### emission: Impossible

### 2013 WHO Review of Evidence on Health Aspects of Air Pollution



Summary background documentation for Health Protection Forum

**13 November 2013** 

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### 1.0 Background

The last review on the evidence of air pollution effects on health undertaken by the World Health Organisation (WHO) was published in 2006 and included data up to the end of 2004. The resulting *WHO Air Quality Guidelines Global Update 2005,* provided uniform targets that would **protect the large majority of individuals from the adverse effects on health of air pollution** (WHO, 2006). However, 80% of the population in the European WHO region currently live in cities with ambient levels above the global guidelines for particulate matter.

2013 is the "Year of Air" in the European Union. The *WHO Review of Evidence on Health Aspects of Air Pollution* sets out to provide answers to 23 key policy-relevant questions from the European Commission (WHO, 2013).

Key aspects to note:

- 29 invited experts did the review
- 32 reviewers provided comment
- 2 expert meetings in Bonn (Aug 2012, Jan 2013)
- Final text adopted at 2<sup>nd</sup> meeting by WHO

Although some of the European Commission questions asked directly for the assessment of individual policies or instruments, the review covered only scientific evidence underlying the policy (i.e. it did not address political arguments). This is because the role of WHO is normative – i.e. WHO evaluates scientific evidence for guidelines and recommendations only. Accordingly, WHO notes the following:

- The review is **not** policy
- The review does **not** consider technical feasibility, economic considerations and other political and social factors

### Notes to the reader:

- 1. Orange text is advice for the New Zealand context
- 2. This summary does not review questions on ozone (QB1- B4) as this is not a priority contaminant for New Zealand.



### 2.0 EC Questions & WHO Answers

- QA1. What new evidence on health effects has emerged since the review work done for the WHO air quality guidelines published in 2006, particularly with regard to the strength of the evidence on the health impacts associated with exposure to PM<sub>2.5</sub>? Based on this new information, do the scientific conclusions given in 2005 require revision?
  - Many more studies since 2005 Global Update.
  - Additional support for mortality and morbidity effects of **short-term exposure** to particulate matter less than 2.5 micrometres in diameter (PM<sub>2.5</sub>) based on several multicity epidemiological studies.
  - Additional support for mortality and morbidity effects of **long-term exposure** to PM<sub>2.5</sub> based on studies of long-term exposure on large cohorts in Europe and North America.
  - Authoritative review concluding long-term PM<sub>2.5</sub> is **causative** for cardiovascular mortality and morbidity
  - New insights into physiological effects and plausible biological mechanisms linking short and long-term PM<sub>2.5</sub> exposure with mortality and morbidity (observed in epidemiological, clinical and toxicological studies)
  - New health outcomes linked to long-term exposure to PM<sub>2.5</sub> (e.g. artherosclerosis, adverse birth outcomes and childhood respiratory disease)
  - **Emerging evidence** suggesting links between long-term PM<sub>2.5</sub> and neurodevelopment and cognitive function as well as other chronic diseases such as diabetes.

Overall conclusion confirms and strengthens the findings of the 2005 Global Update that there is a causative link between  $PM_{2.5}$  and adverse health outcomes.

The evidence base for the association between particulate matter (PM) and short-term, and longterm, health impacts has become larger and broader. Recent long-term studies show associations between PM and mortality at levels well below current annual WHO guideline. Recommend the WHO guideline be revised.



QA2. What new health evidence is available on the role of other fractions or metrics of PM, such as smaller fractions (ultrafines), black carbon, chemical constituents (metals, organics, inorganics, crustal material and PM of natural origin, primary or secondary) or source types (road traffic including non-tailpipe emissions, industry, waste processing...) or exposure times (for example, individual or repeated short episodes of very high exposure, 1 hour, 24 hours, yearly)?

New information shows PM mass comprises fractions with varying types and degrees of health effects. This suggests a role for both chemical composition (such as transition metals and combustion derived primary and secondary organic particles) and physical properties (size, particle number and surface area).

