Committee on the Medical Effects of Air Pollutants

Statement on the evidence for health effects associated with exposure to non-exhaust particulate matter from road transport

Summary

1. COMEAP last reviewed the evidence for differential toxicity of particles in 2015 and acknowledged that there may be variations in toxicity between the various components of $PM_{2.5}$ (COMEAP, 2015). Since subsequent new evidence has emerged of the health impacts from traffic-produced particles, and following the report published by the Air Quality Expert Group (AQEG) on non-exhaust emissions (NEE) from road traffic (AQEG, 2019), the Committee considered that the evidence on non-exhaust particles from road transport and associated health effects should be re-evaluated. Non-exhaust particulate matter (PM) emissions, ie from brake wear, tyre wear, road surface wear and re-suspended road dust currently comprise just under 10% of UK primary particulate emissions and they are expected to become proportionately more important, as vehicle exhaust PM emissions from road transport are expected to decrease over the coming years.

2. Given that non-exhaust particles have a different composition (eg higher metal concentrations) and size distribution from those emitted in vehicle exhausts, they may have different toxicological properties and health consequences. However, epidemiological and toxicological studies undertaken to address this question to date differ widely in their methods and their ability to quantify possible health impacts.

3. Toxicological studies suggest that tyre and brake wear particles have the potential to induce biological effects at higher concentrations, but it is unclear whether real world exposures are high enough for this to be of concern for health. Epidemiological studies of health impacts are not consistent. Several of the studies reviewed found no associations of health outcomes with non-exhaust particles, others did report significant associations but covered a considerable range of different health effects. However, the likely strong correlation of non-exhaust particles with exhaust pollutants in ambient air makes it difficult to identify an independent effect of non-exhaust particles.

4. Taken as whole, the current body of published work is small and does not provide a compelling narrative of adverse health effects of exposure to non-exhaust particles from road transport. Nonetheless, given that there is strong evidence that the exposure to particulate pollutants in ambient air is harmful to health, it is likely that there is some health risk associated with exposure to non-exhaust particles. However, in the absence of any consistent evidence on the relative toxicity per unit

mass exposure of non-exhaust PM, there is insufficient evidence to provide a quantitative comment on the risk associated with inhalation of particles arising from the non-exhaust component of traffic emissions. As this component of traffic emissions will become proportionately more important in future years, it is recommended that new epidemiological and toxicological research should be undertaken to further understand the potential health risk of this aspect of vehicular pollution and to provide a basis for further policy.

Introduction

5. Particulate air pollution is a complex mixture of different components, some of which are expected to be more harmful to health than others. In epidemiological studies, there is heterogeneity in coefficients representing associations between particle mass concentrations and health effects, suggesting that compositional factors may be influential. There is also evidence suggesting that particles of differing composition elicit distinct toxicological responses and interact differently with different cell types.

6. Epidemiological studies have shown adverse health effects are associated with proximity to traffic, or exposure to traffic-derived pollutants, although the extent to which the associations are causal is uncertain (because of the possibility that other correlated risk factors, such as noise, might be responsible to some extent). These adverse health effects include cardiovascular hospitalisations and changes in lung function. Furthermore, metals included in emissions from non-exhaust sources, from brake and tyre wear for example, can catalyse the formation of reactive oxygen species and have been found to enhance the oxidative potential of particles, and hence may increase toxicity elicited via oxidative stress pathways.

7. Differential toxicity is one of the factors that need to be considered when evaluating the effectiveness of interventions that reduce particulate air pollution and in turn, influence health. Hence, the Committee previously reviewed the evidence regarding the differential toxicity of particles and acknowledged that there may be variations in toxicity between the various components of $PM_{2.5}$ (COMEAP, 2015); however, the evidence available did not give a consistent view of relative toxicity. Thus, $PM_{2.5}$ mass has remained the preferred metric for quantitative assessments of the health effects of exposure to particulate air pollution.

8. Considering the growing evidence of the health impacts from traffic-produced particles, and following the report published by the AQEG on NEE from road traffic (AQEG, 2019), the Committee considered that the evidence on non-exhaust particles and associated health effects should be reviewed and evaluated.

Background

9. Non-exhaust PM emissions from road traffic originate mainly from brake wear, tyre wear, road surface wear and re-suspended road dust in the wake of passing traffic. Potential additional sources include engine belts and clutch plates. These emissions have become a significant component of urban air pollution, and they are expected to become proportionately more important, as vehicle exhaust emissions of PM from road transport are expected to decrease over the coming years. In the UK,

emissions data from the National Atmospheric Emissions Inventory (NAEI)[1] in 2016 showed that non-exhaust particles are the main source of primary PM (by mass) from road transport, for both the $PM_{2.5}$ (60%) and PM_{10} (73%) size fractions, and contributed 8.5% and 7.4% of total primary PM_{10} and $PM_{2.5}$ emissions respectively (AQEG, 2019). As reported by AQEG, the NAEI data show that in recent years, vehicle exhaust emissions are decreasing alongside a slow increase in NEE emissions with rising traffic. As a consequence, NEE will contribute a much larger share of the total traffic emissions of particles and this trend is expected to continue in the future.

