Ambient Air Quality NEPM Review

Air Quality Standards Discussion Paper

National Environment Protection Council

- Ministerial Council comprising one Minister from each jurisdiction
- Established under NEPC (Act) 1994
- Is responsible for making, reviewing and varying National Environment Protection Measures (NEPMs)

Ambient Air Quality NEPM

- AAQ NEPM was made in 1998
- Set national air quality standards for six criteria pollutants
  - PM_{10}, O_3, CO, NO_2, SO_2, and lead
- NEPM was varied in 2003 to include advisory reporting standards for $PM_{2.5}$

Ambient Air Quality NEPM

- When NEPM was made a set of future actions were agreed to including
  - Formation of a taskforce to investigate use of risk assessment to set air quality standards
  - Development of standards for $PM_{2.5}$
  - Review of the ozone standards to consider a 1-hour standard of 0.08 ppm
  - Review of the SO_2 to consider a 10min standard
  - Commence a full review of the NEPM within 10 years of it's making

Ambient Air Quality NEPM

- Additional studies conducted
  - Time Activity Study – 2003
  - Multicity Mortality and Morbidity Study – 2010
  - Air Pollution and Children's Health Study – due for completion late 2010
- Standard Setting Working Group
  - Established as a partnership between the health and environment sectors to develop a nationally agreed methodology to develop air quality standards

Review of AAQ NEPM

- Commenced in 2005 with Issues Scoping Paper
- 1st discussion paper on policy framework and monitoring and reporting protocols released in 2007
- 2nd discussion paper on the health evidence for air quality standards - 2010

Review of AAQ standards

- This discussion paper covers
  - The role of air quality standards in air quality management
  - The basis for the current air quality standards in the NEPM
  - New health evidence that has emerged since 1998
  - International trends in air quality standards
  - Form of standards and options for future standards

Role of Air Quality Standards in Air Quality Management

- Standard provides a quantifiable target for environmental quality
  - NEPM standards apply to ambient (outdoor) air
- Standards were designed to be measured at locations generally representative of exposure of the population
  - do not apply at peak sites or ‘hot-spots’

Role of Air Quality Standards in Air Quality Management

- NEPM monitoring protocol does not apply to monitoring or controlling peak concentrations from major sources such as industry or near major roads

- The AAQ NEPM standards drive jurisdictional air quality management actions to meet the nationally agreed benchmark

Current Air Quality Standards

- Based on the understanding of the health effects of the pollutants at the time of making the NEPM
- Full review of the epidemiological and toxicological literature was undertaken
  - Based primarily on overseas data
- The existing air pollutant levels and consideration of the achievability of the standards within 10 years also factored into the final standards
- Costs and benefits of meeting the standards also taken into account

Current AAQ NEPM standards

	Pollutant	Averaging period	Maximum concentration	Goal within 10 years maximum allowable exceedences
1	Carbon monoxide	8 hours	9.0 ppm	1 day a year
2	Nitrogen dioxide	1 hour 1 year	0.12 ppm 0.03 ppm	1 day a year none
3	Photochemical oxidants (as ozone)	1 hour 4 hours	0.10 ppm 0.08 ppm	1 day a year 1 day a year
4	Sulfur dioxide	1 hour 1 day 1 year	0.20 ppm 0.08 ppm 0.02 ppm	1 day a year 1 day a year none
5	Lead	1 year	0.50 µg/m^3	none
6	Particles as PM_{10}	1 day	50 µg/m^3	5 days a year
7	Particles as $PM_{2.5}$	1 day 1 year	25 µg/m^3 8 µg/m^3	

This presentation

- Each pollutant discussed
  - Basis for current standard
  - New evidence
  - International trends in air quality standards

Carbon Monoxide

- Current standards based on achieving carboxyhaemoglobin (COHb) in blood
  - COHb a marker for CO exposure

- Range of health effects considered
  - Cardiovascular effects in healthy adults
  - Cardiovascular effects in people with ischemic heart disease
  - Neurobehavioural effects in healthy adults
  - Foetal effects

Carbon Monoxide

- Linear response between CO and COHb
- People most susceptible to effects of CO are people with existing cardiovascular disease in particular the elderly with ischemic heart disease