Important components or metrics with substantial exposure and health research finding associations and effects are as follows:

### 1. Black carbon

Black carbon (soot) is formed from incomplete combustion. Black carbon concentration is estimated by light absorption methods that measure the light absorption of particles retained in a filter – in absorption units. (Elemental or organic carbon is determined using thermo-optical methods, also on filter samples – in mass concentration units).

**WHO Conclusion:** Black carbon particles are a valuable additional air quality metric for evaluating the health risks of primary combustion particles from traffic, including organic particles, not fully taken into account with PM<sub>2.5</sub> mass.

### 2. Secondary organic aerosols

Secondary aerosols are solid or liquid particles that are formed from other gases (i.e. downwind of primary emissions).

**WHO Conclusion:** There is growing information on the associations of organic carbon with health effects.

### 3. Secondary inorganic aerosols

**WHO Conclusion:** Epidemiological studies continue to report associations between inorganic secondary aerosols such as sulphates or nitrates and human health. Even if not causal, they are a valuable additional air quality metric for evaluating health risks.



### 4. Coarse particles

Coarse (PM<sub>2.5-10</sub>) particulate (including crustal material) is associated with adverse respiratory and cardiovascular effects on health (including premature mortality).

- Toxicological studies report coarse particles can be as toxic as PM<sub>2.5</sub> on a mass basis
- Epidemiological studies show coarse PM has at least as strong short-term effects on respiratory health as PM<sub>2.5</sub> and health effect estimates for cardiovascular outcomes (admissions and physiological effects) were comparable.
- Clinical studies suggest both size fractions are comparable in inducing cardiopulmonary changes in acute exposure settings

### 5. Ultrafine particles

- There is increasing epidemiological evidence associating short-term exposures to ultrafine particles with cardiorespiratory health effects as well as the central nervous system.
- Clinical and toxicological studies show ultrafine particles (in part) act through different mechanisms to larger particles.

### 6. Transition metals and metal compounds

- The evidence shows no patterns for transition metals as a general category.
- Most evidence has been found for an association between nickel and cardiovascular hospital admissions.

### 7. Source type

• **Extreme caution** is required when attributing health effects to sources based on health impact assessment studies that use specific components of PM.

### 7.1 Traffic

• There is conflicting evidence about biodiesel fuel exhaust emissions being less harmful to human health relative to petroleum-based diesel emissions. This may relate to importance of total mix of emissions (gaseous as well as particulate emissions) and relative toxicity of non-exhaust emissions (brake wear and tyre dust).

### 7.2 Industry

• No general conclusions, rather source and outcome specific (e.g. strike at copper smelter associated with decreased mortality in the US, municipal waste incinerators associated with non-Hodgkin's lymphoma in France).



#### 7.3 Biomass combustion

- Includes residential wood combustion, wildfires and agricultural burning
- Systematic review in 2007 concluded there is **no reason to consider PM from biomass combustion less harmful** than particles from other urban sources.
- Available studies suggest cardiovascular effects of particles from biomass combustion may be comparable to traffic-related particles.
- Studies show associations between wood smoke and adverse effects on cardiovascular health (also ear inflammation, infant bronchiolitis, preterm birth) (but not development of asthma or low birth weight).
- Intervention study in small town in British Colombia with lots of wood burners. Installed HEPA filters and found improvements in endothelial function and decreased inflammatory biomarkers.

### 7.4 Desert dust

• Episodes have been linked with cardiovascular hospital admissions and mortality in a number of recent epidemiological studies.

### 7.5 Ocean and sea

- WHO states little epidemiological evidence of the harmfulness of sea salt.
- Clinical studies show clear evidence that PM dominated by sea salt is far less toxic than equal amounts of combustion-derived PM.
- However this appears to be contradicted by a study presented at CASANZ conference in Sydney earlier this year so hold that thought.

### 8. Exposure time

- Epidemiological studies show further evidence that long-term exposure to PM<sub>2.5</sub> is associated with both mortality and morbidity.
- Evidence base is weaker for PM<sub>10</sub>, hardly any long-term studies available for coarse particles (PM<sub>2.5-10</sub>).
- Strong evidence that daily exposures to PM are associated with both mortality and morbidity immediately and in subsequent days. Repeated (multiple day) exposures may result in larger health effects than the effects of single days.
- Long-term effects > sum of all short-term effects.



