Eur. J. Oncol., vol. 16, n. 1, pp. 9-13, 2011

Evaluation of the effects of reducing air pollution on human health

Stima degli effetti della riduzione dell'inquinamento atmosferico sulla salute umana

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Summary

Background. **The relationship between the long-term and short-term effects of atmospheric pollution has been assessed through many studies and shown to be linear, with no evidence for either a threshold effect or a short-term forward shift in mortality (harvesting).** ***Aim.*** **The evaluation of the effects of a reduction in air pollution on health.** ***Discussion.*** **In the city of Milan, 199 deaths are annually attributed to short-term and 1,178 to long-term air pollution effects, considering an annual average of 20 μg/m³ of particulate with a diameter less than 10 υm (PM_{10}). If the mean annual particulate matter concentrations in the city were reduced by just 10%, an immediate reduction of 31 deaths per year due to natural causes would be observed and after 3 or 4 years this reduction would be of 210 deaths per year for natural causes, 27 of which due to lung cancer, as a result of the reduced incidence.** ***Conclusions.*** **Even a small reduction in particulate atmospheric air pollution would be an important means of improving population health, as indicated by a**

Riassunto

Scenario. **La relazione sia tra gli effetti a breve termine sia tra gli effetti a lungo termine e l'inquinamento atmosferico risulta lineare e senza soglia. Inoltre l'inquinamento non anticipa decessi che si sarebbero comunque verificati ma rappresenta un effetto netto sulla salute.** ***Finalità.*** **La stima degli effetti della riduzione dell'inquinamento sulla salute e dei tempi in cui questi si realizzano.** ***Discussione.*** **Prendendo come esempio la città di Milano, 199 decessi per cause naturali sono attribuibili annualmente ad effetti a breve termine e 1.178 ad effetti a lungo termine rispetto ad una media annuale di 20 μg/m³ di particolato di diametro inferiore ai 10 υm (PM_{10}). Se l'inquinamento da particolato fine fosse ridotto del solo 10% vi sarebbe nell'immediato una riduzione di 31 decessi all'anno per cause naturali. Nel giro di 3-4 anni la riduzione si manifesterebbe anche sugli effetti a lungo termine con un calo atteso di 210 morti/anno di cui 27 per cancro polmonare.** ***Conclusioni.*** **Anche una modesta riduzione dell'inquinamento da particolato portereb-**

Received/Pervenuto 8.10.2010 - Accepted/Accettato 12.1.2011
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non-negligible reduction in mortality in general and in lung cancer mortality in particular. Eur. J. Oncol., 16 (1), 9-13, 2011

Key words: **air pollution, mortality, lung cancer, health benefit, estimation**

be a vantaggi importanti per la salute umana. Eur. J. Oncol., 16 (1), 9-13, 2011

Parole chiave: **inquinamento atmosferico, mortalità, cancro del polmone, beneficio per la salute, valutazione**

List of abbreviations:
AR: Attributable risk
ICD-9: 9th Revision of the International Classification of Diseases
PM: Particulate matter
PM_{10}: Particulate matter with a median aerodynamic diameter less than 10 micrometers
$PM_{2.5}$: Particulate matter with a median aerodynamic diameter less than 2.5 micrometers
RR: Relative risk

Introduction

Atmospheric pollution has short-term and long-term effects on human health. The former are adverse health events, such as deaths and hospital admissions; these effects are estimated considering the daily (occasionally hourly) variations in pollutant concentrations measured through monitoring stations. Long-term effects are principally assessed through prospective observational studies on groups of people (cohorts) exposed to varying levels of air pollution over long periods, in whom other factors − such as smoking, diet, workplace exposure, alcohol consumption and body-mass index − are taken into account to assess the net adverse health effects of air pollution.

Numerous gaseous, liquid and particulate pollutants are present in the atmosphere. However the concentration of breathable particles − those less than 10 μm in diameter (PM_{10}) − and the concentration of the finer fraction of breathable particles (less than 2.5 μm, $PM_{2.5}$) − are the most useful measures, since they have well established correlations with several adverse health events (1) and because plausible mechanistic pathways by which these particles cause damage have been described (2, 3). When breathed in, these particles elicit inflammation in the alveoli and bronchioles, and enter the circulatory system (4) where they have various adverse effects including increasing clotting (5).

Short-term effects

The short-term effects of particulate air pollution are estimated by analysing the relationship between daily variations in pollutant concentrations and the adverse events (deaths and hospital admissions for respiratory and cardiovascular conditions) that can now be easily monitored through electronically archived health data. In these studies other factors that can confound this relationship are considered, such as day of the week, temperature, and influenza outbreaks.

