# Setting Health-based Air Quality Standards

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## 1 Introduction

Health-based air quality standards form one of the cornerstones of the air quality management process. Progress in improving air quality without explicit air quality standards is possible, as occurred in the UK between the 1950s and 1980s, but in any modern rational system of air quality management, some form of health-based objective is needed, and generally these are referred to as air quality standards.

To set air quality standards in context it is necessary to understand the entire air quality management process (see Figure 1); the reader is referred to recent articles by Middleton[1] and Lloyd.[2] There are basically three major facets:

(a) *Monitoring and public information.* This provides the necessary knowledge of current air quality and can inform people of periods when some form of preventative or protective activity is desirable at the individual, corporate or community level. Provision of data to the public also serves a purpose in generating informed public pressure for better air quality.
(b) *Air quality standards.* These are benchmarks of acceptability against which the monitoring data may be judged. They can serve as long-term objectives which control strategies are designed to meet or, as in the case of European Limit Values, can provide firm legislative ceilings which, if exceeded, can result in legal action and the requirement for immediate remedial measures.
(c) *Control policy design and implementation.* If air quality standards represent the desired endpoint, then this is the means of achieving it. Normally some form of numerical model is used to determine the most cost-effective means of reducing emissions so as to meet air quality standards, and subsequently legal controls are applied which are designed to bring about the necessary

[1] D. R. Middleton, in *Air Quality Management*, Issues in Environmental Science & Technology, vol. 8, ed. R. E. Hester and R. M. Harrison, Royal Society of Chemistry, Cambridge, 1997.
[2] A. C. Lloyd, in *Air Quality Management*, Issues in Environmental Science & Technology, vol. 8, ed. R. E. Hester and R. M. Harrison, Royal Society of Chemistry, Cambridge, 1997.

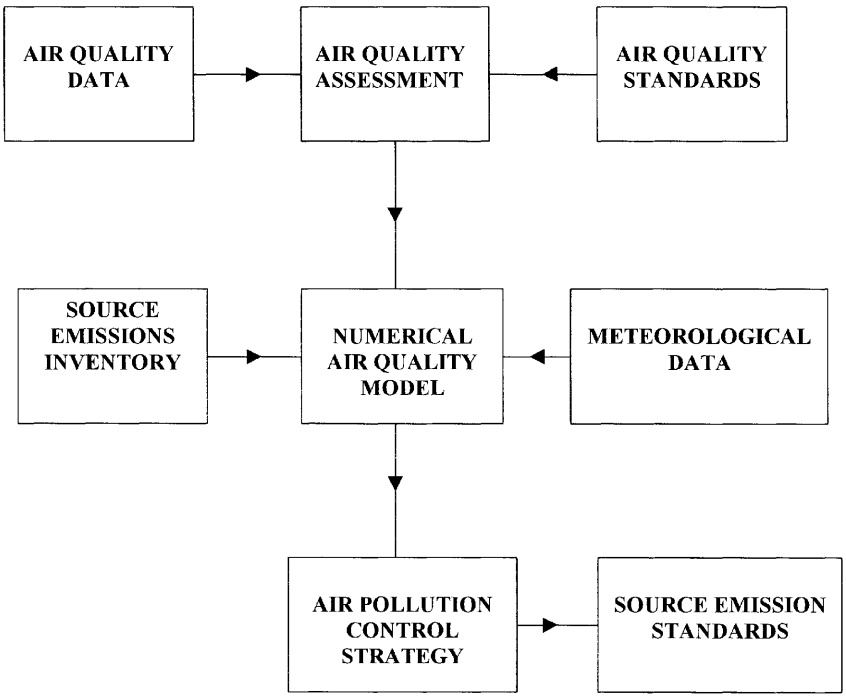


**Figure 1** Schematic of the air quality management process

emissions reductions. The European Auto-Oil Programme described by Skouloudis[3] is an excellent example of this process in action.

Thus, air quality standards are the benchmark of acceptability of air quality. A more formal definition might be the following:

> An air quality standard is a concentration of an air pollutant below which effects on human health are expected to be zero or negligibly small at a population level.

It must be recognized that air quality standards are designed to protect the health of populations rather than of every individual. Thus, highly susceptible individuals, *e.g.* brittle (very severe) asthmatics or those who contract cancer as a result of exposure to very low concentrations of environmental chemicals, may suffer serious personal consequences even when concentrations are within air quality guidelines. In general, air quality standards do seek to protect sensitive individuals such as normal asthmatics, but not the most sensitive members of the population; as will be discussed later, it is impracticable to set standards for genotoxic carcinogens which guarantee that there will be zero risk of consequent cancer cases.

There is no universally agreed distinction between the terms *standard*, *guideline* and *objective* when applied to air quality. In the UK the term 'standard' is used in

[3] A. N. Skouloudis, in *Air Quality Management*, Issues in Environmental Science & Technology, vol. 8, ed. R. E. Hester and R. M. Harrison, Royal Society of Chemistry, Cambridge, 1997.

