	Wojtyniak et al.	1996	Poland	Wroclaw	1979-1989	0.000075	0.000129	0.08	54.3
8	Anderson et al.	1996	UK	London	1987-1992	0.001204	0.000315	1.21	14.6
9	Spix and Wichmann	1996	Germany	Koeln	1975-1985	0.000267	0.000239	0.27	37.4
10	Zmirou et al.	1996	France	Lyon	1985-1990	0.000258	0.000525	0.26	29.3
11	Cropper et al.	1997	India	New Delhi	1991-1994	0.000413	0.000182	0.41	207.9
12	Ostro et al.	1995	Chile	Santiago	1989-1991	0.000750	0.000130	0.75	115.4
13	Perez et al.	1999	Spain	Cartagena	1992-1996	-0.000164	0.000093	-0.16	31.6
14	Contin et al.	1999	Spain	Bilbao	1992-1996	0.001503	0.000488	1.51	43.1
15	Burillo et al.	1999	Spain	Valencia	1994-1996	0.001262	0.000494	1.27	44.2
16	Labaca et al.	1999	Spain	Madrid	1992-1995	-0.000441	0.000225	-0.44	37.8
17	Trunk et al.	1999	Spain	Vigo	1991-1994	0.000349	0.000330	0.35	98.1
18	Daponte-Codina et al.	1999	Spain	Huelva	1993-1996	0.002460	0.001361	2.49	42.5
19	Martinez et al.	1999	Spain	Gijon	1993-1996	0.001088	0.000696	1.09	45.6
20	Martinez et al.	1999	Spain	Oviedo	1993-1997	-0.001149	0.000961	-1.14	43.5
21	Boillos et al.	1999	Spain	Vitoria-Gasteiz	1990-1994	0.000628	0.000487	0.63	51.2
22	Blasco et al.	1999	Spain	Castellon	1991-1995	0.001499	0.001020	1.51	24.6
23	Ontoso et al.	1999	Spain	Pamplona	1991-1995	0.002941	0.002470	2.98	21.7
24	Ocana-Riola et al.	1999	Spain	Sevilla	1992-1996	-0.002013	0.000650	-1.99	45.1

25	Ballester et al.	1996	Spain	Valencia	1991-1993	0.000895	0.000310	0.90	67.7
26	Schwartz	1991	USA	Cincinnati	1977-1982	0.001059	0.000305	1.07	41.8
27	Kinney et al.	1995	USA	LA	1985-1990	0.000488	0.000266	0.49	58.0
28	Schwartz	1993	USA	Birmingham, Al.	1985-1988	0.001044	0.000415	1.05	47.9
29	Schwartz and Dockery	1992	USA	Steubenville	1974-1984	0.000693	0.000149	0.70	61.1
30	Ito et al.	1995	USA	Cook c.	1985-1990	0.000583	0.000218	0.58	38.0
31	Schwartz and Dockery	1992	USA	Philadelphia	1973-1980	0.001202	0.000238	1.21	42.5
32	Pope et al.	1992	USA	Utah valley	1985-1989	0.001470	0.000310	1.48	47.0
33	Pope	1999	USA	Ogden	1985-1995	0.001360	0.000620	1.37	32.1
34	Pope	1999	USA	Salt Lake City	1985-1995	0.000460	0.000230	0.46	41.2
35	Pope	1999	USA	Provo/Orem	1985-1995	0.000870	0.000270	0.87	38.4
36	Dockery et al.	1992	USA	St. Louis	1985-1986	0.001500	0.000690	1.51	27.6
37	Dockery et al.	1992	USA	Kingston	1985-1986	0.001600	0.001490	1.61	30.0
38	Schwartz	1994	USA	Detroit	1973-1982	0.000993	0.000264	1.00	47.9
39	Kelsall	1997	USA	Philadelphia	1974-1988	0.000603	0.000194	0.60	37.0
40	Mar et al.	2000	USA	Phoenix	1995-1997	0.001060	0.000535	1.08	46.5
41	Schwartz et al.	1996	USA	Boston	1979-1986	0.001193	0.000252	1.21	24.5
42	Schwartz et al.	1996	USA	Knoxville	1980-1987	0.000896	0.000430	0.91	32.0
43	Schwartz et al.	1996	USA	St. Louis	1979-1987	0.000598	0.000228	0.61	30.6
44	Schwartz et al.	1996	USA	Steubenville	1979-1987	0.000896	0.000379	0.91	45.6
45	Schwartz et al.	1996	USA	Portage	1979-1987	0.000698	0.000532	0.71	17.8
46	Schwartz et al.	1996	USA	Topeka	1979-1988	-0.000501	0.000276	-0.51	26.7
47	Ostro et al.	1999	USA	Coachella Valley, CA	1989-1992	0.000900	0.000400	0.91	62.0
48	Hong et al.	1999	South Korea	Inchon	1995-1996	0.000700	0.000300	0.70	71.2
49	Lee et al.	1999	South Korea	Seoul	1991-1995	0.000904	0.000181	0.91	50.9
50	Lee et al.	1999	South Korea	Ulsan	1991-1995	-0.000018	0.000362	-0.02	39.7
51	Peters et al.	2000	Czech	Coal Basin	1982-1994	0.000678	0.000273	0.68	66.7
52	Peters et al.	2000	Germany	Bavaria	1982-1994	0.000073	0.000250	0.07	28.4
53	Xu et al.	2000	China	Shenyang	1992	0.000315	0.000100	0.32	236.5
54	Xu et al.	1994	China	Beijing	1989	0.000200	0.000164	0.20	206.3
55	Borja-Aburto et al.	1997	Mexico	Mexico city	1990-1992	0.000887	0.000164	0.89	118.8
56	Castillejos et al.	2000	Mexico	Mexico city	1992-1995	0.001813	0.000426	1.83	44.6
57	Verhoeff et al.	1996	Netherlands	Amsterdam	1986-1992	0.000602	0.000379	0.60	38.0
58	Hoek et al.	1997	Netherlands	Rotterdam	1983-1991	0.000975	0.000389	0.98	23.1

59	Hoek et al.	2000	Netherlands		1986-1994	0.000178	0.000078	0.18	34.0
60	Bremner	1999	UK	London	1992-1994	0.000260	0.000231	0.26	28.5
61	Wietlisbach et al.	1996	Switzerland	Zurich	1984-1989	0.000855	0.000382	0.86	25.4
62	Wietlisbach et al.	1996	Switzerland	Basel	1984-1989	0.003018	0.000545	3.06	24.9
63	Hales et al.	2000	New Zealand	Christchurch	1988-1993	0.000128	0.000043	0.13	28.0
64	Morgan et al.	1998	Australia	Sydney	1989-1993	0.000764	0.000300	0.77	18.0
65	Simpson et al.	1997	Australia	Brisbane	1987-1993	0.000815	0.000276	0.82	26.9
66	Simpson et al.	2000	Australia	Melbourne	1991-1996	0.000300	0.000459	0.30	19.0
67	Michelozzi et al.	1998	Italy	Rome	1992-1995	0.000493	0.000195	0.49	64.8
68	Cadum et al.	1999	Italy	Turin	1991-1996	0.001356	0.000214	1.38	66.0
69	Burnett et al.	1998	Canada	Toronto	1980-1994	0.000409	0.000134	0.41	32.9
70	Ostro et al.	1999	Thailand	Bangkok	1992-1995	0.000900	0.000300	0.91	66.1

Sources: The data are taken from the studies referenced in column 1 of the table.