In this paper we will be concerned only with estimating the effects of a reduction in air pollution levels on mortality for non-violent causes. However the method can easily be extended to estimate the effects or reduced air pollution on hospital admissions for respiratory and cardiovascular conditions.

Table 1 shows the relative risks of short-term effects due to a 10 $\mu g/m^3$ increase in PM_{10} as estimated by the World Health Organization (6) based on the numerous studies on short-term effects available in the literature. The EpiAir study (7), on the relationship between short-term mortality and PM concentrations in 10 Italian cities over the period 2001-2005, found relative risks of death similar to those reported by the WHO. The data in Table 1 can also indicate the percentage increases in adverse events with increase in PM_{10} concentrations. For example a relative risk of 1.006 implies a 0.6% increase in the number of events relative to "baseline" per each 10 $\mu g/m^3$ increase of PM_{10}. As shown

Table 1 - World Health Organization estimates of relative risks of short-term adverse health effects per 10 μg/m³ increase in PM_{10} concentration, for all ages

Adverse health effect	ICD-9 code	Relative risk	95% confidence interval
Total mortality excluding violent deaths	000-799	1.006	1.004-1.008
Mortality for cardiovascular conditions	390-459	1.009	1.005-1.013
Mortality for respiratory conditions	460-519	1.013	1.005-1.020
Hospital admissions for cardiovascular conditions	390-429	1.003	1.000-1.006
Hospital admissions for respiratory conditions	460-519	1.006	1.002-1.011

by Ostro *et al.* (8) the mean annual number of short-term deaths associated with a given pollution level can be estimated simply by multiplying the difference between the mean annual PM concentration and an arbitrarily chosen reference level for PM and this coefficient, in this case 0.6%.

A study on air pollution in Italian cities over the period 2001 to 2005, found that the mean annual PM_{10} in Milan − as determined by 4 background monitoring stations and one on a busy arterial road − was 52 μg/m³ (9). Over the same period the mean annual number of deaths in the city due to natural causes was 10,347 (10).

If we choose the reference value for PM_{10} as the WHO target level of 20 μg/m³ (11), we can calculate the proportion of deaths that are a result of PM_{10} levels being above 20 μg/m³. The increased proportion of short-term non-violent mortality due to air pollution, compared to our chosen reference value of 20 μg/m³, would be the concentration difference in increments of 10 μg/m³ of PM_{10} multiplied by the percentage increment, i.e. (52-20) / 10 x 0.6 = 1.92%.

This 1.92% translates into 10,347 x 1.92/100 = 199 extra deaths per year, as short-term effect attributable to the fact that mean annual PM_{10} levels are 32 μg/m³ above the target level 20 μg/m³. This however is a conservative estimate because the data from time series do not take into account the effects of high PM_{10} levels for several successive days (12). Furthermore the estimate has considered only PM and not the other atmospheric pollutants (CO, SO_2, NO_x, etc.) that also contribute to mortality.

It has been argued that atmospheric pollution only anticipates death by a few days in persons who would have died anyway (i.e. belong to a susceptible pool). In this case, the observed mortality increase following increased pollution should drop in a few days and return to normal (the so-called "harvesting" effect). This does not happen and in fact mortality increases in the subsequent days, if pollutant concentrations remain high over many days (12, 13). Data from EpiAir project (7) also found no evidence for a supposed harvesting effect (F. Forastiere, personal communication, 25 November 2009). The lack of harvesting can be only explained if the risk pool never becomes depleted − i.e. if continuing high levels of atmospheric pollution worsen the health of less susceptible persons, recruiting them to the risk pool, and causing many of them to die some days later. The lack of harvesting therefore indicates that the short-term increase in mortality with reference to days of high pollution is mainly due to extra deaths − deaths advanced by months or by years, not days (12) − and which could be avoided if pollution levels were lower.

It has been shown that the relationship between short-term mortality and air pollution levels appears to be linear (13). The implication is that deaths will be reduced in proportion to any reduction in air pollution levels. If, for example the mean annual PM_{10} concentration in the city of Milan was reduced by a modest 10% − from 52 μg/m³ to 47 μg/m³ − there would be an annual mean of 31 fewer deaths. The following calculation shows: difference between 52 and 47 = 5 μg/m³ PM_{10}; given that the relative risk of short-term death is 1.006 per 10 μg/m³ increment in PM_{10} we have: 5 (reduction in PM_{10} concentration) /10 (percentage change per 10 μg/m³ difference) x 0.6 (percentage change) = 0.3%. In terms of number of deaths we have 0.3% x 10,347 = 31.