Carbon Monoxide-
Current health evidence

- Epidemiological studies show that CO is linked to increases in mortality from cardiovascular disease
  - USEPA (2009) concluded that overall evidence suggestive of a causal effect at environmentally relevant exposures

Carbon Monoxide-
Current health evidence

- Epidemiological studies show CO linked to increases in hospital admissions and emergency department attendances for cardiovascular disease
  - Ischemic heart disease
  - Congestive heart failure
- Strongest effects in the elderly (>65 years) and people with existing heart disease

Carbon Monoxide

Australian Studies

- Australian studies show similar effects to European and US studies

- A meta-analysis of 5 Australian cities and 2 New Zealand cities found a 2.3% increase in hospital admissions for ischemic heart disease and 2.9%increase in admissions for myocardial infarction per 0.9 ppm increase in 8-hour CO

- Associations were also found for increases in admissions for the elderly for all CVD (2.2%), all cardiac disease (2.8%) and cardiac failure (6%) per 0.9ppm increase in 8-hour CO

Carbon Monoxide
Australian Studies

- Single city studies conducted in Sydney, Melbourne and Perth have also found consistent associations with admissions and emergency department attendances for cardiovascular disease and CO

- Results similar to those obtained in the meta-analysis

Carbon Monoxide- Current health evidence

- Ambient CO also associated with birth and developmental outcomes
  - Most compelling evidence for preterm birth and cardiac birth defects
- Studies conducted in the US show exposure to CO early in pregnancy (first month and first trimester) linked to preterm births
- Limited studies showing links to cardiac birth defects
  - Findings supported by toxicological studies

Carbon Monoxide-
Current health evidence

- Evidence that CO is linked to low birth weights, small for gestational age (SGA and retarded intrauterine growth (IUGR)

- Australian studies have shown that CO levels in Sydney are linked with SGA.
  - No clear effect was found for preterm birth

Carbon Monoxide-
Current health evidence

- Epidemiological studies have shown no evidence of a threshold for effect

- Sensitive groups –
  - People with existing cardiovascular disease
  - The elderly
  - Foetus

Findings of the Review of CO

- Australian studies have found associations between CO at current levels and increases in mortality and hospital admissions for cardiovascular disease

- Effects also found for adverse birth outcomes

- Findings consistent with international studies
  - Strongest evidence for hospital admissions and emergency department visits for ischemic heart disease and congestive heart failure

Findings of the Review of CO

- USEPA (2009) concluded that the evidence from epidemiological studies is coherent with that from controlled human exposure and animal toxicological studies
  - Indicates a direct effects of short-term CO exposures on cardiovascular morbidity at current ambient levels
- USEPA and WHO concluded that such effects are plausible
  - Long-term low level exposure could result in COHb concentrations used in controlled exposure studies

Nitrogen Dioxide
Current standards

- Health Effects
  - Decreases in lung function
  - Increases in respiratory illness
  - Increases in hospital admissions and emergency department attendances for respiratory and cardiovascular disease and asthma
  - Some evidence for increases in mortality

Nitrogen Dioxide

Current standards

- People most susceptible to effects of NO_2
  - People with existing respiratory and cardiovascular disease
  - People with asthma
  - Children

Current Health Evidence

- International studies show associations between ambient NO_2 concentrations and increases in all cause mortality
  - Effect estimates 0.5 to 3.6% increase in mortality per 20ppb increase in 24-hour NO_2 and 30ppb increase in 1-hour NO_2)
- Both cardiovascular and respiratory mortality have been associated with increases in short-term NO_2
  - no evidence of association with long-term exposure to NO_2

Current Health Evidence

- Epidemiological studies show consistent associations between short-term NO_2 and hospital admissions and emergency department attendances particularly for children with asthma

- Studies also show increases in asthma symptoms and medication usage linked to short-term NO_2

Current Health Evidence

- Clinical studies show asthmatics are more susceptible to exposure to NO_2
- Short-term exposures to NO_2 is associated with airway reactivity and enhanced inflammatory response in people with asthma
- Animal toxicology studies support the findings of epidemiological and controlled exposure studies

Nitrogen Dioxide

Current Health Evidence

- Effects greatest for respiratory effects
- No strong evidence from international studies for an association between short-term NO_2 and cardiovascular outcomes
- Some evidence for association with hospital admissions and emergency department attendances for cardiovascular disease
  - No clear mechanistic evidence