Notes:

- ¹ The information concerning the effect from exposure to air pollution on the risk of mortality uncovered by the various studies was expressed in a number of alternative ways. For those studies not expressing the effect in terms of the original regression coefficient from Poisson model, the results had to be converted. The natural logarithm of relative risk (RR) divided by number of units of air pollution increase, i.e. DP , gives the original regression coefficient, b , (relative risk indicates the ratio of the probability of occurrence of a given effect between two different exposure levels or exposure groups): i.e. $b = \ln(RR) / \Delta P$. If the effect is given in terms of percentage increase in mortality associated with a certain increase in pollutant level, the relative risk may be obtained by dividing the percentage increase by 100 and adding one. Finally, when the effect is given in terms of a coefficient of elasticity (i.e. change in mortality rate due to a change in the logarithm of pollution), the relative risk is obtained by multiplying the coefficient of elasticity, $e_{M,P}$, with the change in logarithm of pollution and taking its exponential: i.e. $RR = \exp(e_{M,P} * \Delta \ln(P))$.
- ² The standard error was expressed in a manner consistent with the estimated effect, and was hence converted in a similar manner (see footnote 1). Some studies only gave information on the confidence interval, and the standard error was calculated from this information.
- ³ The change in daily mortality (%) per $10\mu\text{g}/\text{m}^3$ increase in PM_{10} was calculated as follows: $(\text{EXP}(b * 10) - 1) * 100$, where b is the original regression coefficient.
- ⁴ Each study in the meta-analysis supplied mean values of daily data over the study period for either TSP, BS, or PM. TSP and PM_{13} were converted to PM_{10} using the factors of 0.55 and 0.77, respectively, and black smoke was considered equal to PM_{10} . Note that this implied dividing the estimated coefficients in studies using the TSP and PM_{13} measures by 0.55 and 0.77, respectively, in order to convert these into being PM_{10} or BS effects.

Table D2: Data gathered from SIMA and WHO

OBS	OVER65	GNP	GINI	DIST	EDUC	DALE	LEAB	PHYSICIANS
1	13.68	10815	32.70	353663	82.84	72.50	76.79	3.40
2	13.08	10727	32.50	348628	90.90	72.80	76.44	3.77
3	9.99	8313	19.50	162099	89.74	66.60	71.03	3.30
4	9.78	6465	27.20	175835	76.01	66.20	70.79	1.90
5	9.80	6465	27.20	175835	76.32	66.20	70.79	1.94
6	9.70	6465	27.20	175835	77.63	66.20	70.92	2.06
7	9.77	6465	27.20	175835	76.01	66.20	70.86	1.90
8	15.61	15693	34.35	539069	88.13	71.70	75.57	1.50
9	15.11	18834	28.10	529247	83.68	70.40	72.55	2.26
10	13.46	14982	32.70	489919	95.82	73.10	76.45	2.90
11	4.47	1543	31.77	49026	59.48	53.20	61.14	0.41
12	6.14	4597	53.41	245503	79.50	68.60	73.70	1.09
13	15.08	14367	32.50	466936	91.90	72.80	77.18	4.10
14	15.08	14367	32.50	466936	91.90	72.80	77.18	4.10
15	15.39	14867	32.50	483182	91.90	72.80	77.51	4.13
16	14.93	14100	32.50	458240	91.90	72.80	76.93	4.07
17	14.61	13573	32.50	441127	91.90	72.80	76.80	4.02
18	15.24	14575	32.50	473695	91.90	72.80	77.29	4.12
19	15.24	14575	32.50	473695	91.90	72.80	77.29	4.12
20	15.39	14809	32.50	481293	91.90	72.80	77.45	4.12
21	14.45	13325	32.50	433061	91.90	72.80	76.78	3.98
22	14.77	13865	32.50	450611	91.90	72.80	76.88	4.04
23	14.77	13865	32.50	450611	91.90	72.80	76.88	4.04
24	15.08	14367	32.50	466936	91.90	72.80	77.18	4.10
25	14.45	13387	32.50	435077	91.90	72.80	76.80	4.00
26	11.13	11848	40.10	475118	95.37	70.00	73.74	1.92
27	12.12	20213	40.10	810555	94.15	70.00	74.82	2.40
28	12.00	19070	40.10	764707	95.25	70.00	74.68	2.40
29	11.05	11680	40.10	468368	95.82	70.00	73.54	1.99
30	12.12	20213	40.10	810555	94.15	70.00	74.82	2.40
31	10.71	10095	40.10	404810	93.90	70.00	72.86	1.83

32	12.06	19724	40.10	790932	94.66	70.00	74.75	2.40
33	12.29	22466	40.10	900901	94.81	70.00	75.14	2.50
34	12.29	22466	40.10	900901	94.81	70.00	75.14	2.50
35	12.29	22466	40.10	900901	94.81	70.00	75.14	2.50
36	11.89	17235	40.10	691124	96.05	70.00	74.59	2.40
37	11.89	17235	40.10	691124	96.05	70.00	74.59	2.40
38	10.85	10964	40.10	439646	95.37	70.00	73.12	1.92
39	11.30	13791	40.10	553036	95.57	70.00	73.84	2.09
40	12.46	28527	40.80	1163888	96.43	70.00	75.93	2.63
41	11.52	14408	40.10	577741	95.89	70.00	74.25	1.99
42	11.64	15444	40.10	619294	95.78	70.00	74.37	1.99
43	11.58	15029	40.10	602658	95.78	70.00	74.31	1.99
44	11.58	15029	40.10	602658	95.78	70.00	74.31	1.99
45	11.58	15029	40.10	602658	95.78	70.00	74.31	1.99
46	11.64	15707	40.10	629851	95.57	70.00	74.36	1.99
47	12.4	22998	40.10	922200	93.08	70.00	75.31	2.47
48	5.73	13885	31.60	438777	98.30	65.00	72.31	1.15
49	5.37	11357	31.60	358882	91.04	65.00	70.96	1.02
50	5.37	11357	31.60	358882	91.04	65.00	70.96	1.02
51	12.32	10831	26.60	288103	90.84	68.00	71.78	2.77
52	14.90	19805	29.00	574344	89.29	70.40	75.00	2.99
53	5.90	1748	41.50	72549	60.90	62.30	69.01	1.54
54	5.49	1291	41.50	53591	56.40	68.87	69.01	1.54
55	4.03	6385	50.30	321171	57.53	65.00	70.59	1.13
56	4.19	6914	50.30	347759	60.55	65.00	70.78	1.20
57	12.65	15945	31.50	502262	95.46	72.00	76.96	2.55
58	12.38	13982	31.50	440431	96.32	72.00	76.77	2.44
59	12.75	16734	32.10	537151	96.3	72.00	77.06	2.55
60	15.76	17520	36.10	632486	92.37	71.70	76.29	1.55
61	14.22	20006	34.60	692209	80.48	72.50	77.17	1.58
62	14.22	20006	34.60	692209	80.48	72.50	77.17	1.58
63	11.17	13755	43.90	603855	89.13	69.20	75.47	1.95
64	11.23	16716	33.70	563335	90.66	73.20	77.29	2.30
65	11.06	16278	33.70	548559	90.37	73.20	76.90	2.30
66	11.54	18838	35.20	663111	93.03	73.20	77.85	2.40

67	15.83	18962	27.30	517649	87.23	72.70	77.17	5.20
68	15.86	18953	29.30	555335	87.13	72.70	77.17	5.20
69	10.64	16149	31.50	508697	91.12	72.00	76.59	2.09
70	4.81	5284	46.20	244100	37.4	60.2	69.99	0.23

Sources: Data for all of the variables except Disability-Adjusted Life Expectancy (DALE) were obtained from SIMA (World Bank's internal database system). The DALE estimate for the population of each country was found in the World Health Report, Annex Table 5, of the World Health Organisation.