**Table 1** Air quality standards recommended by EPAQS

| *Pollutant* | *Concentration* | *Averaging period* |
|---|---|---|
| Benzene | 5 ppb | 1 year running mean |
| 1,3-Butadiene | 1 ppb | 1 year running mean |
| Sulfur dioxide | 100 ppb | 15 minutes |
| Nitrogen dioxide | 150 ppb | 1 hour |
| Ozone | 50 ppb | 8 hour running mean |
| Carbon monoxide | 10 ppm | 8 hour running mean |
| $PM_{10}$ | 50 $\mu g\,m^{-3}$ | 24 hour running mean |

the sense defined above, whilst specific objectives are shorter-term policy objectives to be met *en route* to long-term compliance with the standard. The World Health Organization, however, produces what it refers to as guidelines which nonetheless conform to the above definition of a standard. The WHO uses this term in order to convey the idea that individual countries should set their own standards based on the WHO guidelines, but taking regard of individual socio-economic considerations. Thus, a national standard could be greater than or less than the corresponding WHO guideline.

## 2 Sources of Air Quality Standards

There are now a number of authoritative independent sources of air quality standards. This article will make no attempt to review exhaustively or intercompare such standards, and indeed, the majority of the discussion will centre around the work of the UK Expert Panel on Air Quality Standards (EPAQS) and the WHO Working Group which produced the revised WHO Air Quality Guidelines for Europe. A summary of the EPAQS recommendations[4–10] available at the time of writing is presented in Table 1 and a selection of the WHO recommendations,[11] omitting those for indoor air and ecotoxic effects, is presented in Table 2. Other sources of air quality standards are the European Commission and the US Environmental Protection Agency. These differ from the EPAQS and WHO recommendations in one important regard: both can have legal force.

The standards recommended by EPAQS (see Table 1) have been adopted by the UK Government as long-term benchmarks for air quality. In some instances they have been translated into Objectives in the National Air Quality Strategy, which take into account costs and benefits and which it is intended to achieve by the year 2005. Objectives for seven pollutants have been set down in regulation (The Air Quality Regulations 1997), which triggers a duty, set down in Part IV of

[4] Expert Panel on Air Quality Standards, *Benzene*, HMSO, London, 1994.
[5] Expert Panel on Air Quality Standards, *Ozone*, HMSO, London, 1994.
[6] Expert Panel on Air Quality Standards, *Carbon Monoxide*, HMSO, London, 1994.
[7] Expert Panel on Air Quality Standards, *1,3-Butadiene*, HMSO, London, 1994.
[8] Expert Panel on Air Quality Standards, *Sulphur Dioxide*, HMSO, London, 1995.
[9] Expert Panel on Air Quality Standards, *Particles*, HMSO, London, 1995.
[10] Expert Panel on Air Quality Standards, *Nitrogen Dioxide*, HMSO, London, 1996.
[11] World Health Organization, *Revised Air Quality Guidelines for Europe*, WHO European Office, Copenhagen, 1998.

**Table 2** Summary of objectives in the UK National Air Quality Strategy[12]

| *Pollutant* | *Standard* | | *Objective—to be achieved by 2005* |
|---|---|---|---|
| | *concentration* | *measured as* | |
| Benzene | 5 ppb | Running annual mean | 5 ppb |
| 1,3-Butadiene | 1 ppb | Running annual mean | 1 ppb |
| Carbon monoxide | 10 ppm | Running 8-hour mean | 10 ppm |
| Lead | 0.5 $\mu g\,m^{-3}$ | Annual mean | 0.5 $\mu g\,m^{-3}$ |
| Nitrogen dioxide | 150 ppb | 1 hour mean | 150 ppb, hourly mean[a] |
| | 21 ppb | Annual mean | 21 ppb, annual mean[a] |
| Ozone | 50 ppb | Running 8-hour mean | 50 ppb, measured as the 97th percentile[a] |
| Fine particles | 50 $\mu g\,m^{-3}$ | Running 24-hour mean | 50 $\mu g\,m^{-3}$ measured as the 99th percentile[a] |
| Sulfur dioxide | 100 ppb | 15 minute mean | 100 ppb measured as the 99.9th percentile[a] |

[a]These objectives are to be regarded as provisional.

the Environment Act 1995, upon local government to address issues of unacceptable air quality, as defined by the air quality objectives, within their boundaries. There are, however, no penalties for exceedence. The Standards and Objectives adopted in the March 1997 UK National Air Quality Strategy,[12] now under review, appear in Table 2. For some pollutants, such as nitrogen dioxide and benzene, the Objective is identical to the Standard. For others, however, a percentile compliance varying from 97% for ozone to 99.9% for sulfur dioxide is specified. Thus, some exceedences of the Standard are envisaged even in the year 2005.

The philosophy used by EPAQS to recommend standards has been very similar to that used by the WHO in setting their air quality guidelines for Europe. The first set of European guidelines was published in a book entitled 'Air Quality Guidelines for Europe' in 1987 and have proved to be extremely influential. The guidelines for the 'irritant' air pollutants, carbon monoxide and trace metals were designed to protect even sensitive members of the population and to incorporate an additional margin of safety, and this ethos is also fundamental to the recent revisions.[11] This is also the philosophy behind EPAQS standards. The major difference between WHO recommendations and EPAQS is that for the non-threshold pollutants, such as the genotoxic carcinogens and $PM_{10}$, the WHO cites exposure–response gradients rather than recommending a guideline. It is therefore left to individual governments to determine national standards

[12] Department of the Environment, *The United Kingdom National Air Quality Strategy*, Stationery Office, London, 1997.

