



Met Office

Air quality and health: a review of literature

For Government Services

October 2015

Christophe Sarran



Contents

Key messages	2
Health impacts of air pollution	2
Cardiovascular impact (coronary events, atrial fibrillation, ischemic stroke)	2
Lung cancer	4
Dry eye disease	4
Allergic respiratory disease (asthma)	4
Respiratory disease (other, e.g. chronic obstructive pulmonary disease)	5
Paediatric respiratory infections (bronchitis, pneumonia)	6
Cognitive function	6
The paediatric central nervous system.....	6
Requirements for model development	7
Policy implications.....	8
References.....	10

A search of literature on air pollution and health published in the past decade reveals a wealth of research comprised in thousands and tens of thousands of peer-reviewed papers. The review of the literature presented here is limited to published reviews that have already summarised results on the links between air pollution and health, and made recommendations on air quality policy, research and model development.

The purpose of this review is, firstly, to present the state of knowledge evidenced by measurements of the impacts of air pollution, and secondly, to make recommendations concerning policy implications and specifically on requirements for model development.

Key messages

- **The health impacts are multiple and complex, with contributions from both short and long term exposures from a multitude of pollutants and no known thresholds below which pollutant concentrations are safe.**
- **High resolution air quality models are required to address the needs of the health sector: these can be developed with a combination of statistical downscaling and adaptive grid refinement techniques.**
- **Investment in collaborative programmes of interdisciplinary research and service development involving stakeholders, scientists and policy makers is necessary to develop effective health protection interventions.**

Health impacts of air pollution

Cardiovascular impact (coronary events, atrial fibrillation, ischemic stroke)

Risk of mortality for cardiovascular disease has been reported to increase by 0.36% (95% CI [0.24%; 0.49%]) for each 10 $\mu\text{g}/\text{m}^3$ increase in short-term exposure to PM_{10} and 0.63% (95% CI [0.35%; 0.91%]) for each 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$ but was not associated with an increase of 10 ng/m^3 in nickel in $\text{PM}_{2.5}$, with a change in mortality risk of 0.60% (95% CI [-0.10%; 1.20%]). Morbidity risk increases by 0.37% (95% CI [0.17%; 0.56%]) for each 10 $\mu\text{g}/\text{m}^3$ increase in PM_{10} and 0.56% (95% CI [0.36%; 0.75%]) and 0.50% (95% CI [0.10%; 0.90%]) for each 10 $\mu\text{g}/\text{m}^3$ increase in $\text{PM}_{2.5}$. For long-term exposure, 23% (95% CI [19%; 26%]) and 55% (95% CI [51%; 60%]) increases in cardiovascular mortality risk were linked to 10 $\mu\text{g}/\text{m}^3$ increases in PM_{10} (Lu et al., 2015).

The specific composition of PM may be significant with cardiovascular mortality having been linked to potassium, OC, EC, sulphate, selenium, sodium, bromine, nitrate, nickel, vanadium, aluminium, arsenic, silicon and chloride; similarly, cardiovascular morbidity has been linked to OC, EC, oxygenated hydrocarbons, zinc, potassium, sulphate, selenium, bromine, nickel, aldehydes, calcium, copper, chromium, benzene and aluminium (Rohr et al., 2012).

The review by Costa et al. (2014) found that an increase of $10 \mu\text{g}/\text{m}^3$ in annual mean PM_{10} levels was associated with a 12% increased risk of coronary events ($\text{RR} = 1.12$, 95% CI [1.01; 1.25]), while the risk of atrial fibrillation and ischemic stroke increased within 1.5 to 2 hours of short term exposure (measured by P-wave complexity, a precursor). Long term exposure to PM is linked to atherosclerosis and short term exposure to plaque rupture, arrhythmia and acute cardiovascular events. A rise in mortality due to cardiopulmonary causes by 6% to 15% for $10 \mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$ is linked to chronic $\text{PM}_{2.5}$ exposure. For short term exposure, the excess risk of cardiovascular hospital admission is estimated between 0.6% and 1.7% for $10 \mu\text{g}/\text{m}^3$ of PM_{10} , 0.5% and 3.4% for $10 \mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$. Two to 24 h exposures were associated with myocardial infarction, arrhythmia and ischemic stroke.