Long-term effects

Air pollution is also related to long-term adverse effects on health. These are mainly investigated through prospective studies where other determi-

nants including smoking, diet, and workplace exposure, are considered. Using the results of published prospective studies in this area, the WHO estimated relative risks of long-term adverse effects due to $PM_{2.5}$ (6), for people aged 30 yrs. and over. Some of these risks are shown in Table 2. It is noteworthy that the long-term effects of air pollution are an order of magnitude greater than the short-term effects shown in Table 1. According to the WHO (6), fine particulate matter ($PM_{2.5}$) represents 70% of PM_{10} in urban air and a study in the city of Milan also found that $PM_{2.5}$ represented the 75% of the PM_{10} (14).

We can estimate the long-term deaths per year due to $PM_{2.5}$ in Milan in an analogous way to the evaluation of short-term mortality.

Difference between 52 μg/m³ PM_{10} in Milan and WHO target level of 20 μg/m³ = 32 μg/m³; of this difference 70% is $PM_{2.5}$ i.e. 32 x 0.7 = 22.4 μg/m³ $PM_{2.5}$.

Given that the relative risk of long term death is 1.06 per 10 μg/m³ increment in $PM_{2.5}$ (Table 2) the estimated relative risk for Milan residents is 22.4 /10 x (0.06) +1 = 1.13 (relative to 20 μg/m³).

Since the entire population is exposed, the attributable risk (AR) due to the excess $PM_{2.5}$ is given by:

AR = (RR − 1)/RR = (1.13 − 1)/1.13 = 11.5%.

This 11.5% represents the proportion of deaths per year that are attributable to the fact that mean annual PM_{10} levels in Milan are 32 μg/m³ above the 20 μg/m³ target.

Since 1% of mortality due to natural causes occurs in people less than 30 years of age (10), 10,347 x 0.99 = 10,243 deaths per year occur in Milan residents over 30 years of age. Therefore 11.5% of 10,243 = 1,178 of those deaths occur as a long-term consequence of PM_{10} levels being above target.

The 2008 study of Schwartz *et al.* (15) showed that there is a linear relationship between air pollution levels and long-term effects. Using the WHO relative risk data, we can therefore estimate the annual reduction in number of long-term deaths due to a 10% reduction in particulate matter pollution in the city of Milan.

We suppose a reduction in mean annual PM_{10} from 52 μg/m³ to 47 μg/m³ which translates into

5 x 0.7 = 3.5 μg/m³ reduction in $PM_{2.5}$.

Thus 3.5 / 10 x 0.06 = 0.021 is the excess relative risk compared to a supposed PM_{10} level 47 μg/m³, which gives a relative risk of 1.021. Thus the AR = (1.021 – 1) 1.021 = 2%, indicating that reducing mean PM_{10} levels by 10% would reduce long-term mortality for natural causes in those people aged 30 yrs. and over by 2% per year or 10,243 x 0.02 = 210 deaths per year.

Now let us examine the effect of a 10% pollution reduction in Milan on lung cancer deaths.

Again we suppose a 3.5 μg/m³ reduction in $PM_{2.5}$, so that 3.5 / 10 x 0.08 (WHO excess risk for lung cancer mortality) = 0.028 is the excess relative risk, which translates to a relative risk of 1.028.

Hence the AR= (1.028 – 1) / 1.028 = 0.27%.

On average 997 lung cancer deaths per year in Milan are observed (7). We can assume that 997 x 0.027 = 27 lung cancer deaths per year would be avoided if air pollution were reduced by 10%.

Long-term effects are those resulting from the accumulation of tissue damage over many years. The damage slowly progresses to the point where it manifests clinically as a disease. Sub-clinical disease can usually be detected before it becomes clinically evident: a good example would be the progressive increase in thickness in atheromatic/atherosclerotic plaques (16), that correlates with increasing likelihood of acute cardiovascular events. If the progression of lesions could be slowed, one might expect an observable reduction in the incidence of clinical illness in a short time. The paper of Schwartz *et al.* (15) modelled air pollution as a time-varying covariate in an extended follow-up study of the Harvard Six Cities Study, controlling for smoking, body mass index, and other covariates. Modelling showed that non-violent deaths and lung cancer associated with fine particle exposure mainly occurred within 1-3 years of exposure, implying that a reduction in air pollution can be expected to produce improvements on a short time scale.