Appendix E: Regression Results for the Full Sample Using OLS and VWLS

. reg regco poll over65 gnp gini educ dale

Source	SS	df	MS	Number of obs = 70
Model	3.99686254	6	.666143757	F(6, 63) = 1.16
Residual	36.1019116	63	.573046215	Prob > F = 0.3377
Total	40.0987741	69	.581141654	R-squared = 0.0997
				Adj R-squared = 0.0139
				Root MSE = .757

regcol	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
poll	-.0031723	.003138	-1.011	0.316	-.0094432	.0030986
over65	.0117826	.0617333	0.191	0.849	-.1115815	.1351468
gnp	.0000198	.0000273	0.724	0.472	-.0000349	.0000744
gini	.0294887	.0185537	1.589	0.117	-.0075879	.0665652
educ	-.011089	.0122062	-0.908	0.367	-.0354811	.0133032
dale	.0030789	.0522493	0.059	0.953	-.1013329	.1074907
_cons	.1696556	2.989451	0.057	0.955	-5.804285	6.143596

. reg regco poll over65 gnp dist educ dale

Source	SS	df	MS	Number of obs = 70
Model	3.64420902	6	.60736817	F(6, 63) = 1.05
Residual	36.4545651	63	.57864389	Prob > F = 0.4023
Total	40.0987741	69	.581141654	R-squared = 0.0909
				Adj R-squared = 0.0043
				Root MSE = .76069

regcol	Coef.	Std. Err.	t	P> t	[95% Conf. Interval]	
poll	-.0028316	.0031369	-0.903	0.370	-.0091003	.003437
over65	-.0060344	.0581366	-0.104	0.918	-.1222111	.1101424
gnp	-.000055	.000071	-0.775	0.441	-.000197	.0000869
dist	2.10e-06	1.53e-06	1.376	0.174	-9.51e-07	5.15e-06
educ	-.01555	.0120439	-1.291	0.201	-.0396177	.0085178
dale	.0276085	.0494173	0.559	0.578	-.071144	.1263611
_cons	.090421	3.002442	0.030	0.976	-5.90948	6.090322

. wls regco poll over65 gni educ dale, sd(se)

Variance-weighted least-squares regression
 Goodness-of-fit chi2(63) = 281.45
 Prob > chi2 = 0.0000

Number of obs = 70
 Model chi2(6) = 47.57
 Prob > chi2 = 0.0000

regcol	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.001852	.0006687	2.770	0.006	.0005414	.0031626
over65	-.0838304	.0167197	-5.014	0.000	-.1166004	-.0510604
gni	.0000402	8.65e-06	4.648	0.000	.0000233	.0000572
gni	-.0042486	.0035616	-1.193	0.233	-.0112292	.002732
educ	-.0019518	.0040647	-0.480	0.631	-.0099185	.0060149
dale	.0341614	.0122922	2.779	0.005	.0100691	.0582537
_cons	-1.365648	.6996823	-1.952	0.051	-2.737	.005704

. wls regco poll over65 gni dist educ dale, sd(se)

Variance-weighted least-squares regression
 Goodness-of-fit chi2(63) = 279.67
 Prob > chi2 = 0.0000

Number of obs = 70
 Model chi2(6) = 49.35
 Prob > chi2 = 0.0000

regcol	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0016237	.0006708	2.421	0.015	.0003091	.0029384
over65	-.0845519	.0154389	-5.477	0.000	-.1148116	-.0542921
gni	.0000605	.0000155	3.912	0.000	.0000302	.0000908
dist	-5.59e-07	3.12e-07	-1.791	0.073	-1.17e-06	5.27e-08
educ	-.0014427	.0040749	-0.354	0.723	-.0094294	.0065441
dale	.0300672	.0117339	2.562	0.010	.0070692	.0530653
_cons	-1.257962	.6975314	-1.803	0.071	-2.625098	.1091746

Appendix F: Regression Results for a Random Sample of Sample Combinations.

In the regression results supplied below, each country enters with only one observation.

```
. ml model d0 d0prog1 (regco = poll over65 gnp gini educ dale ) / tau2 if c1>0
```

```
Number of obs = 21
Wald chi2(6) = 6.08
Prob > chi2 = 0.4147
Log likelihood = -41.361076
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0008634	.0016022	-0.539	0.590	-.0040036	.0022769
over65	-.0086392	.0356966	-0.242	0.809	-.0786033	.0613248
gnp	.0000103	.0000206	0.498	0.618	-.0000301	.0000507
gini	.0129299	.0102547	1.261	0.207	-.0071689	.0330287
educ	-.0038472	.0064883	-0.593	0.553	-.016564	.0088696
dale	.0074275	.027462	0.270	0.787	-.046397	.061252
_cons	-.0765322	1.354843	-0.056	0.955	-2.731975	2.578911
tau2						
_cons	.0748617	.0294253	2.544	0.011	.017189	.1325343

```
. ml model d0 d0prog (regco = poll over65 gnp gini educ dale ) / tau2 if c2>0
```

```
Number of obs = 21
Wald chi2(6) = 11.83
Prob > chi2 = 0.0658
Log likelihood = -59.636477
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005737	.0026475	-0.217	0.828	-.0057627	.0046153
over65	-.0957259	.053393	-1.793	0.073	-.2003742	.0089224
gnp	.0000858	.0000347	2.472	0.013	.0000178	.0001538
gini	-.0028934	.0155448	-0.186	0.852	-.0333607	.0275739
educ	-.0228124	.0108283	-2.107	0.035	-.0440355	-.0015892
dale	.030321	.0346143	0.876	0.381	-.0375217	.0981637
_cons	.518916	1.860709	0.279	0.780	-3.128007	4.165839
tau2						
_cons	.2902382	.1074444	2.701	0.007	.079651	.5008253

```
. ml model d0 d0prog3 (regco = poll over65 gnp gini educ dale ) / tau2 if c3> 0
Number of obs = 21
Wald chi2(6) = 12.81
Prob > chi2 = 0.0462
Log likelihood = -46.552821
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0004785	.0013978	-0.342	0.732	-.0032181	.0022611
over65	-.0058429	.0380754	-0.153	0.878	-.0804693	.0687835
gnp	8.93e-06	.0000215	0.416	0.678	-.0000332	.000051
gini	.0117473	.0099307	1.183	0.237	-.0077166	.0312112
educ	-.0164378	.0070205	-2.341	0.019	-.0301978	-.0026778
dale	.0274064	.027457	0.998	0.318	-.0264083	.0812211
_cons	-.5030326	1.31468	-0.383	0.702	-3.079759	2.073694
tau2						
_cons	.0669139	.0361628	1.850	0.064	-.0039638	.1377916

```
. ml model d0 d0prog4 (regco = poll over65 gnp gini educ dale ) / tau2 if c4>0
Number of obs = 21
Wald chi2(6) = 12.82
Prob > chi2 = 0.0460
Log likelihood = -56.603155
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0026665	.002568	1.038	0.299	-.0023666	.0076996
over65	.0534209	.047811	1.117	0.264	-.0402869	.1471288
gnp	.000069	.000031	2.222	0.026	8.15e-06	.0001298
gini	.022429	.0140201	1.600	0.110	-.0050498	.0499079
educ	-.0131625	.0086045	-1.530	0.126	-.0300271	.0037021
dale	-.0188534	.0313242	-0.602	0.547	-.0802477	.0425409
_cons	.6598235	1.636398	0.403	0.687	-2.547458	3.867105
tau2						
_cons	.2100956	.0834808	2.517	0.012	.0464763	.373715

```
. ml model d0 d0prog5 (regco = poll over65 gnp gini educ dale ) / tau2 if c5> 0
Number of obs = 21
Wald chi2(6) = 20.96
Prob > chi2 = 0.0019
Log likelihood = -39.845363
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0006733	.0014577	0.462	0.644	-.0021837	.0035302
over65	-.1097985	.0293858	-3.736	0.000	-.1673936	-.0522033
gnp	.0000457	.0000188	2.433	0.015	8.89e-06	.0000826
gini	-.005942	.0081729	-0.727	0.467	-.0219607	.0100767
educ	-.0086456	.0061046	-1.416	0.157	-.0206103	.0033191
dale	.0405547	.0228899	1.772	0.076	-.0043087	.0854181
_cons	-.8398555	1.200993	-0.699	0.484	-3.193758	1.514047
tau2						
_cons	.0551182	.0249164	2.212	0.027	.0062829	.1039535