Nitrogen Dioxide

Australian Evidence

- Australian multicity studies have shown ambient NO_2 is associated with increases in mortality and hospital admissions for all cause, respiratory and cardiovascular causes

- The effects are similar or greater than those observed in Europe and US
  - Similar to Canada
  - 0.11 to 0.9% increase in mortality per 1 ppb increase in NO_2 (Australia) cf 0.03 to 0.04% increase (US)

Nitrogen Dioxide

Current Health Evidence

- International reviews vary in assessment of an association between long-term exposure to NO_2 and respiratory symptoms and asthma prevalence and incidence
  - USEPA suggestive but not sufficient for causality for respiratory morbidity or asthma incidence
  - Cal EPA – evidence clearly demonstrates respiratory health effects and long-term NO_2
  - WHO evidence of long-term exposures associate with increases in respiratory symptoms and decreases in lung function

Nitrogen Dioxide

Current Health Evidence

- Southern Californian Children's Health Study demonstrates some of the strongest evidence of a long-term effect of NO_2
  - reduced lung growth for children exposed to higher NO_2 over an 8-year period
  - Risk factor for chronic disease and premature mortality later in life
  - Effects observed in areas with average NO_2 of 18 to 57ppb

Nitrogen Dioxide

Current Health Evidence

- Infants, children, and the elderly more susceptible to the effects of NO_2
- People with asthma and other chronic respiratory and cardiovascular disease are particularly vulnerable

Findings of Review of NO_2

- Studies from Australia, US and Europe found positive associations between short-term exposures to NO_2 and increases in mortality and hospital admissions mainly for respiratory disease
  - Effects greater in Australia and Canada
- Effects independent of other pollutants
- Observed at current ambient levels of NO_2

Findings of Review of NO_2

- Long-term exposures linked to changes in lung growth in children and respiratory symptoms in asthmatic children
  - Effects observed at levels between 0.03 and 0.04 ppm.

- Short-term effects observed at levels between 0.018 and 0.036ppm
  - Well below current standard of 0.12ppm
  - No evidence of a threshold for effect

Ozone Current standards

- Health effects considered
  - Mortality
  - Reduced lung function in healthy adults and children
  - Exacerbation of asthma
  - Increases in respiratory symptoms
  - Increased airway responsiveness
  - Airway inflammation

Ozone Current standards

- Evidence of increases in mortality from respiratory and cardiovascular causes associated with both 1-hour and 8-hour O_3
- Increases in hospital admissions and emergency department attendances also observed
- Changes in lung function also observed
- Symptoms included cough and chest pain on inspiration

Ozone Current standards

- Findings of epidemiological studies supported by controlled exposure and toxicological studies

- Decision on 1-hour and 4-hour standards based on consideration of ozone formation patterns and exposure in Australian cities

Ozone Current Evidence

- In 2005 NEPC completed preliminary work for the review of the ozone standards
  - Found that standards should be based on 1-hour, 4-hour and 8-hour averaging periods to account for exposure in Australian cities

- Short-term (1-4 hour exposures) linked to increases in mortality, hospital admissions and emergency department attendances mainly for respiratory causes

Ozone Current Evidence

- Effects are greatest in the warm season and in the elderly
- Studies from Europe, US and Australia showing similar associations
- European studies show 1.8% increase in all cause mortality. 2.7% increase in cardiovascular mortality and 6.8% increase mortality per 30ppb increase in 8-hour ozone

Ozone Current Evidence

- No evidence for threshold for effect

- Exposure to ozone linked to range of respiratory outcomes including
  - decreases in lung function,
  - increases in respiratory symptoms,
  - increased respiratory inflammation,
  - increased airway responsiveness

Ozone Current Evidence

- Studies show increases in emergency department attendances for asthma linked to both 1-hour an 8-hour ozone
  - Effects greatest in warm season

- Evidence for cardiovascular effects not as strong as for respiratory effects
  - USEPA concluded evidence for cardiovascular effect inconclusive

Ozone Current Evidence

- Findings of controlled exposure studies show that exposure to ozone at ambient levels are associated with
  - Decreased inspiratory capacity
  - Bronchoconstriction
  - Rapid, shallow breathing during exercise
  - Cough and pain on inspiration