10. Table 1 gives percentage contributions of exhaust, brake and tyre wear, and road wear to total PM_{10} and $PM_{2.5}$ concentrations derived from the Pollution Climate Mapping (PCM) model. Averages across a selection of monitoring sites with co-located PM_{10} and $PM_{2.5}$ measurements are provided along with values for two specific monitoring sites (London North Kensington and London Marylebone Road). The average percentage results for background sites are consistent with modelled population-weighted mean background concentrations for the whole of the UK and are thus reasonably representative of average population exposure. Note that brake and tyre wear have been modelled together.

Table 1. Percentage contributions of non-exhaust particles to total modelled ambient concentrations. Source PCM model results for 2017, which is based on 2016 NAEI.

	PM_{10}			**$PM_{2.5}$**		
	Exhaust	Brake & tyre wear	Road wear	Exhaust	Brake & tyre wear	Road wear
Average of background sites (30 monitoring sites)	1	2	1	2	2	1
London North Kensington	2	6	2	4	5	2
Average of roadside sites (15 monitoring sites)	4	11	4	5	8	3
London Marylebone Road	6	16	6	9	13	5

11. Considerable measurement evidence exists that non-exhaust PM sources result in significant increases in concentrations of PM and some metals at roadside locations (AQEG, 2019). This is particularly evident in many urban environments, where roads are often characterised by (a) greater braking per kilometre and (b) higher traffic volumes. There is little current legislation related to the composition of brakes and tyres. Whilst a standard test for brake wear emissions is under development in anticipation of future guidance and regulation, tyre wear emissions are more difficult to measure.

12. It has been suggested that, because battery-electric vehicles (BEVs) are

[1] UK NAEI – National Atmospheric Emissions Inventory https://naei.beis.gov.uk/

heavier than equivalent internal combustion engine vehicles, this may imply greater abrasion emissions and dust resuspension. However, regenerative braking reduces brake wear and vehicle weight is understood to have only a small influence on dust resuspension. Nonetheless, tyre and road wear are likely be weight-dependent. Therefore, the net effect is uncertain (AQEG, 2019).

Physicochemical characteristics of non-exhaust PM

13. NEE cover a wide range of particle sizes, from ultrafine (<0.1 µm diameter) to beyond the PM_{10} size range (>10 µm). The NAEI uses ratios of $PM_{2.5}/PM_{10}$ for tyre wear of 0.7, brake wear of 0.4, and road abrasion of 0.54 (AQEG, 2019). Resuspended road dust particles also contribute to both the fine (<2.5 µm) and coarse (2.5-10 µm) size fractions, but are not included in the national inventory. Although there is evidence that both tyre wear and brake wear can generate ultrafine fraction particles, quantitative data on the contribution of these sources (tyre and brake wear) to the ultrafine fraction of the entire vehicle fleet is lacking. A measurement study carried out on Marylebone Road in London attributed 37% of NEE to resuspended dust, 53% to brake dust and 10% to tyre dust (Harrison et al., 2012). The method was unable to distinguish road surface wear from resuspension and both are reported in the resuspension total. The study measured size distributions, showing a slight predominance of coarse particles, most notable for the resuspension source.

14. Non-exhaust PM at the roadside is often characterised by elevated concentrations of metals (eg brake wear: copper, barium, antimony; tyre wear: zinc; dust from road surfaces: iron, aluminum) and is recognised as an important source of atmospheric metals. The UK emissions inventory estimates contributions of 47% and 21% to national total emissions of copper and zinc respectively.

Research questions

15. In view of the increasing importance of and interest in NEE from road transport, it was felt important to address the following questions:

- What evidence is available on the potential health effects of non-exhaust emitted particles?
- What conclusions can be drawn based on the available evidence?
- What further research is needed in this area?

16. In order to address these research questions, a rapid evidence review was carried out by searching for journal papers relevant to non-exhaust particle emissions and associated health effects, which identified 56 relevant studies (details are given in Annex A).

Health effects associated with exposure to non-exhaust PM from road transport

17. Non-exhaust particles are of a different composition and size distribution from

those emitted in engine exhaust and may have different toxicological properties and health consequences.