**Table 3** Revised WHO air quality guidelines for Europe (second edition)

| *Substance* | *Guideline value* | *Averaging time* |
|---|---|---|
| Classical air pollutants | | |
| Carbon monoxide | 100 $mg\,m^{-3}$ | 15 min |
| | 60 $mg\,m^{-3}$ | 30 min |
| | 30 $mg\,m^{-3}$ | 1 hour |
| | 10 $mg\,m^{-3}$ | 8 hour |
| Ozone | 120 $\mu g\,m^{-3}$ | 8 hour |
| Nitrogen dioxide | 200 $\mu g\,m^{-3}$ | 1 hour |
| | 40 $\mu g\,m^{-3}$ | Annual |
| Sulfur dioxide | 500 $\mu g\,m^{-3}$ | 10 min |
| | 125 $\mu g\,m^{-3}$ | 24 hour |
| | 50 $\mu g\,m^{-3}$ | Annual |
| Particulate matter | Exposure–response | |
| Inorganic pollutants | | |
| Arsenic | $1.5 \times 10^{3}\,(\mu g\,m^{-3})^{-1}$ | UR[a]/lifetime |
| Cadmium | 5 $ng\,m^{-3}$ | Annual |
| Chromium ($Cr^{VI}$) | $4 \times 10^{-2}\,(\mu g\,m^{-3})^{-1}$ | UR[a]/lifetime |
| Fluoride | No guideline | |
| Lead | 0.5 $\mu g\,m^{-3}$ | Annual |
| Manganese | 0.15 $\mu g\,m^{-3}$ | Annual |
| Mercury | 1.0 $\mu g\,m^{-3}$ | Annual |
| Nickel | $3.8 \times 10^{-4}\,(\mu g\,m^{-3})^{-1}$ | UR[a]/lifetime |
| Platinum | No guideline | |
| Organic pollutants | | |
| Benzene | $6 \times 10^{-6}\,(\mu g\,m^{-3})^{-1}$ | UR[a]/lifetime |
| 1,3-Butadiene | No guideline | |
| Dichloromethane | 3 $mg\,m^{-3}$ | 24 hour |
| Formaldehyde | 0.1 $mg\,m^{-3}$ | 30 min |
| PAH (BaP) | $8.7 \times 10^{-5}\,(ng\,m^{-3})^{-1}$ | UR[a]/lifetime |
| Styrene | 0.26 $mg\,m^{-3}$ | 1 week |
| Tetrachloroethylene | 0.25 $mg\,m^{-3}$ | 24 hour |
| Toluene | 0.26 $mg\,m^{-3}$ | 1 week |
| Trichloroethylene | $4.3 \times 10^{-7}\,(\mu g\,m^{-3})^{-1}$ | UR[a]/lifetime |

[a]UR = Unit Risk (see text).

suited to local circumstances. Further discussion of the unit risk value is provided later in this article. In the last year or two the WHO European office, using advisers drawn internationally, has revised the air quality guidelines for Europe, and at the time of writing a second edition is in the press. The guidelines listed in Table 3 are the outcome of that revision process.

In contrast, the European Union has limit and guide values for a number of pollutants. The limit values have legal force and unless a specific derogation is granted, member states are expected to ensure that air quality complies with the limit values embodied in the various air quality Directives. Currently, Directive limit values are in force for sulfur dioxide, smoke/particulate matter, nitrogen dioxide and lead, and public information thresholds and alert levels are set for ozone. The EU has, however, recently adopted a new framework Directive on

**Table 4** Substances for which EU air quality standards are currently proposed

| |
|---|
| Sulfur dioxide |
| Nitrogen dioxide |
| Fine particulate matter |
| Suspended particulate matter |
| Lead |
| Ozone as at present (Directive 92/72/EC) |
| Benzene |
| Carbon monoxide |
| Polyaromatic hydrocarbons |
| Cadmium |
| Arsenic |
| Nickel |
| Mercury |

Ambient Air Quality Assessment and Management (96/62/EC) under which individual Daughter Directives will be set, dealing with monitoring protocols and air quality standards for a range of specific pollutants. The current list of substances for which EU air quality standards are to be developed is given in Table 4. At the time of writing, formal proposals have been published for the first five substances on the list (fine and suspended particulate matter are taken together as PM), and these appear in Table 5. Working groups are currently developing further proposals on carbon monoxide, benzene and ozone. It must be emphasized that any proposed values may well be changed before entry into community law.

In the US, ambient air quality standards are set by the US EPA and have legal force, although, in general, policy is directed towards long-term achievement of air quality standards rather than immediate prosecution of infringements. The current US EPA standards appear in Table 6. These differ somewhat from the EPAQS and WHO standards, at least in part because of the extensive consultation of industry and other pressure groups which goes on whilst setting the standards. The economic costs and benefits of compliance are not considered directly in the standards setting process, but an economic analysis is also conducted by the EPA (see chapter by M. Lippmann in this volume). In the UK, consideration of costs and benefits and consultation in the industry and pressure groups takes place when the Government publishes its response to the recommendations of EPAQS, and any draft national objectives that may be developed from those standards.

## 3 Setting Air Quality Standards

The essence of the process of setting air quality standards is easily explained. Maynard[13] describes the sequence in the following way:

(a) Understand the exposure–response relationship of the pollutant in question
(b) Decide on an acceptable level of effects

[13] R. L. Maynard, in *Setting and Managing Standards for Air Quality*, Cambridge Environmental Initiative Professional Seminar Series, Cambridge, 1994.