- This suggests effects are not just due to exacerbations, but may also be due to progression of underlying diseases.
- This potentially means that annual guidelines are more relevant than 24-hour average guidelines. BUT in New Zealand (as in Europe) daily PM<sub>10</sub> is highly correlated with annual PM<sub>10</sub> (see Figure 1). Therefore in New Zealand daily PM<sub>10</sub> is also important.
- There is significant evidence from toxicological and clinical studies that peak exposures to short duration (less than a few hours) combustion-derived particles leads to immediate physiological changes. Also supported by epidemiological observations.



### Figure 1 Annual average PM<sub>10</sub> correlation with daily average PM<sub>10</sub> in New Zealand\*

\*Data courtesy Kuschel *et al.* 2012. Based on 71 monitoring sites from Kaitaia to Bluff. Data of all 2nd highest 24-hour average and annual average PM<sub>10</sub> (averaged over all years of available data 2004-2010)



# QA3. EU legislation currently has a single limit value for exposure to PM<sub>2.5</sub>, which is based on an annual averaging period. Based on the currently available health evidence, is there a need for additional limit values (or target values) for the protection of human health from exposures over shorter periods of time?

The following points need to be considered in legislative decisions.

- 1. Although short-term effects may contribute to chronic health problems, those affected by short-term exposures are not necessarily the same as those suffering from the consequences of long-term exposures.
- 2. Not all biological mechanisms relevant to acute effects are necessarily relevant to the long-term effects and vice versa.
- 3. In periods with high PM<sub>2.5</sub> concentrations, health relevant action may be taken by citizens, public authorities and other constituencies.
- 4. Areas that have relatively moderate long-term average concentrations of PM<sub>2.5</sub> may still have episodes of fairly high concentrations.

In light of the above considerations, the scientific evidence supports the health impacts and the need to regulate concentrations for both short-term averages (such as 24-hour averages) and annual means.

- Europe currently only has an annual PM<sub>2.5</sub> limit.
- New Zealand currently only has a PM<sub>2.5</sub> daily 'reporting' guideline

# QA4. What health evidence is available to support an independent limit value for PM<sub>10</sub> (in parallel to (i) an annual average limit for PM<sub>2.5</sub> and (ii) multiple limits to protect from short-term and long-term exposures to PM<sub>2.5</sub>)?

A sizable amount of scientific literature exists on the short-term and long-term health effects of  $PM_{10}$  at concentrations below the current European limit values. The following arguments make it clear that  $PM_{10}$  is not just a proxy measure of  $PM_{2.5}$ .

- There is increasing evidence for the adverse effects on health of coarse particles (PM<sub>10-2.5</sub>). Short-term effects on health of coarse particles have been observed independently of those related to fine particles (PM<sub>2.5</sub>).
- 2. New European studies further strengthen the evidence for an association between long-term exposure to  $PM_{10}$  and health especially for respiratory outcomes and for health benefits from the reduction in long-term mean concentrations of  $PM_{10}$  to levels far below the current EU limit value for  $PM_{10}$ .



3. Coarse and fine particles deposit at different locations in the respiratory tract, have different sources and composition, act through partly different biological mechanisms, and result in different health outcomes.

Therefore, maintaining independent short-term and long-term limit values for ambient  $PM_{10}$  in addition to  $PM_{2.5}$ , to protect against the health effects of both fine and coarse particles, is well supported.

- Currently PM<sub>10</sub> daily standard and PM<sub>10</sub> annual guideline in New Zealand (only)
- This provides strong support for PM<sub>10</sub> and PM<sub>2.5</sub> daily and annual average standards in New Zealand
- QA5. EU legislation has a concentration limit value and an exposure reduction target for PM<sub>2.5</sub>. To decide whether it would be more effective to protect human health through exposure reduction targets rather than limit or target values it is important to understand (among other things, such as exposure, costeffectiveness, technical feasibility) the shape of the concentrationresponse function. What is the latest evidence on thresholds and linearity for PM<sub>2.5</sub>?