Franchini et al. (2012) provide a comprehensive review of effects of air pollution on cardiovascular disease. Short term effects include rises of 0.7% of hospital admissions for ischemic heart disease, 0.8% for congestive heart failure, per $10 \mu\text{g}/\text{m}^3$ increase in PM_{10} . Acute myocardial infarction events were found to increase by 4.5% for each $10 \mu\text{g}/\text{m}^3$ of $\text{PM}_{2.5}$, and an increase in risk by 48% when exposure within 2 hours of symptoms rose by $25 \mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$. Increases in mortality and morbidity from atherothrombosis and stroke have been linked to PM and CO. Recently, a study has reported association of venous thromboembolism and pulmonary embolism with O_3 , SO_2 , NO_2 and PM. For long term effects, markers of atherosclerosis such as carotid intima-media thickness (increase by 4% per $10 \mu\text{g}/\text{m}^3$ $\text{PM}_{2.5}$) and aortic calcifications were correlated with $\text{PM}_{2.5}$ levels. A 70% ($\text{OR} = 1.70$, 95% CI [1.30; 2.23]) increase in risk of deep vein thrombosis has been estimated for $10 \mu\text{g}/\text{m}^3$ rises of $\text{PM}_{2.5}$ levels.

In toxicological studies, ultrafine carbon exposure has been linked to heart rate variability and cardiac repolarisation, $\text{PM}_{2.5}$ to reduced brachial artery diameter, decreased diastolic blood pressure, increased blood fibrinogen and reduced vascular function, and zinc to oxidative stress and inflammation (Cassee et al., 2013).

Lung cancer

Incidence of lung cancer is increased by 22% (RR = 1.22, 95% CI [1.03; 1.45]) per 10 $\mu\text{g}/\text{m}^3$ of chronic exposure to PM_{10} (reviewed by Costa et al., 2014). A review by Huang et al. (2014) found that lung cancer incidence and mortality are positively correlated with annual mean concentrations of NO_2 , with $p < 0.05$ for most years. Air pollution has also been recently classified as “carcinogenic to humans” as a leading cause of cancer deaths (International Agency for Research on Cancer, 2013).

Dry eye disease

While it is recognised by Alves et al. (2014) that the effect of air pollution on dry eye disease incidence and prevalence is “not entirely understood”, epidemiologic studies have found that dry eye disease has been linked to PM, CO_2 , NO_2 and elemental carbon.

Allergic respiratory disease (asthma)

A review by D’Amato et al. (2015) found that NO_2 exposure is associated with shortness of breath and increased allergic response to allergens and increased emergency hospital attendances of children with asthma. Exposure to O_3 by asthma sufferers is associated with worsening airway inflammation and increased risk of asthma exacerbation and emergency hospital attendances. Chronic exposure to high O_3 levels is linked to increased airway inflammation in healthy persons and may result in increasing prevalence of asthma. PM_{10} exposure has been associated with asthma exacerbations in children as well as increased prevalence rates. PM composition may be significant as acute exposure to diesel particles is linked lung function abnormalities and chronic exposure is associated with diminished lung function. Specific elements of PM linked to asthma morbidity include OC, EC, sulphate and water soluble metals (Rohr et al., 2012).

Kim et al. (2013) report health impacts on asthma sufferers for exposures to O_3 as low as 0.12 ppm for one hour, 0.2 to 0.6 ppm for NO_2 , 0.3 to 0.6 ppm for SO_2 and 2 mg/m^3 of VOC. There was no minimum reported for PM though increases by 10 $\mu\text{g}/\text{m}^3$ in the same day have an impact.

Costa et al. (2014) review of the health effects of NO_2 found a 1.37% (95% CI [0.59%; 2.15%]) increase in asthma hospital admissions for 10 $\mu\text{g}/\text{m}^3$ increases in 24-hour concentrations of NO_2 . Incidence of asthma is associated with long term high NO_2 levels with RR = 1.09 (95% CI [1.05; 1.14]) per 10 $\mu\text{g}/\text{m}^3$ NO_2 .

Lin and Zacharek (2012) argue that increases in CO₂, O₃ and NO₂ levels will cause a rise in the health burden for allergic respiratory diseases, for instance an increase of 7.3% in emergency asthma attendances related to Summer O₃ is expected in the next 10 years. Prevalence of hay fever was also linked CO, NO₂, SO₂ and PM.