**Table 5** Health-based limit values proposed by the European Commission

| *Pollutant* | *Limit value* | *Margin of tolerance* | *Target date* |
|---|---|---|---|
| $SO_2$ | 350 $\mu g\,m^{-3}$ (1-hour average) not to be exceeded more than 24 times a year | 43% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2005 | 1 January 2005 |
| | 125 $\mu g\,m^{-3}$ (24-hour average) not to be exceeded more than 3 times a year | None | 1 January 2005 |
| $NO_2$ | 200 $\mu g\,m^{-3}$ (1-hour average) not to be exceeded more than 8 times a year | 50% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2010 | 1 January 2010 |
| | 40 $\mu g\,m^{-3}$ (annual average) | 50% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2010 | 1 January 2010 |
| $PM_{10}$ (Stage 1) | 50 $\mu g\,m^{-3}$ (24-hour average) not to be exceeded more than 25 times a year | 50% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2005 | 1 January 2005 |
| | 30 $\mu g\,m^{-3}$ (annual average) | 50% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2005 | 1 January 2005 |
| $PM_{10}$ (Stage 2) | 50 $\mu g\,m^{-3}$ (24-hour average) not to be exceeded more than 7 times a year | None | 1 January 2010 |
| | 20 $\mu g\,m^{-3}$ (annual average) | None | 1 January 2010 |
| Lead | 0.5 $\mu g\,m^{-3}$ (annual average) | 100% on commencement of Directive, falling linearly to 0% between 1.1.2001 and 1.1.2005 | 1 January 2005 |

**Table 6** US ambient air quality standards

| *Pollutant* | *Measurement period* | *Concentration* |
|---|---|---|
| Sulfur dioxide | Annual arithmetic mean | 30 ppb (80 $\mu g\,m^{-3}$) |
| | 24 hour average (not to be exceeded more than once per year) | 140 ppb (365 $\mu g\,m^{-3}$) |
| Particulate: $PM_{10}$ | 24 hour average (99%ile) | 150 $\mu g\,m^{-3}$ |
| $PM_{10}$ | Annual arithmetic mean | 50 $\mu g\,m^{-3}$ |
| $PM_{2.5}$ | 24 hour average (98%ile) | 65 $\mu g\,m^{-3}$ |
| $PM_{2.5}$ | Annual arithmetic mean | 15 $\mu g\,m^{-3}$ |
| Carbon monoxide | 8 hour average (not to be exceeded more than once per year) | 9 ppm (10 000 $\mu g\,m^{-3}$) |
| | 1 hour average (not to be exceeded more than once per year) | 35 ppm (40 000 $\mu g\,m^{-3}$) |
| Ozone | 8 hour average (annual fourth highest daily maximum) | 80 ppb |
| | 1 hour average (not to be exceeded more than once per year) | 120 ppb (235 $\mu g\,m^{-3}$) |
| Nitrogen dioxide | Annual arithmetic mean | 53 ppb (100s $\mu g\,m^{-3}$) |
| Lead | Maximum arithmetic mean averaged over a calendar quarter | 1.5 $\mu g\,m^{-3}$ |

(c) Set standard so that effects do not exceed those specified as acceptable

Clearly this process contains elements which will be interpreted differently by different standard setting bodies. The understanding of the exposure–response relationships has notably varied between standard setting bodies for some pollutants. This is usually because different degrees of weight are attached to the importance of the various published studies. Unfortunately, as will be described shortly, the base of data upon which exposure–response relationships may be established is often weak and sometimes contradictory, and hence different views may be taken according to the weight put on particular studies. For example, in the case of nitrogen dioxide, whilst population-based epidemiological studies have demonstrated effects at rather modest levels of exposure to $NO_2$, controlled exposure studies in the laboratory have shown that much higher concentrations of $NO_2$ are needed to elicit a response in these circumstances. There are various possible interpretations of this apparent contradiction, amongst them being that the chamber studies have been poorly designed and have failed to recognize important health outcomes, or, on the other hand, the population-based epidemiological studies have been subject to confounding from the co-variation

of nitrogen dioxide with other pollutants such as particulate matter, and therefore the effects which they attribute to nitrogen dioxide are in fact the result of exposures to other pollutants. This point is elaborated on in the next section.

Furthermore, even if there is a clear understanding of the exposure–response relationships, then different panels will take a different view of what is an acceptable level of effects. Maynard[13] comments that 'on an international scale, little agreement on what constitutes acceptable effects has been reached: opinions vary from no effect to effects significantly less than those produced by other more uncontrollable environmental factors such as variations in temperature and epidemics of mild infections such as colds'. The acceptability will, in the eyes of some, also take account of costs as well as benefits and clearly the judgements made are highly individual and subjective overall.

## 4 Understanding Exposure–Response Relationships

The base of data available for understanding exposure–response relationships comes from four major sources. These are essentially complementary and, in an ideal world, information would be available from all four, and when integrated would give a coherent whole. Often this is not the case.