```
. ml model d0 d0prog6 (regco = poll over65 gnp gini educ dale ) / tau2 if c6>0
Number of obs = 21
Wald chi2(6) = 10.36
Prob > chi2 = 0.1105
Log likelihood = -60.052485
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0011176	.002853	-0.392	0.695	-.0067094	.0044742
over65	.0157608	.0593338	0.266	0.791	-.1005314	.1320529
gnp	.0000434	.0000409	1.062	0.288	-.0000367	.0001236
gini	.020833	.0160285	1.300	0.194	-.0105823	.0522484
educ	-.0217648	.0107806	-2.019	0.043	-.0428944	-.0006352
dale	.0076838	.0362123	0.212	0.832	-.063291	.0786586
_cons	.5178108	1.89681	0.273	0.785	-3.199869	4.235491
tau2						
_cons	.3100155	.112988	2.744	0.006	.0885632	.5314679

```
. ml model d0 d0prog7 (regco = poll over65 gnp gini educ dale ) / tau2 if c7>0
Number of obs = 21
Wald chi2(6) = 11.10
Prob > chi2 = 0.0852
Log likelihood = -50.665526
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0008796	.0017237	-0.510	0.610	-.0042581	.0024988
over65	-.020491	.0450024	-0.455	0.649	-.1086942	.0677121
gnp	.00004	.0000242	1.653	0.098	-7.43e-06	.0000874
gini	.0211933	.0125281	1.692	0.091	-.0033613	.0457478
educ	-.0060512	.0082835	-0.731	0.465	-.0222866	.0101843
dale	-.0073191	.0344719	-0.212	0.832	-.0748827	.0602445
_cons	.6446868	1.638074	0.394	0.694	-2.565879	3.855253
tau2						
_cons	.1220844	.0483076	2.527	0.011	.0274033	.2167655

```
. ml model d0 d0prog8 (regco = poll over65 gnp gini educ dale ) / tau2 if c8>0
Number of obs = 21
Wald chi2(6) = 11.56
Prob > chi2 = 0.0724
Log likelihood = -57.789363
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0021584	.002653	0.814	0.416	-.0030414	.0073582
over65	-.0557361	.0490866	-1.135	0.256	-.1519441	.0404719
gnp	.000095	.0000329	2.886	0.004	.0000305	.0001596
gini	.0022487	.0143583	0.157	0.876	-.025893	.0303903
educ	-.0144162	.0099787	-1.445	0.149	-.033974	.0051417
dale	.0154026	.03134	0.491	0.623	-.0460227	.0768278
_cons	-.0106618	1.692653	-0.006	0.995	-3.3282	3.306876
tau2						
_cons	.2323947	.0916842	2.535	0.011	.0526969	.4120925

. ml model d0 d0prog9 (regco = poll over65 gnp gini educ dale) / tau2 if c9>0

Number of obs = 21
Wald chi2(6) = 20.60
Prob > chi2 = 0.0022

Log likelihood = -36.620218

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0005383	.0011383	0.473	0.636	-.0016928	.0027694
over65	-.0262215	.0278982	-0.940	0.347	-.0809011	.028458
gnp	.000017	.0000148	1.153	0.249	-.0000119	.000046
gini	.0044916	.0068194	0.659	0.510	-.0088743	.0178574
educ	-.0163131	.0052966	-3.080	0.002	-.0266943	-.0059319
dale	.0362147	.0190977	1.896	0.058	-.0012162	.0736455
_cons	-.8936275	.9963862	-0.897	0.370	-2.846509	1.059254
tau2						
_cons	.0270157	.0143073	1.888	0.059	-.0010261	.0550575

. ml model d0 d0prog10 (regco = poll over65 gnp gini educ dale) / tau2 if c10>0

Number of obs = 21
Wald chi2(6) = 11.38
Prob > chi2 = 0.0772

Log likelihood = -59.331796

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0004563	.0025717	-0.177	0.859	-.0054968	.0045842
over65	.0150591	.0538812	0.279	0.780	-.0905461	.1206643
gnp	.0000571	.0000308	1.858	0.063	-3.13e-06	.0001174
gini	.0164117	.0159063	1.032	0.302	-.0147641	.0475875
educ	-.0202872	.0102959	-1.970	0.049	-.0404668	-.0001075
dale	-.0036402	.0345555	-0.105	0.916	-.0713676	.0640873
_cons	1.174953	1.795952	0.654	0.513	-2.345049	4.694956
tau2						
_cons	.264388	.1024882	2.580	0.010	.0635148	.4652612

. ml model d0 d0prog11 (regco = poll over65 gnp gini educ dale) / tau2 if c11>0

Number of obs = 21
Wald chi2(6) = 14.21
Prob > chi2 = 0.0273

Log likelihood = -46.567254

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005675	.001445	-0.393	0.695	-.0033995	.0022646
over65	-.0946786	.034787	-2.722	0.006	-.16286	-.0264973
gnp	.0000355	.0000225	1.574	0.116	-8.71e-06	.0000797
gini	.0008488	.0098769	0.086	0.932	-.0185097	.0202072
educ	-.0096141	.0071078	-1.353	0.176	-.0235452	.004317
dale	.038468	.0273919	1.404	0.160	-.0152191	.0921552
_cons	-.7223563	1.318953	-0.548	0.584	-3.307457	1.862745
tau2						
_cons	.0714972	.0340504	2.100	0.036	.0047597	.1382347

. ml model d0 d0prog12 (regco = poll over65 gnp gini educ dale) / tau2 if c12>0

Number of obs = 21
Wald chi2(6) = 13.37
Prob > chi2 = 0.0376

Log likelihood = -60.762544

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0023236	.002649	0.877	0.380	-.0028682	.0075155
over65	.0138613	.0516725	0.268	0.789	-.0874149	.1151375
gnp	.0000739	.000036	2.056	0.040	3.45e-06	.0001444
gini	.0103033	.0142352	0.724	0.469	-.0175972	.0382038
educ	-.023356	.0098721	-2.366	0.018	-.0427051	-.004007
dale	.0107396	.0310045	0.346	0.729	-.0500281	.0715073
_cons	.1546618	1.654462	0.093	0.926	-3.088025	3.397349
tau2						
_cons	.2184805	.0934211	2.339	0.019	.0353786	.4015825

. ml model d0 d0prog13 (regco = poll over65 gnp gini educ dale) / tau2 if c13>0

Number of obs = 21
Wald chi2(6) = 6.75
Prob > chi2 = 0.3445

Log likelihood = -53.687934

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0008195	.0018503	-0.443	0.658	-.004446	.002807
over65	-.0352514	.0395734	-0.891	0.373	-.1128139	.042311
gnp	.0000246	.0000248	0.993	0.321	-.000024	.0000733
gini	.0117938	.0116333	1.014	0.311	-.011007	.0345947
educ	-.0045706	.0071821	-0.636	0.525	-.0186472	.009506
dale	.0036871	.0319272	0.115	0.908	-.058889	.0662632
_cons	.3479319	1.575462	0.221	0.825	-2.739917	3.435781
tau2						
_cons	.1082984	.0518228	2.090	0.037	.0067275	.2098693

. ml model d0 d0prog14 (regco = poll over65 gnp gini educ dale) / tau2 if c14>0

Number of obs = 21
Wald chi2(6) = 10.32
Prob > chi2 = 0.1119

Log likelihood = -58.404488

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0002928	.0027661	0.106	0.916	-.0051286	.0057142
over65	-.0668596	.0534701	-1.250	0.211	-.1716591	.03794
gnp	.000087	.000039	2.228	0.026	.0000105	.0001635
gini	.0055993	.0147901	0.379	0.705	-.0233886	.0345873
educ	-.018301	.0100758	-1.816	0.069	-.0380492	.0014471
dale	.0208245	.0333586	0.624	0.532	-.0445571	.0862061
_cons	.2107019	1.785615	0.118	0.906	-3.289038	3.710442
tau2						
_cons	.267619	.1003434	2.667	0.008	.0709495	.4642885