### Thresholds

- For studies of short-term exposure, there is substantial evidence on associations observed down to very low levels of PM<sub>2.5</sub>. The data clearly suggest the absence of a threshold below which no one would be affected.
- Likewise long-term studies give no evidence of a threshold.
- Some recent studies have reported effects on mortality at concentrations below an annual average of 10 μg/m<sup>3</sup>.

### Linearity

- The European studies of short-term exposure that have rigorously examined concentrationresponse functions have not detected significant deviations from linearity for ambient levels of PM<sub>2.5</sub> observed in Europe.
- Few long-term studies have examined the shape of the concentration-response functions. There are, however, suggestions of a steeper exposure-response relationship at lower levels (supra-linear) from analyses of studies from different areas around the globe and with different ranges and sources of exposure.



• A steeper exposure-response relationship at lower levels (such as in New Zealand) may mean that the benefits of cleaner air are under-estimated.

In the absence of a threshold and in light of linear or supra-linear risk functions, public health benefits will result from any reduction in PM<sub>2.5</sub> concentrations, whether or not the current levels are above or below the limit values.

### QA6. Based on currently available health evidence, what PM metrics, health outcomes and concentration-response functions can be used for health impact assessment?

The evidence base supports quantification of the effects of several PM metrics and both short-term and long-term exposures.

- Specifically, a large body of evidence from cohort studies exists to support quantification of the effects of long-term exposure to PM<sub>2.5</sub> on both mortality (all-cause and cardiovascular) and morbidity.
- In addition, studies of short-term exposure support quantification of the acute effects of PM<sub>2.5</sub> on several morbidity outcomes.
- There are other PM metrics for which response functions have been published for at least some health outcomes, including PM<sub>10</sub>, the coarse fraction of PM<sub>10</sub>, black carbon, sulphate and others.
- Alternative metrics, such as black carbon, may be used in sensitivity analyses.

NB: One needs to keep in mind that the impact derived for different PM metrics should not be summed up, given that the effects and sources are not fully independent.

• Reassuring to see that HAPINZ outcomes match those recommended by WHO

# QC1. There is evidence of increased health effects linked to proximity to roads. What evidence is available that specific air pollutants or mixtures are responsible for such increases, taking into account co-exposures such as noise?

• Elevated health risks associated with living in close proximity to roads is unlikely to be explained by PM<sub>2.5</sub> mass since this is only slightly elevated near roads.



- In contrast, levels of such pollutants as ultrafine particles, carbon monoxide, nitrogen dioxide (NO<sub>2</sub>), black carbon, polycyclic aromatic hydrocarbons, formaldehyde and some metals are more elevated near roads.
- Individually or in combination, these are likely to be responsible for the observed adverse effects on health.
- This means all our assessment methodologies in New Zealand are out of date.
- QC2. Is there any new evidence on the health effects of nitrogen dioxide (NO<sub>2</sub>) that impact upon the current limit values? Are long-term or short-term limit values justified on the grounds that NO<sub>2</sub> affects human health directly, or is it linked to other co-emitted pollutants for which NO<sub>2</sub> is an indicator substance?
  - Yes there is lots of new evidence (extensively reviewed by WHO).
  - Both short-term and long-term studies have found adverse associations at levels below 2005 WHO limit values.
  - Chamber and toxicological studies provide mechanistic support for causal interpretation of respiratory effects.
  - WHO recommends reviewing both 1-hour and annual guidelines.
  - NB: A 2007 study by the California Air Resources Board found that long-term exposure to nitrogen dioxide may lead to changes in lung function growth in children, symptoms in asthmatic children and pre-term birth (CARB, 2007).
  - As elsewhere in the world, ambient levels of nitrogen dioxide in New Zealand are **not** reducing in parallel with reductions of particle emissions from vehicles.

### QC3. Based on existing health evidence, what would be the most relevant exposure period for a short-term limit for NO<sub>2</sub>?

- The most relevant exposure period based on existing evidence is 1 hour because 1-hour peak exposures in chamber studies have been shown to produce acute respiratory health effects.
- 1-hour and 24-hour average concentrations are well correlated in urban areas.
- 1-hour guideline is sufficient



### QC4. Based on currently available health evidence, what NO<sub>2</sub> metrics, health outcomes and concentration-response functions can be used for health impact assessment?