*Controlled human exposure studies* in the laboratory have the advantage of offering good definition of acute effects and precise knowledge of exposure concentrations. They are excellent for allowing identification of the effects of a single pollutant, and particularly good for pollutants which act as respiratory tract irritants and therefore elicit changes in lung function over relatively short time periods which are capable of being measured. Thus, for a small range of pollutants eliciting modest reversible effects, chamber studies provide excellent data which have proved useful in setting standards for pollutants such as nitrogen dioxide and sulfur dioxide. Whilst it should be possible to study simple mixtures in chambers, rather little work has been conducted with mixtures, and indeed, it is not possible to simulate the full complexity of an urban air pollutant mix. The measures of health impact used in chamber studies are generally tests of lung function such as $FEV_1$ (Forced Expiratory Volume in 1 second: the volume of air expired forcibly in one second) or FVC (Forced Vital Capacity: the total volume of air which can be expelled forcibly), but the health significance of small changes in these parameters when reversible, either for the individual or for the population as a whole, is extremely difficult to judge. Additionally, biochemical tests are now available which can demonstrate the initiation of inflammatory processes, but again the long-term health significance of such inflammation for the individual is very difficult to determine, and hence for studies showing an exposure–response gradient, it is very hard to know at what point on that curve to determine an acceptable level of effect. A useful application of chamber studies has been in research on the interaction between gaseous air pollutants and airborne allergens (*e.g.* grass pollen or house dust mite allergen), a topic very hard to study through epidemiology.

Secondly, there are *epidemiological studies in the general population.* These have the great advantage of offering definitions of effects of real pollutant mixtures on whole populations if confounding factors can be adequately controlled. The

latter can be a major problem, since such apparently routine variables as air temperature can have major impacts on such profound outcomes as mortality. Nonetheless, epidemiological studies have tended to be the backbone of the standard setting process, proving extremely valuable for irritant pollutants and particles. Studies in which day-to-day changes in airborne pollutant loading are related to health service data such as hospital admissions have been especially persuasive. However, there are persistent doubts in some cases whether epidemiology can correctly identify the harmful pollutant in a complex urban mixture, or indeed whether it is the mix itself which is responsible for the observed response. Many of the important recent epidemiological studies have used a time series design. These involve relating measurements of daily average air pollution collected over a period of a year or more to a health outcome such as mortality or hospital admission on the same day or lagged by anything up to three days after the air pollution event. The health data are controlled for factors such as season and temperature referred to above, which can have major impacts on health, and when all such controls have been applied, the resultant day-to-day changes in morbidity or mortality are related to the air pollution measurements. These studies have proved extremely powerful in understanding the influence of air pollutants, especially particulate matter.

Nowadays most urban air pollutants have a common source in road traffic and since the main controls on concentrations are the rate of emission and the prevailing weather, traffic-generated pollutants tend to vary in much the same way from day-to-day. Therefore, a day with a high concentration of carbon monoxide will probably also have a high concentration of nitrogen dioxide and particulate matter. Consequently, disentangling the influences of the different pollutants when they vary from day-to-day in such a similar manner can be extremely difficult. Not all pollutants co-vary in this manner. For example, ozone tends to correlate positively with nitrogen dioxide in the summer but inversely in the winter months, hence making separation of the effects of the two pollutants much more straightforward when full annual data are utilized. The alternative cross-sectional cohort study design, as was used in the Harvard Six Cities study discussed later, relates measurements of air pollutant concentrations in different cities to the rates of mortality or morbidity in those cities. The Six Cities study was conducted using a time period over which traffic-generated pollutants were not as dominant as currently and therefore some separation of the effects of different pollutants was more straightforward.

*Studies of occupationally exposed workers* are particularly valuable in the case of chemical carcinogens, where frequently they provide the only source of real world data. In general, the effects of airborne chemical carcinogens are insufficiently large to be demonstrated through epidemioloical studies of the general population, and ethical considerations obviously rule out the use of chamber studies for chemical carcinogens. The residual problem with occupational studies is that the concentrations of air pollutants encountered far exceed those to which the general population are exposed, and extrapolation to lower concentrations is very much an act of faith. However, for genotoxic carcinogens there are reasonable grounds for believing that the dose–response function is approximately linear without any threshold and an assumption of this kind is

**Table 7** Categories of air pollutant toxic action

| *Category* | *Pollutant* | *Threshold* |
|---|---|---|
| Irritants | Ozone, nitrogen dioxide, sulfur dioxide | May show a threshold |
| Asphyxiant | Carbon monoxide | May show a threshold |
| Genotoxic carcinogens | Benzene, 1,3-butadiene | Not believed to have a threshold |
| Enzyme inhibitors | Lead | May show a threshold |
| Mechanism uncertain | Particles | No threshold demonstrable at population level |

generally inherent in the standard setting process, although for bodies such as the US EPA, who have taken quantitative risk assessment to a far greater extent in the standard setting process than other organizations, the use of other more sophisticated models is routine.

Finally, data from *animal studies* can occasionally prove useful. Such information usually has a value in demonstrating mechanisms rather than illuminating exposure–response functions. It may possibly throw light on the relative toxicity of chemical carcinogens for which human data are very sparse, but otherwise animal data have little direct use in the standard setting process.

## 5 Determining an Acceptable Level of Effects

This can often prove more difficult than defining exposure–response relationships. Table 7 attempts to categorize common air pollutants according to their mechanism of action. For each mechanism of action, different considerations come into play in looking for an acceptable level of effect.