. ml model d0 d0prog15 (regco = poll over65 gnp gini educ dale) / tau2 if c15>0

Number of obs = 21
Wald chi2(6) = 15.76
Prob > chi2 = 0.0151

Log likelihood = -41.859212

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0002745	.0011956	-0.230	0.818	-.0026179	.0020688
over65	-.0284832	.0336784	-0.846	0.398	-.0944917	.0375253
gnp	.0000135	.0000162	0.829	0.407	-.0000184	.0000453
gini	.00618	.0087359	0.707	0.479	-.0109421	.0233021
educ	-.0186527	.0064701	-2.883	0.004	-.0313339	-.0059715
dale	.0406472	.0241686	1.682	0.093	-.0067223	.0880167
_cons	-.8771405	1.15596	-0.759	0.448	-3.14278	1.388499
tau2						
_cons	.0473597	.0244992	1.933	0.053	-.0006577	.0953772

. ml model d0 d0prog16 (regco = poll over65 gnp gini educ dale) / tau2 if c16>0

Number of obs = 21
Wald chi2(6) = 11.29
Prob > chi2 = 0.0800

Log likelihood = -58.651617

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0024846	.0028603	0.869	0.385	-.0031216	.0080907
over65	.0161462	.0531891	0.304	0.761	-.0881026	.120395
gnp	.000088	.0000344	2.559	0.010	.0000206	.0001554
gini	.0216805	.0159241	1.361	0.173	-.0095302	.0528911
educ	-.0135433	.0099639	-1.359	0.174	-.0330722	.0059857
dale	-.0222279	.0352143	-0.631	0.528	-.0912466	.0467908
_cons	1.091324	1.841617	0.593	0.553	-2.518179	4.700826
tau2						
_cons	.2773465	.0998763	2.777	0.005	.0815925	.4731005

. ml model d0 d0prog17 (regco = poll over65 gnp gini educ dale) / tau2 if c17>0

Number of obs = 21
Wald chi2(6) = 5.15
Prob > chi2 = 0.5247

Log likelihood = -45.685287

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0011067	.0018063	-0.613	0.540	-.0046471	.0024337
over65	-.0193044	.0378261	-0.510	0.610	-.0934422	.0548335
gnp	.000016	.0000257	0.622	0.534	-.0000343	.0000663
gini	.0105211	.0110593	0.951	0.341	-.0111548	.032197
educ	-.0031256	.0074347	-0.420	0.674	-.0176973	.0114461
dale	.0069095	.0302115	0.229	0.819	-.0523039	.066123
_cons	.0878029	1.481664	0.059	0.953	-2.816204	2.99181
tau2						
_cons	.0979595	.0394004	2.486	0.013	.0207362	.1751828

. ml model d0 d0prog18 (regco = poll over65 gnp gini educ dale) / tau2 if c18>0

Number of obs = 21
Wald chi2(6) = 13.17
Prob > chi2 = 0.0405

Log likelihood = -58.798355

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0000502	.0027792	-0.018	0.986	-.0054973	.0053969
over65	.016604	.0555866	0.299	0.765	-.0923438	.1255518
gnp	.0000595	.0000397	1.497	0.134	-.0000184	.0001373
gini	.0148796	.0148493	1.002	0.316	-.0142245	.0439837
educ	-.0274776	.0102835	-2.672	0.008	-.0476328	-.0073223
dale	.0131231	.0333718	0.393	0.694	-.0522845	.0785306
_cons	.5365526	1.795015	0.299	0.765	-2.981612	4.054717
tau2						
_cons	265658	.1032305	2.573	0.010	.0633298	.4679861

. ml model d0 d0prog19 (regco = poll over65 gnp gini educ dale) / tau2 if c19>0

Number of obs = 21
Wald chi2(6) = 11.98
Prob > chi2 = 0.0623

Log likelihood = -48.332331

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0009277	.0017356	-0.534	0.593	-.0043294	.0024741
over65	-.0633143	.0431759	-1.466	0.143	-.1479376	.0213089
gnp	.0000413	.0000253	1.632	0.103	-8.30e-06	.0000909
gini	.011207	.012489	0.897	0.370	-.013271	.035685
educ	-.0107936	.0080988	-1.333	0.183	-.026667	.0050798
dale	.0131947	.0349878	0.377	0.706	-.0553802	.0817696
_cons	.349176	1.660689	0.210	0.833	-2.905714	3.604066
tau2						
_cons	.1246866	.0499158	2.498	0.012	.0268535	.2225198

. ml model d0 d0prog20 (regco = poll over65 gnp gini educ dale) / tau2 if c20>0

Number of obs = 21
Wald chi2(6) = 11.28
Prob > chi2 = 0.0802

Log likelihood = -56.706088

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0010669	.0025493	0.419	0.676	-.0039295	.0060634
over65	-.0200338	.0488143	-0.410	0.682	-.115708	.0756404
gnp	.0000873	.0000354	2.469	0.014	.000018	.0001566
gini	.0105159	.0144621	0.727	0.467	-.0178293	.0388611
educ	-.0145633	.0097144	-1.499	0.134	-.0336033	.0044766
dale	-.001246	.0318375	-0.039	0.969	-.0636463	.0611543
_cons	.6808426	1.658054	0.411	0.681	-2.568883	3.930568
tau2						
_cons	.2300906	.0867875	2.651	0.008	.0599901	.400191

. ml model d0 d0prog21 (regco = poll over65 gnp gini educ dale) / tau2 if c21>0

Number of obs = 21
Wald chi2(6) = 21.63
Prob > chi2 = 0.0014

Log likelihood = -41.592121

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0004138	.0013287	0.311	0.755	-.0021905	.0030181
over65	-.0163723	.0310397	-0.527	0.598	-.077209	.0444644
gnp	3.17e-06	.0000194	0.163	0.870	-.0000349	.0000413
gini	.000022	.007641	0.003	0.998	-.014954	.0149981
educ	-.0205853	.0056896	-3.618	0.000	-.0317367	-.009434
dale	.0500904	.0212421	2.358	0.018	.0084567	.091724
_cons	-1.282094	1.096724	-1.169	0.242	-3.431634	.8674454
tau2						
_cons	.0396367	.021711	1.826	0.068	-.002916	.0821895

. ml model d0 d0prog22 (regco = poll over65 gnp gini educ dale) / tau2 if c22>0

Number of obs = 21
Wald chi2(6) = 11.09
Prob > chi2 = 0.0857

Log likelihood = -60.190957

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0006364	.0025486	-0.250	0.803	-.0056315	.0043587
over65	.0214666	.055022	0.390	0.696	-.0863746	.1293078
gnp	.0000544	.0000308	1.765	0.078	-6.02e-06	.0001148
gini	.0176825	.0163909	1.079	0.281	-.0144432	.0498081
educ	-.0197888	.0105649	-1.873	0.061	-.0404956	.000918
dale	-.0054879	.03559	-0.154	0.877	-.075243	.0642673
_cons	1.204919	1.845444	0.653	0.514	-2.412084	4.821923
tau2						
_cons	.27935	.1080409	2.586	0.010	.0675938	.4911062