- Reduction in inspiration results in decrease in lung function such as FVC and together with bronchoconstriction, decreases in FEV_1

Ozone Current Evidence

- Long-term exposure to ozone not associated with increases in mortality
- Some evidence of increase in lung cancer in non-smoking population in high ozone areas
- Long-term exposures linked to changes in lung function in both healthy adults and people with asthma
  - Supported by results of animal toxicological studies

Ozone Australian Evidence

- Results with mortality mixed
  - Most recent studies show no association

- Multicity studies show increases in hospital admissions for respiratory causes and ozone in the warm season
  - Supported by single city studies

Ozone Australian Evidence

- Studies also show ozone associated with risk of preterm birth in Sydney and Brisbane

- Associations found for 1-hour, 4-hour and 8-hour ozone levels for all outcomes

Findings of Ozone Review

- Recent studies support findings of 2005 report
- Exposure to ozone linked to increases in mortality, hospital admissions and emergency department attendances mainly for respiratory causes
  - Linked to 1-hour, 4-hour and 8-hour ozone at current ambient levels
- Ozone also linked to changes in lung function in both long-term and short-term studies

Sulfur Dioxide Current standards

- Both short-term and long-term effects considered

- Exposure to SO_2 creates acute irritant response
  - Leads to coughing, wheezing, sputum production, increased incidence of respiratory infections, aggravation of asthma and COPD
  - Asthmatics particularly sensitive to SO_2 and respond very quickly (minutes)

Sulfur Dioxide Current standards

- Increases in mortality and hospital admissions for asthma and respiratory disease
- Increases in respiratory symptoms
- Results from both epidemiological studies and controlled human exposure studies used to derive standard
- Asthmatics considered as most sensitive group

Sulfur Dioxide New Evidence

- Epidemiological studies show association between short-term exposures and increases in daily mortality from respiratory and cardiovascular effects
  - Effects stronger for respiratory outcomes
  - Intervention study in Hong Kong provides strong evidence of an independent effect

- Strongest evidence of effect comes from controlled human exposure studies and respiratory effects
  - Effects greatest when people are exercising

Sulfur Dioxide New Evidence

- Epidemiological evidence supported by controlled human exposure studies and animal toxicological studies conducted near ambient concentrations show link with SO_2 measures of respiratory health
  - Respiratory symptoms, inflammation, and airway hyperresponsiveness

- Effects more pronounced in asthmatic children and the elderly

Sulfur Dioxide New Evidence

- Strongest evidence for a causal relationship comes from controlled human exposure studies reporting increased respiratory symptoms and decreased lung function following 5-10 min exposures

- Exposure duration not critical
  - Response is rapid
  - Continuing exposure does not increase effect
  - Observed in people with mild to moderate asthma

Sulfur Dioxide New Evidence

- Epidemiological studies show consistent and robust associations for hospital admissions and emergency department attendances for respiratory causes
  - No consistent effect found for cardiovascular disease
- Australian studies show association for hospital admissions for respiratory causes
  - Both adults and infants

Sulfur Dioxide New Evidence

- Evidence for long-term effects limited
  - Some evidence for an association with mortality
  - Inconsistent findings for respiratory morbidity
- Some associations found for exacerbation in asthma, bronchitis and respiratory symptoms in children
- Evidence inadequate to infer causal relationship (USEPA, 2008)

Sulfur Dioxide New Evidence

- Studies have shown exposure to SO_2 and associations with low birth weights and premature births
  - Evidence limited
- No threshold observed for adverse effects
- Asthmatics and the elderly most susceptible to effects of SO_2
  - Also people with COPD and cardiovascular disease and children

Findings of the Review of SO_2

- Epidemiological studies show association with mortality, in particular respiratory mortality
- Also associations with respiratory and cardiovascular morbidity
- Asthmatics most susceptible group
- No threshold for effects
- Effects observed at current levels of SO_2

Lead current standards

- Wide range of health effects
  - Reduction in learning ability and IQ in children
  - Increased blood pressure in adult males
  - Effects on central nervous system
  - Effects on both male and female reproductive systems
  - Anaemia
  - Preterm deliveries
  - Reduced birth weights

Lead current standards

- Standards based on blood lead levels not exceeding 10ug/dL
- Foetuses infants and children most susceptible to the effects of lead
- Standards based on IQ deficits in children
  - No evidence of threshold for effect in epidemiological studies