Taking first the so-called 'irritant' pollutants, the approach taken by the WHO and EPAQS has been to determine a lowest observable effect level, taking into account sensitive groups where data are available, incorporation of a margin of safety to allow for more sensitive subjects than can take part in chamber studies and to set a standard which should be 'safe' for all groups. Since it is assumed that at least at an individual level the irritant pollutants have a threshold, it should be possible to set a standard protective of the kinds of individuals who took part in chamber studies, as well as some more sensitive groups through the incorporation of the margin of safety. An example of this approach is sulfur dioxide, where the key effect is that of bronchoconstriction in asthmatics. The lowest observable effect level from chamber studies is approximately 200 ppb over a few minutes. EPAQS[8] recommended to a standard of 100 ppb averaged over 15 minutes to incorporate a margin of safety and to allow for concentrations above this level occurring for periods of less than 15 minutes.

A second example is that of ozone, where the key effect is a reduction in lung capacity. EPAQS[5] considered the lowest observable effect level to be 80 ppb exposure over 6.6 hours, and incorporation of a margin of safety led to a standard of 50 ppb over eight hours. This approach assumes that there is a threshold for injury by ozone, although some have argued that this may not be the case, and indeed, some epidemiological data published after EPAQS made its recommen-

dation suggest there is not. The concept of there being no threshold for adverse effects of ozone upon humans raises a very interesting paradox. Ozone concentrations are generally higher in rural than urban areas, and current daytime rural concentrations in the northern hemisphere typically show hourly averages of up to 50 ppb and eight hourly averages of 30–40 ppb in the absence of any severe pollution event.[14] This background of ozone arises from two sources. Around half of it comes from downward mixing of stratospheric ozone and is therefore wholly natural. The other half is the result of perturbation of the lower atmosphere by nitrogen oxides from anthropogenic combustion processes, which interact with methane and carbon monoxide to form ozone in sunshine. There is good reason to think that the human species and its antecedents in the evolutionary chain evolved in the presence of about 20 ppb of ozone, as this is a natural background which cannot be reduced without an undesirable reduction in stratospheric ozone concentrations. It seems unlikely that such a level of ozone would cause an adverse affect, and it is possible that the epidemiological studies suggesting that there is no threshold may be either insufficiently sensitive to recognize a threshold at this level, or may be seeing an effect which is the result of confounding by some other pollutant which varies in concentration in the same way as ozone. The latter is a distinct possibility, given the complexity of atmospheric photochemistry. Reduction of ground-level ozone concentrations to their pre-industrial level would require reductions in emissions of $NO_x$ and VOC which would be so great as to change completely the nature of society as we know it. It seems most unrealistic to imagine that this would happen, and therefore if one accepts that there is either no threshold for the adverse effects of ozone or that the threshold is at the natural ozone background of around 20 ppb, then one must also accept that any realistically attainable air quality standard will involve health consequences from ozone for a small proportion of the population.

Carbon monoxide can be described as an asphyxiant since it takes up the oxygen carrying capacity of blood. In this case the key effect in standard setting has been the induction of angina in cardiovascular disease patients during exercise, for which the lowest observable effect level occurs at 3–4% carboxyhaemoglobin in blood. Incorporation of a safety margin aimed to ensure that carboxyhaemoglobin concentrations do not exceed 2.5% led EPAQS[6] to an air quality standard of 10 ppb over 8 hours exposure, and the WHO to a similar value for 8 hours and higher concentrations for shorter time periods.

For genotoxic carcinogens such as benzene, 1,3-butadiene and polycyclic aromatic hydrocarbons (PAHs), there is no totally safe level of exposure. The WHO have analysed the occupational disease studies and come up with an excess risk of contracting cancer following lifetime exposure. Taking for example the unit risk factor for benzene of $6 \times 10^{-6} (\mu g\, m^{-3})^{-1}$, this implies that six persons in a population of one million will contract cancer when exposed for their lifetime to a benzene concentration of $1\ \mu g\, m^{-3}$. Faced with the problem of setting a numerical air quality standard rather than an exposure–response gradient, EPAQS[4] used essentially the same occupational data to identify an exposure concentration below which a large cohort of workers showed no significant

[14] R. M. Harrison, in *Pollution: Causes, Effects & Control*, ed. R. M. Harrison, Royal Society of Chemistry, Cambridge, 1996.

excess of disease. In the case of benzene, this concentration was 500 ppb. EPAQS then divided by an exposure duration factor, which allows for the greater duration of exposure of the general population (*i.e.* 24 hours a day, 365 days a year) relative to the occupationally exposed population (40 hours per week, 46 weeks per year). The exposure duration factor used was 10. EPAQS divided also by a safety factor to protect sensitive groups, and adopted a factor of 10 for this. The application of both factors leads to an air quality standard of 5 ppb, and additionally a long-term target of 1 ppb was recommended. Both concentrations were expressed as running annual averages, reflecting the fact that it is long-term integrated exposure to chemical carcinogens rather than short-term excursions which are believed important in determining the induction of cancer. This methodology has been explained in depth elsewhere.[15,16]

The US EPA has done much to develop quantitative risk assessment of carcinogens for regulatory applications. Thus, quantitative estimates of carcinogenic potential akin to the unit risk factor cited above for benzene (which originates from the WHO) are used in standard setting. The attraction of this approach is that if unit risk factors can be established with confidence for a range of pollutants, and society can agree a tolerable level of risk, then maximum tolerable exposures to pollutants, and hence environmental quality standards, follow very straightforwardly. Such an approach has formed the basis for setting standards for many years in the field of radiological protection, where data collected from nuclear bomb survivors in Hiroshima and Nagasaki give a reasonable basis for estimation of unit risk factors. In the field of chemical carcinogenesis, the databases from which to estimate unit risk factors are far less substantial, and hence far greater uncertainty attaches to the estimates. Additionally, the unit risk factors obtained can be very sensitive to the model used to derive them, and hence in the UK, the Department of Health Committee on Carcinogenicity recommends against the routine use of quantitative risk assessment models. Hrudey and Krewski[17] have analysed some of the weaknesses in the modelling approaches used by the US EPA.