. ml model d0 d0prog23 (regco = poll over65 gnp gini educ dale) / tau2 if c23>0

Number of obs = 21
Wald chi2(6) = 10.67
Prob > chi2 = 0.0992

Log likelihood = -49.11387

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.001356	.0015867	-0.855	0.393	-.0044658	.0017539
over65	-.0680085	.0376535	-1.806	0.071	-.1418081	.005791
gnp	.0000292	.0000234	1.247	0.212	-.0000167	.0000751
gini	.0056402	.0118187	0.477	0.633	-.017524	.0288045
educ	-.0099035	.0077477	-1.278	0.201	-.0250888	.0052819
dale	.0248575	.0326064	0.762	0.446	-.0390498	.0887649
_cons	-.0674224	1.511432	-0.045	0.964	-3.029776	2.894931
tau2						
_cons	.0993821	.0431607	2.303	0.021	.0147886	.1839756

. ml model d0 d0prog24 (regco = poll over65 gnp gini educ dale) / tau2 if c24>0

Number of obs = 21
Wald chi2(6) = 10.94
Prob > chi2 = 0.0904

Log likelihood = -56.772328

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0024793	.0026744	0.927	0.354	-.0027624	.007721
over65	-.0315979	.0448144	-0.705	0.481	-.1194325	.0562366
gnp	.0000877	.0000348	2.519	0.012	.0000195	.000156
gini	.0041463	.014192	0.292	0.770	-.0236695	.0319621
educ	-.0195454	.0097068	-2.014	0.044	-.0385704	-.0005204
dale	.0130661	.0314044	0.416	0.677	-.0484854	.0746177
_cons	.2302503	1.662984	0.138	0.890	-3.029138	3.489638
tau2						
_cons	.2231022	.0862486	2.587	0.010	.054058	.3921464

. ml model d0 d0pro (regco1 = poll over65 gnp dist educ dale) / tau2 if c1>0

Number of obs = 21
Wald chi2(6) = 4.81
Prob > chi2 = 0.5684

Log likelihood = -60.497596

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005431	.0016626	-0.327	0.744	-.0038017	.0027155
over65	-.0278231	.0316119	-0.880	0.379	-.0897813	.0341351
gnp	-9.20e-06	.0000369	-0.249	0.803	-.0000815	.0000631
dist	6.32e-07	1.00e-06	0.631	0.528	-1.33e-06	2.60e-06
educ	-.0065429	.0060171	-1.087	0.277	-.0183361	.0052503
dale	.0235745	.023129	1.019	0.308	-.0217576	.0689066
_cons	-.3484194	1.341302	-0.260	0.795	-2.977324	2.280485
tau2						
_cons	.0766539	.0310815	2.466	0.014	.0157353	.1375724

. ml model d0 d0pro2 (regco = poll over65 gnp dist educ dale) / tau2 if c2>0

Number of obs = 21
Wald chi2(6) = 12.26
Prob > chi2 = 0.0565

Log likelihood = -78.018915

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0006192	.0026217	-0.236	0.813	-.0057576	.0045192
over65	-.1016589	.0494967	-2.054	0.040	-.1986706	-.0046472
gnp	.000117	.0000667	1.754	0.079	-.0000138	.0002478
dist	-8.32e-07	1.46e-06	-0.570	0.569	-3.69e-06	2.03e-06
educ	-.023113	.0100144	-2.308	0.021	-.0427408	-.0034852
dale	.0305877	.0306722	0.997	0.319	-.0295287	.090704
_cons	.453215	1.84617	0.245	0.806	-3.165212	4.071642
tau2						
_cons	.2835818	.1062127	2.670	0.008	.0754087	.4917548

. ml model d0 d0pro3 (regco = poll over65 gnp dist educ dale) / tau2 if c3>0

Number of obs = 21

Wald chi2(6) = 11.96

Log likelihood = -65.572493

Prob > chi2 = 0.0630

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0000771	.0013923	-0.055	0.956	-.002806	.0026518
over65	-.0204152	.0340243	-0.600	0.548	-.0871016	.0462713
gnp	-.0000154	.0000388	-0.398	0.691	-.0000914	.0000605
dist	7.86e-07	9.00e-07	0.874	0.382	-9.77e-07	2.55e-06
educ	-.0190203	.0065926	-2.885	0.004	-.0319415	-.0060991
dale	.0418641	.0231024	1.812	0.070	-.0034159	.087144
_cons	-.7634688	1.296464	-0.589	0.556	-3.304491	1.777554
tau2						
_cons	.0696536	.0378484	1.840	0.066	-.0045279	.1438352

. ml model d0 d0pro4 (regco = poll over65 gnp dist educ dale) / tau2 if c4>0

Number of obs = 21

Wald chi2(6) = 11.37

Log likelihood = -75.447562

Prob > chi2 = 0.0777

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.003088	.0027298	1.131	0.258	-.0022622	.0084382
over65	.0354656	.0455506	0.779	0.436	-.053812	.1247432
gnp	.0000126	.0000576	0.219	0.827	-.0001003	.0001255
dist	1.83e-06	1.48e-06	1.238	0.216	-1.07e-06	4.74e-06
educ	-.0166334	.0084025	-1.980	0.048	-.0331019	-.0001648
dale	-.0030375	.0288789	-0.105	0.916	-.059639	.0535641
_cons	.7467562	1.709996	0.437	0.662	-2.604775	4.098287
tau2						
_cons	.2300362	.087502	2.629	0.009	.0585355	.4015369

. ml model d0 d0pro5 (regco = poll over65 gnp dist educ dale) / tau2 if c5>0

Number of obs = 21

Wald chi2(6) = 22.62

Log likelihood = -58.125198

Prob > chi2 = 0.0009

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0004273	.0014068	0.304	0.761	-.0023299	.0031845
over65	-.1096787	.0256529	-4.275	0.000	-.1599574	-.0594
gnp	.0000739	.0000319	2.312	0.021	.0000113	.0001365
dist	-8.21e-07	7.08e-07	-1.159	0.247	-2.21e-06	5.67e-07
educ	-.0078769	.0056303	-1.399	0.162	-.018912	.0031583
dale	.0362322	.0191027	1.897	0.058	-.0012085	.0736729
_cons	-.8023469	1.1521	-0.696	0.486	-3.060421	1.455727
tau2						
_cons	.0512909	.0240112	2.136	0.033	.0042299	.098352

```
. ml model d0 d0pro6 (regco = poll over65 gnp dist educ dale ) / tau2 if c6>0
Number of obs = 21
Wald chi2(6) = 9.76
Prob > chi2 = 0.1352
Log likelihood = -78.58854
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0006427	.0029719	-0.216	0.829	-.0064675	.0051822
over65	-.0011285	.056405	-0.020	0.984	-.1116803	.1094232
gnp	-.0000152	.0000677	-0.225	0.822	-.000148	.0001175
dist	1.92e-06	1.69e-06	1.137	0.255	-1.39e-06	5.23e-06
educ	-.0246351	.0104321	-2.361	0.018	-.0450816	-.0041885
dale	.0214091	.0331944	0.645	0.519	-.0436507	.0864688
_cons	.6189596	1.941824	0.319	0.750	-3.186945	4.424864
tau2						
_cons	.3241059	.1150498	2.817	0.005	.0986124	.5495994