Thunderstorm-related asthma crises are caused by the rupture of pollen grains after hydration, releasing inhalable allergen-carrying particles (D'Amato et al., 2015). Though infrequent, epidemic numbers of emergency attendances of subjects with asthma symptoms have been associated with thunderstorms coinciding with the pollen season.

Changes in the long term trends of pollen and spore concentrations due to lengthening seasons of allergen dispersion are estimated to worsen the morbidity rates of allergic disease (Lin and Zacharek 2012). Taxa mentioned include Juniperus, Quereus, Carya, Betula, Cryptomeria japonica and Artemisia for pollen, Cladosporium and Alternaria for spores.

Respiratory disease (other, e.g. chronic obstructive pulmonary disease)

A review by Kim et al. (2015) report that a 10 µg/m³ increase in PM₁₀ and PM_{2.5} was linked to a 1.6% (95% CI [0.4%; 2.9%]) increase in respiratory mortality. Lu et al. (2015) found that risk of mortality for respiratory disease increases by 0.42% (95% CI [0.28%; 0.55%]) for each 10 µg/m³ increase in short-term exposure to PM₁₀, 0.75% (95% CI [0.39%; 1.11%]) for each 10 µg/m³ increase in PM_{2.5}, 3.11% (95% CI 0.59%; 5.70%) and 1.42% (95% CI [0.41%; 2.45%]) for each 10 ng/m³ increase in nickel and vanadium in PM₁₀ and 0.90% (95% CI [0.20%; 1.70%]) for each 10ng/m³ increase in nickel in PM_{2.5}. Morbidity risk increases by 0.51% (95% CI [0.23%; 0.79%]) for each 10 µg/m³ increase in PM₁₀ and 0.80% (95% CI [0.44%; 1.14%]), 1.32% (95% CI [1.02%; 1.61%]) and 0.35% (95% CI [0.12%; 1.64%]) for each 10 µg/m³ increase in PM_{2.5}. Lagravinese et al. (2014) found that in Italy a 6% ± 3% rise in hospital admissions for under 14 year olds is associated with a 10 µg/m³ increase in PM₁₀ levels and a 5% ± 3% rise for over 65 year olds with a 10 µg/m³ increase in O₃ levels. Liu et al. (2014) reviewed the link between PM exposure and chronic bronchitis, in particular the concentration-response curves and their characteristics depending on population susceptibility.

Risk may depend on the specific composition of PM with respiratory morbidity having been linked to manganese, sulphate, water soluble metals and OC (Rohr et al., 2012).

Toxicological studies suggest that PM, NO₂ and NO_x are linked to impaired lung function (Cassee et al., 2013).

Paediatric respiratory infections (bronchitis, pneumonia)

Brugha et al. (2014) describe how pregnant mothers exposed to high levels of 24-hour mean outdoor PM_{2.5} (> 35 µg/m³) gave birth to babies 120 g lighter, and some whose mothers were exposed to even higher levels (> 45.9 µg/m³) suffered more bronchitis and pneumonia, perhaps due to underdeveloped lungs; it has been suggested that there is a link between low birth weight and prenatal exposure to air pollution (Lakshmanan et al., 2015). In children under 2 years, it was found that a 25 µg/m³ increase in 30-day mean PM_{2.5} is associated with a 30% increased risk of bronchitis (RR = 1.30, 95% CI [1.08; 1.58]). Short term (daily) fluctuations in PM_{2.5} organic carbon levels have also been linked with emergency respiratory hospital attendances with a 2 µg/m³ increase associated with a 3% rise in pneumonia cases of all ages. For children exposed to long term air pollution, PM₁₀ (adjusted OR = 1.76, 95% CI [1.00; 3.90], p = 0.051) and NO₂ (adjusted OR = 1.30, 95% CI [1.02; 1.65], p = 0.024) were linked to increased risk of pneumonia.