The next crucial question which has to be addressed is what is a tolerable level of risk? Naturally, there is no universally agreed answer to this question, and indeed, society is notably more tolerant of self-imposed risks (*e.g.* cigarette smoking) than of risks which are perceived as externally imposed such as outdoor air pollution. To put the matter into context, some representative levels of risk associated with well known events appear in Table 8. Given that it is not possible to achieve a situation of zero risk, long-term policy is generally directed at reducing risks to what is termed as a *de minimus* level, usually taken as a lifetime risk of one in $10^6$. For short-term regulatory purposes a lifetime risk of one in $10^5$ or one in $10^4$ is seen as more realistic. Note that the risk estimates in Table 8 apply to annual risk and should therefore be adjusted by a factor of about 80 to give levels of lifetime risk. Thus, an annual risk of one in $10^6$ equates to a lifetime risk of approximately one in $10^4$. What does this mean in practice? A very approximate

[15] R. L. Maynard, K. M. Cameron, R. Fielder, A. McDonald and A. Wadge, *Hum. Exp. Toxicol.*, 1995, **14**, 175.

[16] R. J. Fielder, *Toxicology*, 1996, **113**, 222.

[17] S. E. Hrudey and D. Krewski, *Environ. Sci. Technol.*, 1995, **29**, 370A.

**Table 8** Relative levels of annual risk

| *Descriptor* | *Risk estimate* | *Example* | |
|---|---|---|---|
| High | $>1:100$ | Transmission to susceptible household contact of measles and chickenpox | 1:1–1:2 |
| | | Gastrointestinal effects of antibiotics | 1:10–1:20 |
| Moderate | 1:100 to 1:1000 | Death from smoking 10 cigs/day | 1:200 |
| | | Death, all natural causes, age 40 yrs | 1:850 |
| Low | $1:10^3$ to $1:10^4$ | Death from influenza | 1:5000 |
| | | Death in road accidents | 1:8000 |
| Very low | $1:10^4$ to $1:10^5$ | Death from leukaemia | 1:12 000 |
| | | Death, playing soccer | 1:25 000 |
| | | Death, accident at home | 1:26 000 |
| | | Death, accident at work | 1:43 000 |
| | | Death from homicide | 1:100 000 |
| Minimal | $1:10^5$ to $1:10^6$ | Death, accident on railway | 1:500 000 |
| Negligible | $<1:10^6$ | Death, hit by lightning | $1:10^7$ |
| | | Death, release of radiation from nuclear power station | $1:10^7$ |

Based on ref. 18.

ilustration can be taken from the EPAQS benzene standard, which was set up at 5 ppb ($16\ \mu g\ m^{-3}$); when combined with the WHO unit risk factor this implies a lifetime risk of $1 \times 10^{-4}$, which is comparable with the levels of risk considered tolerable in radiological protection. However, this numerical exercise can give a highly spurious impression of precision to the process and it would be quite wrong for quantitative risk assessment to be regarded as a universal panacea in standard setting.

Perhaps the most difficult of all non-threshold pollutants is particulate matter. The literature contains a substantial body of epidemiological studies, linking a range of adverse health outcomes with day-to-day changes in the concentration of particulate matter within a city, generally measured as $PM_{10}$. These so-called time series studies give no indication of a threshold concentration below which no effects occur, and indeed, Watt and co-workers[19] have argued that although there will be a threshold concentration for individuals below which no harm from particle exposure will occur, this threshold will vary considerably between individuals. Additionally, within a given city, true individual exposure to particulate matter will also vary substantially. Therefore, within the population of a city, because of the wide distribution of individual thresholds and individual exposures, no threshold will be observable. In addition to the time series studies, there are three important cross-sectional studies which have looked at the rates of

[18] Department of Health, *Chief Medical Officer's Report for 1995*, HMSO, London, 1996.
[19] M. Watt, D. Golden, J. Cherrie and A. Seaton, *Occup. Environ. Med.*, 1995, **52**, 790.

mortality and disease in populations with different long-term exposures to particulate matter, and have shown appreciably elevated death rates in the cities with high fine particle concentrations. The interpretation of these results in relation to loss of life expectancy is extremely difficult, and furthermore, whilst the time series studies show that more people die and are admitted to hospital for respiratory and cardiovascular diseases on high particulate matter pollution days, it is unclear whether these events are simply being advanced by a few days, months or years. Hence the impact of particulate matter pollution on the prevalence of disease and reduction of life expectancy in the population as a whole is unclear. Reflecting these uncertainties, the WHO[11] produced a series of tables expressing exposure–response functions for a range of outcomes such as bronchodilator use, cough, lower respiratory symptoms, respiratory hospital admissions and mortality. Faced with the problem of setting a numerical standard, EPAQS[9] acknowledged that it was impossible to set a standard which would be totally protective against all adverse effects and recommended a standard for $PM_{10}$ of $50\,\mu g\,m^{-3}$, 24 hour running mean, as a concentration at which health effects on individuals were likely to be small and the very large majority of individuals will be unaffected. It was noted that a rise from a daily average $PM_{10}$ level of $20\,\mu g\,m^{-3}$ to $50\,\mu g\,m^{-3}$, a concentration which was exceeded on average one day in 10 in a study in Birmingham, UK, would be expected to be associated with just over one extra patient on average being admitted to hospital with respiratory disease daily in a population of one million. This was considered tolerable, although clearly this is a very subjective judgement.