- Short-term exposure associations with respiratory health admissions and all-cause mortality. Use concentration –response functions adjusted for at least PM.
- Long-term nitrogen dioxide exposure association with mortality can be used as sensitivity analysis. Use concentration –response functions adjusted for at least PM.
- Cardiovascular hospital admissions can be used as sensitivity analysis (for both short and long-term).

## QC5. Is there any new evidence on the health effects of air emissions of arsenic, cadmium, mercury, lead and nickel (and their compounds) that would impact upon current target values?

- Arsenic new evidence but insufficient to impact on current target value.
- Mercury no new evidence that would impact on current policy.
- Nickel new evidence but unlikely to impact on current policy.

### Cadmium

- New evidence that should be considered in future review of WHO guideline. Reaching the present WHO guideline does not prevent increasing cadmium levels in agricultural soil by air deposition, and thereby contributing to adverse effects on health in the general population.
- NB: The 2000 WHO Regional Office for Europe air quality guidelines noted that *average* kidney cadmium levels in Europe are very close to the critical level for renal effects. A further increase in dietary intake of cadmium, due to accumulation of cadmium in agricultural soils, must be prevented.

### Lead

- New evidence shows effects on the central nervous system in children and on the cardiovascular system in adults that occur at, or below, present WHO guideline.
- The most recent review used a central estimate of blood lead level of 20 µg/L for an intelligence quotient (IQ) cognitive function decrement of one point in children. The lower confidence limit was 10 µg/L. The review transformed blood lead level into dietary intake



and chose a bilinear model that yielded a 0.5 IQ point decrease at 12  $\mu$ g lead/day (0.6  $\mu$ g.kg/day for a 20 kg child).

- Assuming the relationship in WHO 2000 guidelines is correct, lead in air of about 0.2  $\mu$ g/m<sup>3</sup> (i.e. at the NZ guideline level) would increase blood lead levels by about 12  $\mu$ g/L. Even inhalation alone at this level of lead in air would increase the blood level by about 4  $\mu$ g/L.
- The new evidence shows that effects on the central nervous system in children occur at, or below, the NZ guideline level.

## QC6. Is there any new evidence on health effects due to air emissions of polycyclic aromatic hydrocarbons that would impact on current target values?

- Some polycyclic aromatic hydrocarbons (PAHs) are potent carcinogens, and they are often attached to airborne particles, which may also play a role in their carcinogenicity.
- As PAHs are carcinogenic by a genotoxic mode of action, their levels in air should be kept as low as possible.
- Overall, there is no new evidence from which to propose a new target value. However, it should be noted that, based on previous literature, the existing target value of 1 ng/m<sup>3</sup> of benzo[*a*]pyrene is associated with the lifetime cancer risk of approximately 1 x 10<sup>-4</sup>.
- NB: Domestic heating is the key source of PAHs in New Zealand. Ambient levels in Christchurch consistently exceed the NZ AAQG by an order of magnitude.<sup>1</sup>

Environment Canterbury, 2009. Cavanagh JE 2009. <u>Ambient air polycyclic aromatic hydrocarbon (PAH)</u> concentrations in Timaru 2006–2007 (pdf)



<sup>&</sup>lt;sup>1</sup> Environment Canterbury, 2005. McCauley M 2005. <u>Ambient concentrations of polycyclic aromatic hydrocarbons and dioxins/furans in Christchurch – 2003/2004</u> (pdf). Report No. R05/14. Environment Canterbury.

- QC7. Is there any new evidence on the health effects of short-term (less than 1 day) exposures to SO2 that would lead to changes of the WHO air quality guidelines based on 10 minute and daily averaging periods or the EU's air quality limit values based on hourly and daily averaging periods?
  - 10-minute guideline: a small increase in the safety factor from the current value might be justified when the time comes to reconsider the guideline.
  - Daily guideline: The 24-hour average guideline does not need to be changed if the same method (using a concentration at the low end of the range of concentrations) is followed for setting the guideline.

## QC8. Are there important interactions among air pollutants in the induction of adverse health effects that should be considered in developing air quality policy?