Cognitive function

As reviewed by Tzivian et al. (2015), several studies have linked air pollution to cognitive impairment: urban PM_{2.5} has been associated with cognitive decline, PM_{2.5} with lower verbal learning, O₃ with lower executive function, attention, information processing speed and verbal memory, NO₂ with lower logical memory and manganese with neurodegenerative disease; furthermore, associations have been reported of traffic NO_x levels with anxiety, PM₁₀, O₃ and NO₂ with increased emotional symptoms of depression and PM₁₀ with somatic and affective symptoms.

The paediatric central nervous system

Calderon-Garciduenas et al. (2015) highlight the impact of high levels of air pollution on the central nervous system. Residents exposed to chronic high levels of pollutions show increased risk of mortality with PM₁₀ and O₃, young children being most at risk. Ultrafine PM, PM-lipolysaccharides and metals are suspected to cause a systemic inflammatory response with mediators in the central nervous system. Neuroinflammatory changes are important in chronically exposed children, with symptoms present in 23.5% of residents under 25 years. Inhalation of toxic substances

is a cause of olfactory dysfunction which is associated with early Alzheimer and Parkinson diseases.

Requirements for model development

Brugha et al. (2014) note the benefits and limitations of air quality exposure models. Atmospheric dispersion models can provide good temporal resolution but are “Expensive” and have “Limited coverage” (low spatial resolution), while land use regression models provide suitable high spatial resolution chronic exposure levels but neglect short terms changes in exposure.

Similarly, Arrandale et al. (2011) describe the high resolution of land-use regression models (typically < 50 m) while regional air quality models provide temporal resolved solutions but over larger areas (typically > 1 km). Combining local scale and regional scale models is noted as an area of development. Better exposure assessment is noted to “lead to more precise exposure estimates as well as decreased measurement error and significantly increased study power”. Local scale exposure modelling better satisfies the advanced spatial methods of air pollution epidemiology (Auchincloss et al., 2012).

Sujaritpong et al. (2014) note that the high demand for computational resources in running high resolution air quality models “results in reduced opportunities to investigate the variability of climate models”. It is suggested to use statistical downscaling methods to overcome this challenge especially used in combination with statistical prediction models.

Garcia-Menendez and Odman (2011) argue the case for adaptive grid modelling as a suitable tool to increase resolution while minimising the impact on computational cost. Their review of adaptive grid modelling applications found “that dynamic mesh refinement significantly increases the accuracy of results” and they note that the differences in results between static and adaptive grids demonstrate the benefits of high resolution air quality modelling. Recommendations concerning the technique include statements such as: (1) “Adaptive gridding has consistently proven to be an adequate and highly attractive option to meet increase resolution requirement”; (2) “The potential returns of adaptive grids are enormous while the risks are relatively small”; (3) “the time is right to invest in the development of adaptive grid models”. Referring to Slingo et al. (2009), adaptive grid methods would provide a long term solution to multiscale climate models.

Policy implications

Kim et al. (2014) suggest that global climate change will increase the frequency of poor air quality episodes, in particular concerning O₃ and PM levels. A challenge outlined by Patz et al. (2014) is that the “Development of effective future policies will require understanding the relationship between climate change and health and developing approaches to ensure a sustainable future while protecting health”.

Jahn et al. (2011) argue that, even without epidemiological evidence, it is possible to extrapolate the potential reduction in the health impact from air pollution given suitable concentration-response functions. In particular, their review suggests 117 per 100,000 population reduction in premature deaths (95% CI [91; 164]) for the Chinese city of Guangzhou if the annual mean concentration of PM₁₀ was reduced to 40 µm/m³, 97 per 100,000 (95% CI [52; 137]) if that of PM_{2.5} was 25 µg/m³. Nevertheless, the review by Olmo et al. (2011) concludes that “even when atmospheric pollutants are within the legally established limits, they can be harmful to health”.

Three topics of future research that should be prioritised have been identified by Sujaritpong et al. (2014). These are “estimating future health impacts of extreme air pollution events”, the “behavioural adaptation of populations to cope with a warmer climate, which may modify exposure to air pollution” and “investigate interactions between temperature and air pollution” (though there may be other interactions worth investigating).