The standard setting process described above for particulate matter is based solely on the results of the many published time series studies of air pollution and daily morbidity and mortality. There have also been published three long-term studies[20–22] which give some indication of the effects of chronic exposure to particulate matter over many years. The studies are all cohort studies which take a group of known individuals and follow them forwards in time, accumulating data upon the morbidity and mortality amongst the cohort, who are assessed initially in relation to individual risk factors such as smoking, body mass index and socio-economic status. The studies are cross-sectional in the sense that the subjects studied live in a number of cities (from six in the Harvard Six Cities Study[20] to 151 in the so-called American Cancer Society Study[21]) with differing levels of air pollution. Two of the studies have revealed linear relationships between mortality rates and airborne concentrations of particulate matter measured as $PM_{10}$ or $PM_{2.5}$ after normalizing the data for the other risk factors. The outcomes of these two studies[20,21] suggest major differences in life expectancy due to ambient particle exposure, but without making a number of arbitrary assumptions it is not possible to estimate the number of years of life lost. A further difficulty in using the studies to set air quality standards for particulate

[20] D. W. Dockery, C. A. Pope III, X. Xu, J. D. Spengler, H. J. Ware, M. E. Fay, B. G. Ferris Jr. and F. E. Speizer, *New Engl. J. Med.*, 1993, **329**, 1753.

[21] C. A. Pope III, M. J. Thun, M. W. Namboodiri, W. D. Dockery, J. S. Evans, F. E. Speizer and C. W. Heath, *Am. J. Respir. Crit. Care Med.*, 1995, **151**, 669.

[22] D. E. Abbey, M. D. Lebowitz, P. K. Mills, F. F. Petersen, W. L. Beeson and R. J. Burchette, *Inhal. Toxicol.*, 1995, **7**, 18.

matter is that it is probable that the subjects' exposures having the greatest long-term health impact will have taken place before the commencement of the study and before monitoring data became available. It is in this earlier time frame that concentrations will have been at their highest, and the subjects of the study in their infancy when long-term detriments to their well-being from air pollutant exposure would be greatest. Therefore, despite apparently revealing quite major impacts of particulate matter on health, these studies give little basis for determining exposure–response relationships or for standard setting. A further difficulty, as with the time series studies, is that the cohort studies to date show no threshold below which effects on health are not observable.

## 6 Concluding Remarks

All air quality standards should combine both a concentration and an averaging time. That averaging time reflects the duration of exposure associated with the eliciting of a response from exposure to the pollutant. Thus, for the irritant gases the exposure times are generally relatively short as the effects are acute, whilst for the genotoxic carcinogens the effects are chronic and the averaging times are long. The question also arises as to what locations the standards should be applied. The UK position on this is both rational and perceptive in that the UK National Air Quality Strategy[12] states that 'the objectives should apply in non-occupational, near ground-level outdoor locations where a person might reasonably be expected to be exposed over the relevant averaging period'. This is very important in implicitly including hotspot locations where people spend a significant amount of time in relation to the pollutant and potential effects, but excluding extreme situations that have no real relevance to human health. Clearly, monitoring strategies should be designed to reflect this kind of logic.

Finally, it must be recognized that pollutants can have interactive effects and may possibly act synergistically. Thus, for example, at one time it was believed that smoke and sulfur dioxide acted synergistically to elicit a greater effect than the sum of the two acting independently. This was largely a matter of faith as at the time the two pollutants had a major common source in coal combustion, and when one pollutant was elevated in concentration, the other was also. More recent thinking has suggested that the two pollutants tend to act independently of one another. A recent authoritative report[23] reviewing literature evidence for the health impact of pollutant mixtures concluded that there was little hard evidence available to suggest that pollutant interactions were particularly important. However, recent epidemiological studies have failed to disentangle in a wholly consistent manner the impacts of the many different pollutants whose concentrations tend to co-vary in the atmosphere due to common sources (usually motor traffic) and the same meteorological influences on concentation. Thus, although some studies appear to point to individual pollutants, others are more equivocal, and some studies have been totally unable to disaggregate clearly the effects of the various pollutants in the urban mix. A view is therefore commonly being expressed that the health effects being identified result from exposure to the mix

[23] Department of Health, Advisory Group on the Medical Aspects of Air Pollution Episodes, *Health Effects of Exposures to Mixtures of Air Pollutants*, HMSO, London, 1995.

as a whole and that attribution to individual components should be conducted with considerable caution. Thus, there are a variety of expert views on this matter, which is itself a very important one in standard setting. It is to be hoped that this issue will clarify over the next few years as epidemiological studies improve in their sophistication.