- There is very little evidence from health studies that the mixture of air pollutants results in significantly more health effects (synergy) than would be expected based on the information for single pollutants. However, this is largely due to a lack of data and methodological limitations.
- Synergistic biological effects between ultrafine particles and transition metals and between particles and volatile organic compounds have been shown to indicate a larger combined impact on human health than would be expected from the separate entities.
- Airborne particles of any kind can carry aeroallergens or toxic condensed vapours, such that their impact can be substantially larger than without particles. There is a trend that the smaller the particles, the stronger the adjuvant effects. Limited evidence has been published suggesting that nitrogen dioxide can enhance allergic responses.
- In general, reduction of one component will not result in a significant increase in the health risks associated with other components. The implications for reducing PM, on (semi)volatile organic compound formation, are not evident.
- There is some evidence of interactions between pollutants and high temperature.
- Changing the air pollution mixture due to changing fuels may, under certain conditions, lead to more harmful emissions.



### QC9. Are there critical data gaps to be filled to help answer A, B and C questions more fully in the future?

- More epidemiological studies that contribute to updated exposure–response functions based on meta-analyses for integrated risk assessments.
- The coordinated application of atmospheric science, epidemiological, controlled human exposure and toxicological studies. Such studies should include better characterization of the pollution mix, improved exposure assessments and better identification of susceptible groups in the general population.
- Assessment of (currently regulated) pollutants together, as opposed to independently, including other size fractions and metrics of PM.
- Studies of health impacts of reducing traffic-related air pollution.
- Air pollution should be considered to be one complex mix, and conditions under which this mix has the largest effect on human health need to be identified.
- Studies using the *one-atmosphere* concept to investigate the effects on health of complex mixes.
- Atmospheric modelling, in conjunction with validation studies that use targeted monitoring campaigns, to research health effects.

# QC10. What is the contribution of exposure to ambient air pollution to the total exposure of air pollutants covered by the regulations, considering exposures from indoor environments, commuting and workplaces?

- This section is not as relevant for New Zealand (except Auckland) as it focuses on three primary routes of exposure (indoor, commuting and outdoor).
- However, it is important to note that solid-fuel-fired indoor fireplaces and stoves, where used under suboptimal conditions, dominate the high end of exposures to PM<sub>2.5</sub>, black carbon, ultrafine particles, carbon monoxide, benzene and benzo[a]pyrene of the individuals affected (excluding smoking).



### QD1. What new information from epidemiological, toxicological and other relevant research on health impacts of air pollution has become available that may require a revision of the EU air quality policy and/or WHO air quality guidelines notably for PM, ozone, NO<sub>2</sub> and SO<sub>2</sub>?

- In many cases, these (new studies) have shown associations with adverse health outcomes at pollutant levels lower than those in the studies on which the 2005 Global Update of the WHO air quality guidelines were based. This is particularly true for PM, ozone and nitrogen dioxide.
- In light of this, we would recommend that WHO begins the process of developing revisions to the earlier guidelines, with a view to completing the review by 2015.

### Particulate matter

- Recommend revising PM<sub>10</sub> and PM<sub>2.5</sub> daily and annual guidelines
- Since the 2005 Global Update of the WHO air quality guidelines, many new studies from around the world have been performed and published. These studies strengthen the evidence of a linear concentration– (exposure–)effect relationship without a threshold for various health outcomes associated with exposure to PM<sub>2.5</sub> and PM<sub>10</sub> (see answers to Questions A1, A4, and A5). The scientific literature shows also that PM<sub>10</sub> is not just a proxy measurement for PM<sub>2.5</sub>. Coarse and fine particles deposit mostly at different locations in the respiratory tract. The finer the particles are the deeper they can penetrate into the lungs. Independent effects of the coarse fraction are seen in epidemiological studies. The effects of PM<sub>coarse</sub> (10–2.5 µm) and PM<sub>fine</sub> (2.5 µm) may be related to different mechanisms (see Answer to Question A4). PM<sub>coarse</sub> and PM<sub>fine</sub> have different sources too, and the dispersion gradient near the source is different.