Brugha et al. (2014) suggest that future research should “Establish an evidence based approach to improving air quality”, in particular to elucidate whether policy should tackle selected pollutants or support uniform efforts to decrease urban traffic, while at the same time “Design strategies to protect vulnerable groups while waiting for air quality to be improved”. However, there are no means currently to calculate or optimise the risk-benefit ratios of effective yet complex possible interventions (Laumbach et al., 2015). Kelishadi and Poursafa (2010) note “that environmental protection actions, notably for reducing the emission of criteria air pollutants, should be considered for public health measures taken into account for primordial/primary prevention of chronic diseases”. Rohr et al. (2012) suggest that “If some components of PM and air pollution are of greater concern than others, it is important to identify them and for the risk management community to ensure that exposures to them are addressed accordingly”.

Concerning the impact of air pollution on the central nervous system, Calderon-Garciduenas et al. (2015) put forward questions concerning the patho-physiological pathway of the role of PM in paediatric neuroinflammatory responses and the suggested future public health projections in Alzheimer and Parkinson disease prevalence.

Weichenthal et al. (2013) reviewed the impact of polymorphisms in anti-oxidant genes on PM related cardiorespiratory morbidity concluding “further effort is required in evaluating how PM-oxidative burden may be incorporated in ambient air quality management”. Quoting Wild’s editorial of 2005, Arrandale et al. (2011) suggest that developing an exposome would complement the genome for air quality health impact studies and aid in the identification of vulnerable subgroups (e.g. in paediatric asthma). The exposome approach would support gene-environment interaction methods (Holloway et al., 2012) also known as Genome-Wide Interaction Studies (GWIS) which objectives include (1) “Obtaining better estimates of the population-attributable risk for different genetic and environmental risk factors”, (2) Strengthening observed associations between environmental factors and diseases” “to infer causality for environmental exposures” and (3) “Identification of key biological pathways that underlie response to environmental exposures providing both insight into which components of complex mixtures of pollutants cause disease as well as better understanding of pathogenesis allowing development of therapeutic strategies”.

Meta-analyses by Bell et al. (2013) found that mortality risk from PM exposure is statistically higher for older than for younger populations. The analyses also suggest that persons of lower socioeconomic status are at higher risk.

Costa et al. (2014) argue the case for an integrated approach to air quality policies. This would require knowledge of “the costs associated with their implementation”, of “economic benefits that might result from reduction of both quantities of pollutants emitted and their concentrations in the atmosphere” and of “health impacts and other possible benefits (or damages) arising from the adoption of the proposed strategies” including health equity concerns (Benmarhnia et al., 2014). Elsom (2004) goes further in arguing that the work of environmental scientists needs to be effectively integrated with that of policy-makers, planners and the wide range of stakeholders, along with an established development cycle to regularly update any air quality management system. This would require significant investment in staff and equipment in turn requiring political commitment. The measures that present a challenge (Kelly et al., 2012) are “(i) ongoing verification of evidence supporting a link between short-term changes in air pollution and

effects on health”, “(ii) defining whether such a relationship is causal”, “(iii) quantifying the size of the health impact” “following pollution increments that trigger an alert system” “and (iv) determining the magnitude of any reductions in adverse health outcomes”.

A recent report by the secretariat of the World Health Organization (2015) summarises many of these recommendations: “(a) Connecting health statistics to data on levels and sources of air pollution”; “(b) Strengthening monitoring of health outcomes related to air pollution”; “(c) Identifying expected risks to and benefits for health of policy interventions in the most polluting sectors, so as to identify interventions with the most health benefits”; “(d) Engaging in health diplomacy at national, local and international levels”; “(e) Identifying research priorities and supporting investigation of effective interventions in polluting sectors”; “(f) Communicating widely sector policies that offer the most health benefits, and cooperating on communication strategies at global, national and local levels”.

References:

