Moolgavkar - Table of Risks

Table: **Relative Risks and 95% confidence intervals (CI) for the increase in mortality associated with a 10 μg/m^3 increase in fine PM from the various long-term studies of fine PM and mortality. These results illustrate the range of estimates from the various studies, but are not comprehensive in that not all published results are reported here because there are too many. The results depend on the population studied, the models used for analyses (most importantly, control of confounders), and the geographic region. It is clear from this range of results that no single coefficient can be picked out as being the best. Note that if the lower end of the 95% CI includes 1, then the estimated coefficient is not statistically significant. Statistically non-significant coefficients are highlighted in bold.**

Original Harvard Six Cities Study (Dockery et al., 1993)

All-cause mortality: 1.13 (1.03, 1.24) Cardiopulmonary mortality: 1.18 (1.06, 1.32)

Original ACS II Study (Pope et al., 1995)

All-cause mortality: 1.07 (1.04, 1.10) Cardiopulmonary mortality 1.12 (1.07, 1.17)

Laden Reanalysis of Harvard Six Cities Study (Laden et al., 2006)

All-cause mortality: 1.16 (1.07, 1.26) Cardiovascular mortality: 1.28 (1.13, 1.44)

All-cause mortality 1.14 (1.06, 1.22) - with fine PM in year of death as measure

HEI Reanalysis of ACS II Study (Krewski et al., 2000, summary table 6)

All-cause mortality: 1.07 (1.01, 1.13) Cardiopulmonary mortality: 1.11 (1.04, 1.19)

With SO2 included as confounder[1]:
All-cause mortality: 1.01 (0.98, 1.05) Cardiopulmonary mortality: 1.07 (1.01, 1.12)

First Pope Reanalysis of ACS II Study[2] (Pope et al., 2002)

All-cause mortality: 1.06 (1.02, 1.11) Cardiopulmonary mortality: 1.09 (1.03, 1.16)

---

[1] Note that inclusion of SO2 attenuates considerably the coefficient for fine PM and, for all-cause mortality renders it statistically insignificant. Despite this important finding of the Krewski reanalysis of the ACS II data, subsequent analyses of the ACS II data did not include SO2 as a confounder of fine PM effects.

[2] Although SO2 was found to be significantly associated with mortality, it was not included as a confounder in joint pollutant analyses with fine PM. See footnote 6 regarding association of SO2 with mortality.

Second Pope Reanalysis of ACS II Study[3] (Pope et al., 2004, table 4)

All cardiovascular disease: 1.12 (1.08, 1.15) - all-cause mortality not considered.

The ACS II California Study[4] (Jerrett et al., 2005)

With fine PM alone:
All-cause: 1.24 (1.11, 1.37) IHD[5]: 1.49 (1.20, 1.85) CP: 1.20 (1.04, 1.39)

With 44 individual-level covariates:
All-cause: 1.17 (1.05, 1.30) IHD: 1.39 (1.12, 1.73) **CP: 1.12 (0.97, 1.30)**

With "parsimonious" contextual covariates:
All-cause: 1.11 (0.99, 1.25) IHD: 1.25 (0.99, 1.59) CP: 1.07 (0.91, 1.26)

The HEI Update of the ACS II Study[6] (Krewski et al., 2009)

Nationwide Analyses:
All-cause: 1.03 (1.01, 1.04) IHD: 1.12 (1.09, 1.16). CP: 1.06 (1.04, 1.08)

Spatial Analyses in New York:
All-cause: 0.90 (0.70, 1.14) IHD: 1.59 (1.02, 2.50) **CP: 0.73 (0.50, 1.05)**

Spatial Analyses in Los Angeles:
All-cause: 1.14 (1.03, 1.27) IHD: 1.33 (1.08, 1.63) **CP 1.11 (0.97, 1.28)**

The ACS I Study in California (Enstrom, 2005)

All-cause mortality - No other cause of death was considered.

Over the period 1973-1982: 1.04 (1.01, 1.07)
Over the period 1983-2002: 1.00 (0.98, 1.02)
Over the period 1973-2002: 1.01 (0.99, 1.03)

[3] This paper did not consider SO2 at all. The paper also finds a statistically significant PROTECTIVE effect of fine PM on "other cardiovascular disease" mortality and also on respiratory disease mortality. Both these findings are contrary to expectation. Mortality from both these causes was significantly increased among smokers.
[4] Ozone was the only pollutant other than fine PM considered in this study. SO2 was ignored.
[5] IHD = Ischemic heart disease mortality; CP = Cardiopulmonary mortality.
[6] Other pollutants, including SO2, were considered in this analysis. SO2 was found to be significantly associated with all three causes of death. Despite this finding it was not considered in two-pollutant models with fine PM to test the hypothesis that SO2 was a confounder of fine PM effects. This same group of investigators had shown in their 2000 analysis that, in two-pollutant models, the effect of fine PM was greatly attenuated and became statistically insignificant.

Women's Health Initiative Study (Miller et al., 2007)

Cardiovascular disease mortality: 1.76[7] (1.25, 2.47) - No other cause of death was considered.

Teachers' Study[8] (Ostro et al. 2009, table 4)

All-cause mortality: 1.92[7] (1.50, 2.48); CP: 2.12[7] (1.52, 2.94); IHD: 3.38[7] (1.92, 5.96)

[7] Note that this estimated risk implies that fine PM pollution at contemporary levels in the U.S. is far more toxic than a pack a day smoking habit, a biologically highly implausible result.

[8] No gaseous co-pollutants were considered in these analyses.