Epidemiological evidence

18. A number of epidemiological studies (16) have sought to measure associations between non-exhaust particle concentrations from road transport and adverse health outcomes. These include time-series and panel studies conducted in Europe (UK, Poland, Sweden, Spain, Finland), North America (the USA; Atlanta, New York, Los Angeles, Connecticut, Massachusetts, North Carolina), South America (Chile) and Asia (Hong Kong, South Korea). In these studies, the characterization of the pollutant sources was attained by application of source apportionment techniques, such as positive matrix factorization using chemical analysis of collected particle samples or by the use of dispersion models. The health outcomes that have been associated with brake wear, tyre wear and road dust are cardiovascular hospitalisations, changes in acute pulmonary and inflammatory responses, changes in lung function, emergency hospitalisation for respiratory causes, birth weight, heart failure, children's blood pressure, preeclampsia, changes in cardiac and blood parameters in adults, various cardiovascular symptoms, pulmonary diseases and mortality. A tabulated summary of the reviewed studies is provided in Annex B.

19. The studies differ widely in their methods, and their ability to quantify non-exhaust particles. Most of those that use receptor modelling techniques, such as positive matrix factorization (Riediker et al., 2004; Lall et al. 2011; Bell et al., 2013; Dadvand et al., 2014; Heo et al., 2014; Pun et al., 2017; Krall et al., 2018; Rich et al., 2019) are unable to distinguish the different types of non-exhaust particles, and tend to use a single descriptor, such as "road dust". Some use chemical tracers that are representative of wind-blown soils as well as road dusts, and are hence unable to distinguish natural soil particles from contaminated road dust (eg Adamiec et al., 2017). Several of the studies found no associations of health outcomes with non-exhaust particles (eg Desikan et al., 2015; D'Souza et al., 2017), and those that report statistically significant associations cover a considerable range of different health effects. Taken as whole, the current body of published work is small and does not provide a coherent and convincing narrative of adverse health effects of exposure to non-exhaust particles. This may be a consequence of the difficulty in providing a reliable quantification of non-exhaust particle concentrations, and the likely strong correlation of exhaust and non-exhaust pollutants in ambient air, which reduces the ability of epidemiological models to give reliable attribution to exhaust and non-exhaust particles respectively or the fact that small numbers of studies focus on a specific health endpoint.

Toxicological evidence

20. There is a general paucity of toxicological studies considering the potential health effects of the non-exhaust PM from road transport. 39 papers including 12 in vivo and 27 in vitro studies associating various health endpoints with tyre, brake or road wear were reviewed. A more detailed narrative summary can be found in Annex C, and a tabulated summary of the reviewed studies is provided in Annex D.

21. In vivo studies, mainly in rodents, largely focused on the effects of non-exhaust PM on the lungs, and occasionally blood biochemistry or blood biomarkers. Typically, the biological pathways explored/identified are those that are of known importance for urban PM and exhaust PM, eg oxidative stress and inflammation. There is considerable inconsistency between studies, but in most cases it appears that tyre or brake wear particles do have the potential to induce both inflammation and oxidative stress.

22. In vitro studies have employed a number of methodological designs, dose ranges and cell types, although monolayers of A549 cells (a model for human bronchial epithelial cells) are the most frequently studied. As with in vivo studies, there is inconsistency between findings, although the majority do show that (at higher concentrations of) non-exhaust PM can induce cytotoxicity, release of inflammatory cytokines and generate oxidative stress, eg oxidative modification of DNA.

23. While both in vivo and in vitro studies show that non-exhaust PM has the capacity to induce biological actions that are indicative of potential health effects, there are a number of caveats that should be emphasised:

1- There is considerable inconsistency between findings. While the published abstracts from these studies tend to emphasise the positive results, more detailed reading of the full papers show that there are often a similar number of endpoints that are not affected by non-exhaust PM.

2- In general, the significant effects tend to be found only at higher concentrations of non-exhaust PM. For example, in regard to inhalation studies in rodents, one study found that short-term exposure to high concentrations (6 hours at 9 mg/m^3 PM) of brake wear PM induced a modest lung inflammation and mild changes in indicators of systemic inflammation (Gerlofs-Nijland et al., 2019). In contrast, another study that used prolonged exposures of moderate doses (28 days at 0.1 mg/m^3 PM) did not find non-exhaust PM to have effects on any parameter studied (Kreider et al., 2012, see Annex D). An extrapolation of the values derived from the last study was published by Kreider et al. (2019), the results of which suggested that the no adverse effect level in humans was 55 μg/m^3; a level above that of many real-world measurements of non-exhaust PM. While the move towards risk assessment is valuable, there were limitations of research (see Annexes C and D) and the lack of strong signals of adversity is not sufficient in itself to conclude that there is no cause for concern.