- Alves, M., Novaes, P., Morraye, M. de A., Reinach, P.S., and Rocha, E.M., 2014 : Is dry eye an environmental disease? *Arquivos Brasileiros de Oftalmologia* **77**(3) 193-200.
- Arrandale, V.H., Brauer, M., Brook, J.R., Brunekreef, B., Gold, D.R., London, S.J., Miller, J.D., Ozkaynak, H., Ries, N.M., Sears, M.R., Silverman, F.S., and Takaro, T.K., 2011: Exposure assessment in cohort studies of childhood asthma. *Environmental Health Perspectives* **119**(5) 591-597.
- Auchincloss, A.H., Gebreab, S.Y., Mair, C., and Roux, A.V.D., 2012: A review of spatial methods in epidemiology, 2000-2010. *Annual Review of Public Health* **33** 107-122.
- Bell, M.L., Zanobetti, A., and Dominici, F., 2013: Evidence on vulnerability and susceptibility to health risks associated with short-term exposure to particulate matter: a systematic review and meta-analysis. *American Journal of Epidemiology* **178**(6) 865-876.
- Benmarhnia, T., Rey, L., Cartier, Y., Clary, C.M., Deguen, S., and Brousselle, A., 2014: Addressing equity in interventions to reduce air pollution in urban areas: a systematic review. *International Journal of Public Health* **59**(6) 933-944.

Brugha, R., and Grigg, J., 2014: Urban air pollution and respiratory infections. *Paediatric Respiratory Reviews* **15**(2) 194-199.

Calderon-Garciduenas, L., Kulesza, R.J., Doty, R.L., D'Angiulli, A., and Torres-Jardon, R., 2015: Megacities air pollution problems: Mexico City Metropolitan Area critical issues on the central nervous system pediatric impact. *Environmental Research* **137** 157-169.

Cassee, F.R., Heroux, M.E., Gerlofs-Nijland, M.E., and Kelly, F.J., 2013: Particulate matter beyond mass: recent health evidence on the role of fractions, chemical constituents and sources of emission. *Inhalation Toxicology* **25**(14) 802-812.

Costa, S., Ferreira, J., Silveira, C., Costa, C., Lopes, D., Relvas, H., Borrego, C., Roebeling, P., Miranda, A.I., and Teixeira, J.P., 2014: Integrating health on air quality assessment – review report on health risks of two major European outdoor air pollutants: PM and NO₂. *Journal of Toxicology and Environmental Health, Part B: Critical Reviews* **17**(6) 307-340.

D'Amato, G., Baena-Cagnani, C.E., Cecchi, L., Annesi-Maesano, I., Nunes, C., Ansotegui, I., D'Amato, M., Liccardi, G., Sofia, M., and Canonica, W.G., 2013: Climate change, air pollution and extreme events leading to increasing prevalence of allergic respiratory diseases. *Multidisciplinary Respiratory Medicine* **8** 12.

Elsom, D.M., 2004: Developing an appropriate scientific and decision-making framework for effective air quality management. Air Pollution XII, *Proceedings of the 12th International Conference on Modelling, Monitoring and Management of Air pollution* **14** 213-223, Rhodes.

Franchini, M., Guida, A., Tufano, A., and Coppola, A., 2012: Air pollution, vascular disease and thrombosis: linking clinical data and pathogenic mechanisms. *Journal of Thrombosis and Haemostasis* **10**(12) 2438-2451.

Garcia-Menendez, F., and Odman, M., T., 2011: Adaptive grid use in air quality modeling. *Atmosphere* **2**(3) 484-509.

Holloway, J.W., Francis, S.S., Fong, K.M., and Yang, I.A., 2012: Genomics and the respiratory effects of air pollution exposure. *Respirology* **17**(4) 590-600.