```
. ml model d0 d0pro7 (regco = poll over65 gnp dist educ dale ) / tau2 if c7>0
Number of obs = 21
Wald chi2(6) = 10.55
Prob > chi2 = 0.1034
Log likelihood = -69.649745
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0001605	.0017096	-0.094	0.925	-.0035112	.0031903
over65	-.0413539	.0393238	-1.052	0.293	-.1184271	.0357193
gnp	-.000014	.0000462	-0.303	0.762	-.0001046	.0000766
dist	1.67e-06	1.09e-06	1.533	0.125	-4.65e-07	3.81e-06
educ	-.0105373	.0074536	-1.414	0.157	-.0251461	.0040715
dale	.016358	.028497	0.574	0.566	-.0394951	.0722111
_cons	.2807918	1.609129	0.174	0.861	-2.873044	3.434627
tau2						
_cons	.1247839	.0491282	2.540	0.011	.0284944	.2210734

```
. ml model d0 d0pro8 (regco = poll over65 gnp dist educ dale ) / tau2 if c8>0
Number of obs = 21
Wald chi2(6) = 11.55
Prob > chi2 = 0.0729
Log likelihood = -76.281458
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0021756	.0026497	0.821	0.412	-.0030176	.0073688
over65	-.0600763	.0457228	-1.314	0.189	-.1496913	.0295387
gnp	.0000973	.0000624	1.559	0.119	-.000025	.0002195
dist	-2.94e-08	1.39e-06	-0.021	0.983	-2.76e-06	2.70e-06
educ	-.0150989	.0093288	-1.619	0.106	-.0333829	.0031852
dale	.0179741	.0278018	0.647	0.518	-.0365164	.0724646
_cons	-.0228719	1.693375	-0.014	0.989	-3.341826	3.296083
tau2						
_cons	.2315559	.0914157	2.533	0.011	.0523845	.4107273

. ml model d0 d0pro9 (regco = poll over65 gnp dist educ dale) / tau2 if c9>0

Number of obs = 21

Wald chi2(6) = 20.25

Prob > chi2 = 0.0025

Log likelihood = -55.524752

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0006483	.0011288	0.574	0.566	-.0015641	.0028607
over65	-.0373089	.0245416	-1.520	0.128	-.0854096	.0107918
gnp	.0000179	.0000273	0.656	0.512	-.0000355	.0000713
dist	3.58e-08	5.97e-07	0.060	0.952	-1.13e-06	1.21e-06
educ	-.0171846	.0051096	-3.363	0.001	-.0271992	-.00717
dale	.0427803	.0162099	2.639	0.008	.0110094	.0745512
_cons	-1.026349	.9723321	-1.056	0.291	-2.932085	.8793869
tau2						
_cons	.0267869	.01464	1.830	0.067	-.001907	.0554809

. ml model d0 d0pro10 (regco = poll over65 gnp dist educ dale) / tau2 if c10>0

Number of obs = 21

Wald chi2(6) = 10.30

Prob > chi2 = 0.1124

Log likelihood = -78.306758

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0004811	.0026426	-0.182	0.856	-.0056605	.0046983
over65	-.006102	.0500868	-0.122	0.903	-.1042704	.0920663
gnp	.000035	.0000664	0.527	0.598	-.0000951	.0001651
dist	7.60e-07	1.50e-06	0.506	0.613	-2.18e-06	3.70e-06
educ	-.0238628	.0097586	-2.445	0.014	-.0429893	-.0047362
dale	.0115143	.0309565	0.372	0.710	-.0491594	.072188
_cons	1.186873	1.840355	0.645	0.519	-2.420156	4.793902
tau2						
_cons	.2785972	.106044	2.627	0.009	.0707547	.4864396

. ml model d0 d0pro11 (regco = poll over65 gnp dist educ dale) / tau2 if c11>0

Number of obs = 21

Wald chi2(6) = 14.81

Prob > chi2 = 0.0218

Log likelihood = -65.109379

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005965	.0014003	-0.426	0.670	-.0033409	.0021479
over65	-.0993418	.0304978	-3.257	0.001	-.1591165	-.0395672
gnp	.0000449	.0000361	1.242	0.214	-.000026	.0001157
dist	-2.93e-07	8.98e-07	-0.326	0.744	-2.05e-06	1.47e-06
educ	-.0102513	.0063978	-1.602	0.109	-.0227909	.0022882
dale	.0418382	.0224232	1.866	0.062	-.0021105	.0857868
_cons	-.817001	1.259031	-0.649	0.516	-3.284656	1.650653
tau2						
_cons	.066666	.0336532	1.981	0.048	.0007069	.1326251

. ml model d0 d0pro12 (regco = poll over65 gnp dist educ dale) / tau2 if c12>0

Number of obs = 21
Wald chi2(6) = 12.89
Prob > chi2 = 0.0449

Log likelihood = -79.246571

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0025829	.0027126	0.952	0.341	-.0027336	.0078994
over65	.0050301	.048515	0.104	0.917	-.0900575	.1001177
gnp	.0000486	.0000628	0.774	0.439	-.0000745	.0001717
dist	8.53e-07	1.47e-06	0.580	0.562	-2.03e-06	3.74e-06
educ	-.0249288	.0094715	-2.632	0.008	-.0434926	-.0063649
dale	.0179483	.0281221	0.638	0.523	-.03717	.0730667
_cons	.1861631	1.686524	0.110	0.912	-3.119363	3.491689
tau2						
_cons	.2271802	.0945557	2.403	0.016	.0418544	.4125059

. ml model d0 d0pro13 (regco = poll over65 gnp dist educ dale) / tau2 if c13>0

Number of obs = 21
Wald chi2(6) = 5.88
Prob > chi2 = 0.4373

Log likelihood = -72.58448

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005516	.0019391	-0.284	0.776	-.0043521	.0032489
over65	-.0523958	.0356259	-1.471	0.141	-.1222213	.0174297
gnp	6.65e-06	.000042	0.158	0.874	-.0000756	.0000889
dist	5.67e-07	1.14e-06	0.495	0.620	-1.68e-06	2.81e-06
educ	-.0070712	.0066982	-1.056	0.291	-.0201995	.0060571
dale	.0186084	.0272949	0.682	0.495	-.0348887	.0721055
_cons	.0953264	1.575951	0.060	0.952	-2.99348	3.184133
tau2						
_cons	.1136924	.0552138	2.059	0.039	.0054753	.2219095

. ml model d0 d0pro14 (regco = poll over65 gnp dist educ dale) / tau2 if c14>0

Number of obs = 21
Wald chi2(6) = 10.17
Prob > chi2 = 0.1177

Log likelihood = -76.868233

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0003532	.002826	0.125	0.901	-.0051856	.005892
over65	-.0728673	.0500353	-1.456	0.145	-.1709348	.0252001
gnp	.0000774	.0000629	1.230	0.219	-.000046	.0002008
dist	3.28e-07	1.49e-06	0.220	0.826	-2.60e-06	3.25e-06
educ	-.0193666	.0095437	-2.029	0.042	-.0380719	-.0006614
dale	.0254045	.0301878	0.842	0.400	-.0337625	.0845716
_cons	.2222238	1.797065	0.124	0.902	-3.299959	3.744406
tau2						
_cons	.2701833	.1010497	2.674	0.008	.0721296	.468237

```
. ml model d0 d0pro15 (regco = poll over65 gnp dist educ dale ) / tau2 if c15>0
Number of obs = 21
Wald chi2(6) = 15.47
Prob > chi2 = 0.0169
Log likelihood = -60.680402
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0000821	.0011848	-0.069	0.945	-.0024043	.0022401
over65	-.0358928	.029808	-1.204	0.229	-.0943155	.0225298
gnp	-2.20e-07	.0000327	-0.007	0.995	-.0000643	.0000639
dist	4.25e-07	7.72e-07	0.551	0.581	-1.09e-06	1.94e-06
educ	-.019984	.0061018	-3.275	0.001	-.0319434	-.0080246
dale	.0482014	.0201695	2.390	0.017	.00867	.0877328
_cons	-1.009914	1.130495	-0.893	0.372	-3.225644	1.205815
tau2						
_cons	.04813	.025144	1.914	0.056	-.0011514	.0974113

```
. ml model d0 d0pro16 (regco = poll over65 gnp dist educ dale ) / tau2 if c16>0
Number of obs = 21
Wald chi2(6) = 10.87
Prob > chi2 = 0.0924
Log likelihood = -77.156455
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0030332	.0029945	1.013	0.311	-.0028359	.0089023
over65	.0032268	.0495565	0.065	0.948	-.0939022	.1003558
gnp	.0000229	.0000638	0.359	0.719	-.0001022	.0001481
dist	2.07e-06	1.62e-06	1.278	0.201	-1.11e-06	5.25e-06
educ	-.0164941	.0094415	-1.747	0.081	-.0349991	.002011
dale	-.0086707	.0316401	-0.274	0.784	-.0706842	.0533428
_cons	1.207074	1.875864	0.643	0.520	-2.469551	4.883699
tau2						
_cons	.2869027	.1010228	2.840	0.005	.0889017	.4849037