Lead New Evidence

- Children still considered most vulnerable group
- Strong evidence exists for causal relationship between lead and increased blood pressure and hypertension in adults
- Some evidence between lead and increases in mortality and morbidity for cardiovascular causes
  - Inconclusive at this stage

Lead New Evidence

- Epidemiological studies show decreases in cognitive function, in particular IQ, in children at blood lead levels below 10 ug/dL

- Non-linear dose-response between blood lead and neurodevelopmental effects
  - U-shaped curve

- Number of large studies consistently show lead is associated with various neurodevelopmental effects at blood lead levels between 5 and 10ug/dL

Lead New Evidence

- A number of large epidemiological studies have reported associations between lead and indicators of renal function impairment
  - Mean blood lead levels between 3.3 and 4.2 ug/dL
- Results of toxicological studies support findings of epidemiological studies

Findings of the Lead Review

- Children remain most vulnerable group to effects of lead
- Health effects observed at blood lead levels below 10ug/dL
- Strong evidence for causal relationship between lead and hypertension and renal function impairment in adults

Particles Current Standards

- PM_{10} standards based on:
  - Increases in mortality, hospital admissions and emergency department attendances (respiratory and cardiovascular causes)
  - Aggravation of existing disease (respiratory and cardiovascular causes)
  - Altered lung clearance and other host defence mechanisms
  - Increases in respiratory symptoms
  - Decreases in lung function

Particles Current Standards

- Sensitive groups considered
  - Elderly
  - Children
  - People with pre-existing heart and lung disease
- No evidence of a threshold for effects

Particles Current Standards

- $PM_{2.5}$
  - Similar health effects to those considered for PM_{10}
  - Evidence from Australian studies incorporated

Particles New Evidence

- Significant increase in epidemiological studies and studies providing evidence on biological plausibility
- Evidence for both PM_{10} and $PM_{2.5}$ and to a lesser extent ultrafine particles and $PM_{10-2.5}$
- No threshold for effects

Particles New Evidence

- Health effects
  - Increases in daily mortality
    - Much stronger evidence now for cardiovascular causes
  - Effect estimates 0.12-0.8% increase per 10ug/m^3 PM_{10} all cause mortality
  - Australian studies 0.2 % increase per 10ug/m^3 PM_{10} all cause mortality
  - Some heterogeneity observed in effects

Particles New Evidence

- Stronger effects found for cardiovascular causes
  - Effects estimates from multicity studies 0.47 to 0.85% increase per 10ug/m^3 $PM_{2.5}$
- Effects in Canadian and Australian studies greater than those observed in European and US studies
- Associations also found for coarse particles and cardiovascular mortality

Particles New Evidence

- Increases in hospital admissions and emergency department attendances with $PM_{10,}$ $PM_{2.5,}$ $PM_{10\text{-}2.5}$
  - Effects for both cardiovascular and respiratory effects
  - Strong associations for ischemic heart disease and congestive heart failure
  - Also respiratory disease, asthma and COPD

Particles New Evidence

- Associations also found for particles and increases in respiratory symptoms and medication use in asthmatic children
  - Not as consistent in adults
- Particles also linked to reduction in lung function and lung inflammation
- Particles induce oxidative stress, pulmonary injury and inflammation

Particles New Evidence

- New evidence supports biological plausibility for cardiovascular effects
  - Particles shown to interfere with electrical signals in heart disrupting heart function
  - Supported by toxicological studies

Particles New Evidence

- Long-term effects
  - Several new studies
  - Similar outcomes to short-term effects
  - Increases in mortality respiratory and cardiovascular causes
  - USEPA concluded that causal relationship is likely to exist between long-term exposure to $PM_{2.5}$ and mortality

- USEPA concluded that causal relationship exists between long-term exposure to $PM_{2.5}$ and cardiovascular outcomes including mortality
  - Supported by both epidemiological and toxicological studies
- Long-term exposure also linked to decrements in lung growth, increased respiratory symptoms and asthma development
- Children at greater risk from long-term exposures than adults

Particles New Evidence

- Particles also linked to adverse birth outcomes
  - Low birth weight
  - Infant mortality
  - Supported by toxicological evidence
- No threshold for effect
  - Linear dose response