- Huang, Y.B., Song, F.J., Liu, Q., Li, W.Q., Zhang, W., and Chen, K.X., 2014: A bird's eye view of the air pollution-cancer link in China. *Chinese Journal of Cancer* **33**(4) 176-188.
- International Agency for Research on Cancer, 2013: IARC: outdoor air pollution a leading environmental cause of cancer deaths. Press release no. 221, 17 October 2013. Lyon, IARC.
- Jahn, H.J., Schneider, A., Breitner, S., Eissner, R., Wendisch, M., and Kramer, A., 2011: Particulate matter pollution in the megacities of the Pearl River Delta, China – a systematic literature review and health risk assessment. *International Journal of Hygiene and Environmental Health* **214**(4) 281-295.
- Kelishadi, R., and Poursafa, P., 2010: Air pollution and non-respiratory health hazards for children. *Archives of Medical Science* **6**(4) 483-495.
- Kelly, F.J., Fuller, G.W., Walton, H.A., and Fussell, J.C., 2012: Monitoring air pollution: use of early warning systems for public health. *Respirology* **17**(1) 7-19.
- Kim, K.H., Jahan, S.A., and Kabir, E., 2013: A review on human health perspective of air pollution with respect to allergies and asthma. *Environment International* **59** 41-52.
- Kim, K.H., Kabir, E., and Jahan, S.A., 2014: A Review of the consequences of global climate change on human health. *Journal of Environmental Science and Health Part C – Environmental Carcinogenesis and Ecotoxicology Reviews* **32**(3) 299-318.
- Kim, K.H., Kabir, E., and Kabir, S., 2015: A review on the human health impact of airborne particulate matter. *Environment International* **74** 136-143.
- Lagravinese, R., Moscone, F., Tosetti, E., and Lee, H., 2014: The impact of air pollution on hospital admissions: evidence from Italy. *Regional Science and Urban Economics* **49** 278-285.
- Lakshmanan, A., Chiu, Y.H.M., Coull, B.A., Just, A.C., Maxwell, S.L., Schwartz, J., Gryparis, A., Kloog, I., Wright, R.J., and Wright, R.O., 2015: Associations between prenatal traffic-related air pollution exposure and birth weight:

modification by sex and maternal pre-pregnancy body mass index.

Environmental Research **137** 268-277.

Laumbach, R., Meng, Q., and Kipen, H., 2015: What can individuals do to reduce personal health risks from air pollution? *Journal of Thoracic Disease* **7**(1) 96-107.

Lin, G.C., and Zacharek, M.A., 2012: Climate change and its impact on allergic rhinitis and other allergic respiratory diseases. *Current Opinion in Otolaryngology and Head and Neck Surgery* **20**(3) 188-193.

Liu, L., Yu, L.Y., Mu, H.J., Xing, L.Y., Li, Y.X., and Pan, G.W., 2014: Shape of concentration-response curves between long-term particulate matter exposure and morbidities of chronic bronchitis: a review of epidemiological evidence. *Journal of Thoracic Disease* **6** S720-S727.

Lu, F., Xu, D., Cheng, Y., Dong, S., Guo, C., Jiang, X., and Zheng, X., 2015: Systematic review and meta-analysis of the adverse health effects of ambient PM_{2.5} and PM₁₀ pollution in the Chinese population. *Environmental Research* **136** 196-204.

Olmo, N.R.S., Saldiva, P.H.N., Braga, A.L.F., Lin, C.A., Santos, U. P., and Pereira, L.A.A., 2011: A review of low-level air pollution and adverse effects on human health: implications for epidemiological studies and public policy. *Clinics* **66**(4) 681-690.

Patz, J.A., Frumkin, H., Holloway, T., Vimont, D.J., and Haines, A., 2014: Climate change challenges and opportunities for global health. *Journal of the American Medical Association* **312**(15) 1565-1580.

Rohr, A.C., and Wyzga, R.E., 2012: Attributing health effects to individual particulate matter constituents. *Atmospheric Environment* **62** 130-152.

Sujaritpong, S., Dear, K., Cope, M., Walsh, S., and Kjellstrom, T., 2014: Quantifying the health impacts of air pollution under a changing climate – a review of approaches and methodology. *International Journal of Biometeorology* **58**(2) 149-160.

Tzivian, L., Winkler, A., Dlugaj, M., Schikowski, T., Vossoughi, M., Fuks, K., Weinmayr, G., and Hoffmann, B., 2015: Effect of long-term outdoor air pollution and noise on cognitive and psychological functions in adults. *International Journal of Hygiene and Environmental Health* **218**(1) 1-11.

Weichenthal, S.A., Godri-Pollitt, K., and Villeneuve, P.J., 2013: PM_{2.5}, oxidant defence and cardiorespiratory health: a review. *Environmental Health* **12** 40.

World Health Organization, 2015: Health and the environment: addressing the health impact of air pollution. Sixty-Eighth World Health Assembly, provisional agenda item 14.6, 10 April 2015. Geneva, WHO.

Met Office
FitzRoy Road, Exeter
Devon EX1 3PB
United Kingdom

Tel (UK): 0870 900 0100 (Int) : +44 1392 885680
Fax (UK): 0870 900 5050 (Int) :+44 1392 885681
enquiries@metoffice.gov.uk
www.metoffice.gov.uk