### Nitrogen dioxide

- Many studies, not previously considered or published since 2004, have documented associations between day-to-day variations in nitrogen dioxide concentration and variations in mortality, hospital admissions and respiratory symptoms. Also, more studies have now been published showing associations between long-term exposure to nitrogen dioxide and mortality and morbidity. ... Chamber and toxicological evidence provides some mechanistic support for a causal interpretation of the respiratory effects.
- As there is consistent short-term epidemiological evidence and some mechanistic support for causality, ... it is reasonable to infer that nitrogen dioxide has some direct effects.



### QD2. What evidence is available directly assessing health benefits from reducing air pollution?

- There is reasonably consistent evidence from past and more recent studies that decreased air pollution levels, following an intervention or unplanned decrement in pollution, have been associated with improvements in health.
- These findings are supported by a large body of remarkably coherent evidence from studies of both long and short-term exposure to air pollution.



### **3.0** Relevance for Public Health in New Zealand

The <u>Review of evidence on health aspects of air pollution</u> is authoritative, comprehensive and recent (WHO, 2013).<sup>2</sup> Importantly, it includes negative findings as well as positive associations. This increases its credibility.

Recent IARC Press Releases have provided a definitive backdrop to the WHO review:

- June 2012 <u>diesel engine exhaust</u> is carcinogenic to humans (Group 1)<sup>3</sup>
- October 2013 'outdoor air pollution' is carcinogenic to humans (Group 1)<sup>4</sup>
- Both due to increased risk of lung cancer

### **3.1** Key things to note

### **Particulate Matter**

- We should be assessing PM<sub>10</sub> and PM<sub>2.5</sub>
- We should be assessing short-term (i.e. 24 hour) and long-term (i.e. annual) effects of PM
- None of the above is currently required in national regulations or guidance

### Roads

- There is a significant body of research finding positive associations between proximity to busy roads and adverse health effects.
- ARPHS previously published guidelines for distances within which early childhood education centres may suffer poor air quality:
  - Within 150m of a motorway, truck route or other strategic route
  - Within 60m of a district or regional arterial road
  - Within 100m of a petrol station
  - Inside enclosed car parks, e.g. parking buildings
- Auckland Council has proposed separation distances for early childhood education centres and busy roads:
  - o 150 m for motorways and strategic arterial routes; and

<sup>&</sup>lt;sup>4</sup> http://www.iarc.fr/en/media-centre/pr/2013/pdfs/pr221\_E.pdf



<sup>&</sup>lt;sup>2</sup> http://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/2013/review-of-evidence-on-health-aspects-of-air-pollution-revihaap-project-final-technical-report

<sup>&</sup>lt;sup>3</sup> http://www.iarc.fr/en/media-centre/pr/2012/pdfs/pr213\_E.pdf

• 70 m for primary arterials.

### New findings

• Causal link with cancer and new research findings for adverse health effects (e.g. adverse birth outcomes) underline the importance of the issues

### Things to watch out for

- WHO guidelines for PM and nitrogen dioxide are likely to be reduced in future revisions.
- PAHs from wood burners is likely to be an indoor/outdoor issue that is not currently being addressed in New Zealand (primarily due to cost of monitoring).



### 4.0 References

- CARB, 2007. *Review of the California ambient air quality standard for nitrogen dioxide*. Sacramento, California Environmental Protection Agency Air Resources Board. <u>http://www.arb.ca.gov/research/aaqs/no2-rs/no2-doc.htm#TechSuppDoc</u>, accessed 31 Oct 2013
- Kuschel et al., 2012. Updated Health and Air Pollution in New Zealand Study. March, 2012. Data from exposure model available at: http://www.hapinz.org.nz/HAPINZ%20Update\_Exposure%20Model.xlsx, accessed 31 Oct 2013
- World Health Organisation, 2006. *Air Quality Guidelines Global Update 2005, Particulate matter, ozone, nitrogen dioxide and sulfur dioxide.* WHO Regional Office for Europe, Copenhagen Ø, Denmark.
- World Health Organisation, 2013. *Review of evidence on health aspects of air pollution REVIHAAP Project, Technical Report.* WHO Regional Office for Europe, Copenhagen Ø, Denmark.