Particles New Evidence

- Not sufficient evidence at this time to show independent effect of UFP
- Some evidence for coarse particles
- Limited evidence for role of particle composition
  - Different components may target different biological systems

Findings of Particle Review

- Substantial new evidence from time series studies and cohort studies on both short-term and long-term effects
- PM_{10} and $PM_{2.5}$ associated with increases in mortality and morbidity
- Studies in Australia show similar effects to overseas studies
  - Effects greater than in US and Europe but similar to Canada

Findings of Particle Review

- Not sufficient evidence for effects of UFP or particle composition
  - Indicators still PM_{10} and $PM_{2.5}$
  - Increasing evidence that $PM_{10-2.5}$ have an independent effect

- Effects observed at current levels of particles in Australian cities
  - No threshold for effect

Benzene Health Evidence

- Known human carcinogen
- Long-term exposure associated with increased incidence of bone marrow depression and leukaemia (acute myeloid leukaemia)
- Recent meta-analyses found association with increased incidence of Non-Hodgkins' Lymphoma (2008)
- Health information obtained from international studies; no studies have been conducted in Australia

Benzene Health Evidence

Five key occupational cohort studies demonstrating association with leukaemia:

- Goodyear Pliofilm (1981)
- Dow Chemical (1986)
- Chemical Manufacturers Association (1987)
- Chinese Factory Worker cohorts (1997)
- Chinese Shoe Worker study (2004)

Latest ATSDR Minimal Risk Level (2007) based on Chinese Shoe Worker studies

Benzene Health Evidence

Non-cancer health effects:

- Bone marrow damage
- Changes in circulating blood cells (white blood cells & platelets)
- Developmental and reproductive effects
- Cancer at multiple sites

International air quality standards currently stand at 1.5 ppb as annual average. Adopted by EU and UK air quality strategy. No known threshold for carcinogenic effects.

PAHs Health Evidence

Background

- PAHs exist as mixture of compounds, not single compound
- Toxicity of PAHs vary quite markedly
- Most toxic PAH is benzo(a)pyrene (BaP)
- Little information on single, pure PAH
- All health information based on exposure to mixtures of PAH and non-PAH compounds

PAHs Health Evidence

Health Research To-Date

- Epidemiological studies show exposure to PAHs associated with increased mortality due to cancer (e.g. coke oven emissions, roof-tar emissions, and cigarette smoke)
- BaP most extensively studied PAH in experimental animal studies and found to be linked with respiratory tract tumours following inhalation

PAHs Health Evidence

International studies:

- Most evidence from occupational studies where workers exposed to mixtures of PAHs
- Exposure to BaP linked with lung cancer in occupational studies
- Exposure to PAHs also found to be associated with mortality from ischaemic heart disease

PAHs Health Evidence

Australian studies:

- Increased mortality and cancer incidence with exposure to benzene-soluble fraction, BaP, fluoride and inhalable dust in two Australian prebake smelters (2009)

Experimental lab studies:

- Limited studies regarding exposure to PAHs by inhalation—all restricted to BaP
- Tumours observed in all studies with small animals
- Tumours at site of contact and distantly