```
. ml model d0 d0pro17 (regco = poll over65 gnp dist educ dale ) / tau2 if c17>0
Number of obs = 21
Wald chi2(6) = 4.40
Prob > chi2 = 0.6232
Log likelihood = -64.621635
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0008413	.0018175	-0.463	0.643	-.0044037	.002721
over65	-.0346601	.0338917	-1.023	0.306	-.1010866	.0317664
gnp	3.18e-06	.0000423	0.075	0.940	-.0000797	.000086
dist	4.11e-07	1.05e-06	0.392	0.695	-1.65e-06	2.47e-06
educ	-.0057568	.0067782	-0.849	0.396	-.019042	.0075283
dale	.0211657	.0253077	0.836	0.403	-.0284365	.0707678
_cons	-.1742026	1.453949	-0.120	0.905	-3.023891	2.675486
tau2						
_cons	.0978215	.0407657	2.400	0.016	.0179221	.1777209

```
. ml model d0 d0pro18 (regco = poll over65 gnp dist educ dale ) / tau2 if c18>0
Number of obs = 21
Wald chi2(6) = 12.59
Prob > chi2 = 0.0500
Log likelihood = -77.387389
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0002704	.0028722	0.094	0.925	-.005359	.0058999
over65	.0038843	.0524899	0.074	0.941	-.0989941	.1067626
gnp	.0000223	.0000648	0.344	0.731	-.0001046	.0001492
dist	1.23e-06	1.50e-06	0.822	0.411	-1.71e-06	4.18e-06
educ	-.029914	.0098927	-3.024	0.002	-.0493032	-.0105247
dale	.0237677	.0305876	0.777	0.437	-.0361829	.0837182
_cons	.5886153	1.828479	0.322	0.748	-2.995137	4.172368

tau2						
_cons	.2761762	.1047791	2.636	0.008	.0708129	.4815394

```
. ml model d0 d0pro19 (regco = poll over65 gnp dist educ dale ) / tau2 if c19>0
Number of obs = 21
Wald chi2(6) = 11.46
Prob > chi2 = 0.0751
Log likelihood = -67.19496
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	-.0005875	.0017487	-0.336	0.737	-.0040149	.00284
over65	-.0782362	.037872	-2.066	0.039	-.1524639	-.0040085
gnp	.0000215	.0000444	0.484	0.628	-.0000655	.0001086
dist	6.35e-07	1.15e-06	0.554	0.580	-1.61e-06	2.88e-06
educ	-.0133911	.0073358	-1.825	0.068	-.027769	.0009868
dale	.0274538	.0290816	0.944	0.345	-.0295451	.0844526
_cons	.0974354	1.626762	0.060	0.952	-3.090959	3.28583

tau2						
_cons	.1258734	.0509559	2.470	0.014	.0260017	.2257451

```
. ml model d0 d0pro20 (regco = poll over65 gnp dist educ dale ) / tau2 if c20>0
Number of obs = 21
Wald chi2(6) = 10.88
Prob > chi2 = 0.0922
Log likelihood = -75.208808
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
poll	.0012632	.0026155	0.483	0.629	-.0038631	.0063894
over65	-.0289927	.0457436	-0.634	0.526	-.1186485	.0606631
gnp	.000063	.0000616	1.022	0.307	-.0000577	.0001837
dist	8.05e-07	1.49e-06	0.539	0.590	-2.12e-06	3.73e-06
educ	-.0163646	.0091929	-1.780	0.075	-.0343824	.0016532
dale	.006404	.0287015	0.223	0.823	-.0498499	.0626579
_cons	.7199767	1.685094	0.427	0.669	-2.582746	4.0227

tau2						
_cons	.2364317	.0879059	2.690	0.007	.0641392	.4087241

```
. ml model d0 d0pro21 (regco = poll over65 gnp dist educ dale ) / tau2 if c21>0
Number of obs = 21
Wald chi2(6) = 22.75
Prob > chi2 = 0.0009
Log likelihood = -59.812367
```

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]	
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poll	.0001983	.0013292	0.149	0.881	-.002407	.0028035
over65	-.0266413	.0272508	-0.978	0.328	-.080052	.0267693
gnp	.0000232	.0000298	0.777	0.437	-.0000353	.0000817
dist	-5.94e-07	6.93e-07	-0.857	0.391	-1.95e-06	7.65e-07
educ	-.0208296	.0054341	-3.833	0.000	-.0314802	-.010179
dale	.0535833	.0179492	2.985	0.003	.0184035	.0887631
_cons	-1.372144	1.061943	-1.292	0.196	-3.453514	.709226

tau2						
_cons	.0379386	.0208807	1.817	0.069	-.0029867	.078864

. ml model d0 d0pro22 (regco = poll over65 gnp dist educ dale) / tau2 if c22>0

Number of obs = 21

Wald chi2(6) = 9.97

Log likelihood = -79.175377

Prob > chi2 = 0.1259

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]
poll	-.0006727	.0026079	-0.258	0.796	-.0057842 .0044387
over65	-.0002732	.0512389	-0.005	0.996	-.1006996 .1001532
gnp	.0000286	.0000688	0.415	0.678	-.0001062 .0001634
dist	8.58e-07	1.56e-06	0.550	0.582	-2.20e-06 3.91e-06
educ	-.0235602	.0100124	-2.353	0.019	-.0431841 -.0039362
dale	.0106132	.0318721	0.333	0.739	-.051855 .0730814
_cons	1.223484	1.89204	0.647	0.518	-2.484846 4.931814

tau2						
_cons	.293957	.1117149	2.631	0.009	.0749997	.5129142

. ml model d0 d0pro23 (regco = poll over65 gnp dist educ dale) / tau2 if c23>0

Number of obs = 21

Wald chi2(6) = 10.66

Log likelihood = -67.786131

Prob > chi2 = 0.0995

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]
poll	-.0012702	.0015677	-0.810	0.418	-.0043429 .0018025
over65	-.0799726	.0324277	-2.466	0.014	-.1435298 -.0164154
gnp	.0000315	.0000409	0.771	0.441	-.0000486 .0001117
dist	-8.47e-08	1.09e-06	-0.077	0.938	-2.23e-06 2.06e-06
educ	-.012043	.0067827	-1.776	0.076	-.0253369 .001251
dale	.0357296	.0263827	1.354	0.176	-.0159796 .0874388
_cons	-.3160896	1.456501	-0.217	0.828	-3.170778 2.538599

tau2						
_cons	.0962779	.044003	2.188	0.029	.0100336	.1825222

. ml model d0 d0pro24 (regco = poll over65 gnp dist educ dale) / tau2 if c24>0

Number of obs = 21

Wald chi2(6) = 10.80

Log likelihood = -75.175338

Prob > chi2 = 0.0946

regco	Coef.	Std. Err.	z	P> z	[95% Conf. Interval]
poll	.0025545	.0027103	0.942	0.346	-.0027577 .0078666
over65	-.0359248	.0415632	-0.864	0.387	-.1173871 .0455376
gnp	.0000816	.0000603	1.353	0.176	-.0000366 .0001999
dist	2.19e-07	1.46e-06	0.149	0.881	-2.65e-06 3.09e-06
educ	-.0204541	.0090793	-2.253	0.024	-.0382491 -.002659

	dale	.0166926	.0280277	0.596	0.551	-.0382407	.0716259
	_cons	.2262885	1.675956	0.135	0.893	-3.058524	3.511101

tau2							
	_cons	.2254896	.0866677	2.602	0.009	.055624	.3953552