3- As would be expected from the number of sources (ie tyre wear, brake wear or road wear and resuspended dust) and constituents, non-exhaust PM can have a varied size distribution. While there is a general assumption that non-exhaust PM is of a larger size distribution (coarse and the upper ranges of fine) than that of exhaust PM, in many cases there is also a small proportion of ultrafine PM present. The greater relative surface area of the ultrafine particles may make a significant contribution to the biological effects, although this has not been adequately addressed within the evidence reviewed. The degree of penetration to, and deposition in, the lungs is not adequately considered by in vitro models. Larger particle sizes are less likely to be translocated into the blood and organs compared to smaller particles in exhaust PM. Thus, the toxicity of the non-exhaust PM observed in studies investigating lung parameters may not be matched by similar effects in other organ systems.

4- Many studies use PM collected at roadside, which are then separated into different samples based on season of collection, sampling site or broad size fraction. Particle characterisation (usually content of specific metals) is performed to determine which samples are likely to be rich in non-exhaust PM compared to contributions from other sources. However, it should be noted that even samples with a higher proportion of non-exhaust PM overall would still contain PM from a mixture of other non-traffic sources. The proportion of non-exhaust PM from road transport is often not clear, and may vary depending on whether this is determined by mass or particle number.

5- There are very few papers that directly compare the biological effects of non-exhaust PM to other PM types. Isolated in vitro (Gustafsson et al., 2008) and in vivo (Gerlofs-Nijland et al., 2019) studies have shown that some types of non-exhaust PM could have greater effects than the same concentration of exhaust PM, but these observations were not consistent across different types of non-exhaust PM or the different assays used. Kreider et al. (2012) referred to unpublished data from pulmonary instillation of PM to rats showing that non-exhaust PM had no significant effect on lung inflammation, whereas diesel exhaust PM increased the number of inflammatory cells in the lung.

24. Overall, from the limited studies available, the evidence suggests that while non-exhaust PM from road transport can induce inflammation and oxidative stress in biological systems, the evidence as to whether this leads to ‘toxicological actions’ is varied. Where toxicological effects are observed, this may only be seen for some biological parameters and not others. Additionally, often effects are observed only at high doses that are beyond real-world scenarios. There is a need for further studies that directly compare toxicological actions of non-exhaust PM to exhaust PM and investigate the effects of non-exhaust PM in organ systems other than the lung.

Conclusions

25. The main conclusions that can be drawn from the previous discussion are outlined below:

i. There is strong evidence from epidemiological studies that adverse health effects are associated with proximity to traffic, traffic intensity or concentrations of traffic-related air pollutants.

ii. It is not clear to what extent NEE (eg arising from brake, tyre and road wear and resuspension of dust) contributes to the observed associations observed for traffic-related or other particulate pollution.

iii. Traffic volume is forecast to grow in the future, and current projections show consequent increases in NEE. The uptake of battery electric vehicles using regenerative braking may lead to a reduction in the brake wear component of NEE but this is currently unverified.

iv. The limited toxicological studies available suggest that particles from these sources could pose a hazard to health. However, it is not clear whether real-world concentrations of non-exhaust PM from road transport would have significant effects and also whether it would exert similar harm compared to pollutants from traffic exhaust.

v. Better understanding of the potential health risks is therefore needed to inform policy development.

Research recommendations

26. Research recommendations to improve knowledge on this topic through the conduct of toxicological or/and epidemiological studies are summarised below:

Epidemiological research

27. Further research is needed, which should include:

- time series studies using high quality receptor modelling to distinguish particle sources,
- cross-sectional studies that take advantage of more reliable estimates of atmospheric concentrations of non-exhaust particles from road transport (improved estimates are to be expected in the future, as more field measurements of emission factors for non-exhaust particles become available),
- Nested mechanistic research (functional measurements in specific organ systems, biomarkers, metabolomics, etc) to expand epidemiological evidence.

Toxicological research

28. Future research avenues for toxicological studies with non-exhaust PM from road transport include:

- studies using broader concentration ranges of non-exhaust PM, especially lower concentrations that are more representative of environmental scenarios,
- exploration of organ systems other than the lung,
- further exploration of the biological consequences of particle size ranges of non-exhaust PM,
- studies that directly compare toxicity of non-exhaust PM to the particulates from exhaust PM, and other particulate or traffic-related air pollutants,
- controlled inhalation exposures in human subjects to complement animal data and emerging epidemiological studies.

COMEAP
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Membership list of the COMEAP Sub-group on the health effects associated with exposure to non-exhaust particulate matter from road transport

Chair	Professor Frank Kelly
Members	Professor Roy Harrison
	Dr Mark Miller
	Mr John Stedman
	Dr Mike Holland
Secretariat	Dr Christina Mitsakou
	Ms Alison Gowers
	Dr Artemis Doutsi
	Dr Karen Exley