PAHs Health Evidence

Current Status

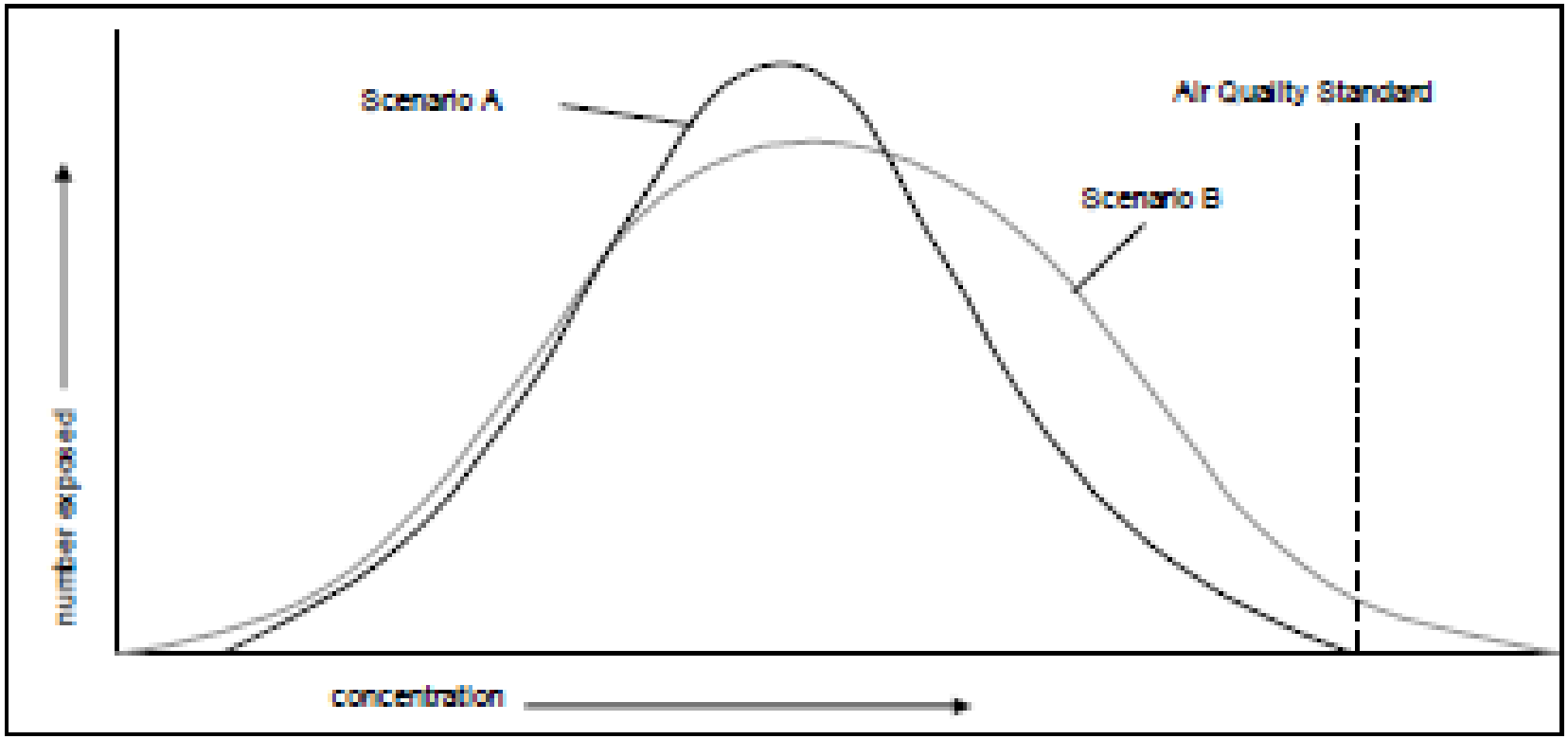
- Monitoring Investigation Level (MIL) for BaP in Air Toxics NEPM is 0.3 ng/m^3 as annual average
- MILs are not ambient air quality standards as in AAQ NEPM, but rather trigger levels for investigation
- If BaP moved to AAQ NEPM, MIL would need to be assessed to ensure level of protection is appropriate

Form of Standards

- Standards
  - Numerical value, averaging time, measurement technique, data handling, may be legally enforceable
- Guidelines
  - Numerical value, averaging time, often only advisory