Table 2 - World Health Organization estimates of relative risks of long-term adverse health effects per 10 μg/m³ increment in $PM_{2.5}$ concentration, for all ages (for deaths of people aged 30 yrs. and over)

Adverse health effect	Relative risk	95% confidence interval
Deaths for non violent causes	1.06	1.02-1.11
Lung cancer	1.08	1.01-1.16
Heart attack	1.18	1.14-1.23

Conversely, failure to reduce air pollution results in deaths that could have been prevented, even though the damage ultimately responsible accumulated over many years: the damage may not have developed into lethal disease if air pollution had been reduced over the 3-4 years preceding death.

We conclude by emphasising the importance of traffic as contributor to particulate matter (14) and the considerable differences between PM concentrations measured on major urban roads and those measured on urban background monitoring stations (17). It is also noteworthy that circadian peaks in PM are closely related to traffic peaks (18). These findings indicate that atmospheric PM and its precursors remain fairly close to the point of emission, suggesting that local measures to reduce traffic emissions will result in rapid reductions in PM population exposure, with positive effects on the health of the city population and on the costs of health care, in a relatively short timeframe.

References

1. Chen Y, Craig L, Krewski D. Air quality risk assessment and management. J Toxicol Env Health, Part A 2008; 71 (9&19): 24-39.
2. WHO Regional Office for Europe. Air quality guidelines for Europe, second edition. Copenhagen. WHO Regional Office for Europe. WHO Regional Publications, European Series No 91, 2000.
3. EPA. Air quality criteria for particulate matter. Research Triangle Park, NC, US Environmental Protection Agency, Office of Air Quality Planning and Standards, 2004.
4. Nemmar A, Hoet PHM, Vanquickeborne B, *et al.* Passage of inhaled particles into the blood circulation in humans. Circulation 2002; 150: 411-4.
5. Brook R, Franklin B, Cascio W, *et al*. Air pollution and cardiovascular disease. A statement for health care professionals from the expert panel on population and prevention science of the American Heart Association. Circulation 2004; 109: 2655-71.
6. Martuzzi M, Mitis F, Iavarone I, *et al*. Health impact of PM10 and ozone in 13 Italian cities. 2006; World Health Organization, Copenhagen.
7. Strafoggia M, Faustini A, Rognoni M, *et al.* Inquinamento atmosferico e mortalità in dieci città italiane. Risultati del progetto EpiAir. E&P 2009; Suppl. 1: 65.
8. Ostro B, Chestnut L. Assessing the health benefits of reducing particulate matter air pollution in United States. Env Res 1998; A76: 94-106.
9. Berti G, Chiusolo M, Grechi D, *et al*. Indicatori ambientali in dieci città italiane (2001-2005): i dati di qualità dell'aria per la sorveglianza epidemiologica. E&P 2009; Suppl. 1: 13-26.
10. Galassi C, Faustini A, Colais P, *et al*. I dati sanitari nel progetto EpiAir. E&P 2009; Suppl. 1: 43-51.
11. WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulphur dioxide. Global update 2005. WHO 2005, Geneva.
12. Schwartz J. Is there harvesting in the association of airborne particles with daily deaths and hospital admission? Epidemiology 2001; 12 (1): 55-61.
13. Schwartz J, Zanobetti A. Using meta-smoothing to estimate dose-response trends across multiple studies, with application to air pollution and daily death. Epidemiology 2000; 11: 666-72.
14. Lonati G, Giugliano M. Size distribution of atmospheric particulate matter at traffic exposed sites in the urban area of Milan (Italy). Atmospheric Env 2006; 40: S264-S274.
15. Schwartz J, Couli B, Lden F, *et al*. The effect of dose and timing of dose on the association between airborne particles and survival. Env Health Persp 2008; 116: 64-9.
16. Kunzli N, Jerret M, Garcia-Esteban R, *et al*. Ambient air pollution and the progression of atheroslerosis in adults PLoS ONE 2010; 5(2): e9096.
17. Namdeo A, Bell MC. Characteristics and health implications of fine and coarse particulates at roadside, urban background and rural sites in UK. Env Int 2005; 31: 565-73.
18. Gomiscek B, Hauck H, Stopper S, *et al*. Spatial and temporal variation of PM1, PM2.5, PM10 and particle number concentration during the AUPHEP project. Atmospheric Env 2004; 38: 3917-34.