Form of Standards

- Allowing exceedences

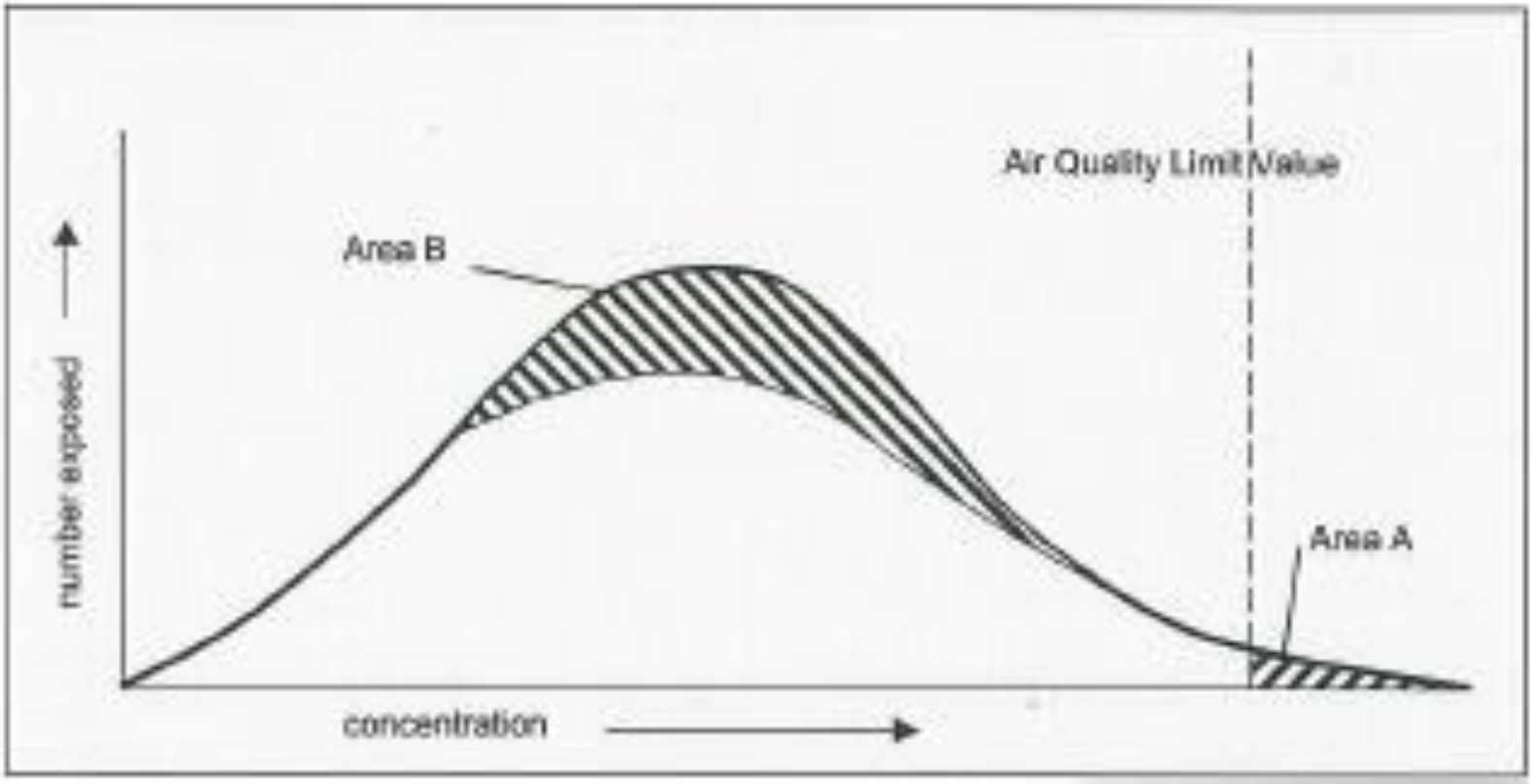


Form of Standards

- Assessing compliance
  - Including an allowable number of exceedences
  - Percentile form (not specifying an allowable number of exceedences)
  - ‘Not to be exceeded’ standard, plus reporting of causes of exceedences and actions taken
  - Enabling ‘exceptional’ or ‘natural’ events to be excluded from assessment of compliance

Form of Standards

- Exposure reduction



Issues to be considered in evaluating the NEPM standards

- Significant amount of new evidence, both internationally and in Australia, about the health effects of criteria pollutants, since the AAQ NEPM was made.
- Of particular importance is that the results show no threshold for health effects associated with exposure to the pollutants.

Issues to be considered…

- The form of the standard can effect the level of protection it affords/the level of risk posed.

For example: allowing exceedances could result in a less protective standard than the same numerical value with no allowable exceedances

Feedback sought

- Is there sufficient new evidence to support a recommendation to NEPC to revise the current standards? Which pollutants?
- Should the standards include allowable exceedances?
- Do you support changes to the reporting protocols that would require more transparent explanation of exceedances of the standards?

Where to from here?

- Around Australia consultation road show
- Submissions close Friday 27 August 2010
- Analysis of submissions from first Discussion Paper, this consultation round, health reviews and the preliminary cost-benefit analysis.
- Preparation of review report to NEPC
- Jurisdictional Reference Network process
- Review report and recommendations to NEPC

Submissions to:

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