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Prepared by:

Ian Yang, Annalicia Vaughan, Felicia Goh
UQ Thoracic Research Centre, The University of Queensland & The Prince Charles Hospital
Zoran Ristovski, Branka Miljevic, Svetlana Stevanovic
International Laboratory for Air Quality & Health, Queensland University of Technology
Brisbane, Australia
Experimental determination of the respiratory tract deposition of diesel combustion particles in patients with chronic obstructive pulmonary disease.

Authors: Londahl J, Swietlicki E, Rissler J, et al.
Reference: Part. Fibre Toxicol. 2012; 9: 30
URL: [http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3464711/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3464711/)
Comments: Patients with COPD are more susceptible to air pollution than healthy individuals, which is likely due to the difference in breathing patterns and lung morphology. This paper assesses the differences in respiratory tract deposition in COPD patients and investigates relationships between deposition, breathing parameters and lung function. This study experimentally exposed seven healthy patients and ten COPD patients to diesel emissions generated by either idling or transient driving in an exposure chamber. The respiratory tract deposition of particles ranging between 10-500nm was measured during spontaneous breathing. This study showed that COPD patients had a higher deposition dose rate (deposited dose per hour) of diesel exhaust particles related to impairment in lung function, primarily due to increased minute ventilation. This increased deposition may partly explain the adverse effects of air pollutant exposure for patients with COPD.

How air pollution influences clinical management of respiratory diseases. A case-crossover study in Milan.

Authors: Santus P, Russo A, Madonini E, et al.
URL: [http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3511062/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3511062/)
Comments: It has been proposed that inhaled air pollutants can trigger inflammatory responses within the lungs, which can lead to the development and exacerbation of lung disease. This study aimed to investigate any correlation between emergency room admissions (ERAs) for respiratory disease and environmental pollutant levels. Over a two-year period, the levels of environmental polyaromatic hydrocarbons, particulate matter, and various gaseous pollutants were measured. This data was then cross referenced with the recorded ERAs due to respiratory diseases on this time period. The study included 45770 ERAs for respiratory diseases including pneumonia, COPD, asthma and upper respiratory tract infections. Overall the study showed a positive association between ERAs for respiratory diseases and exposure to environmental pollutants. In reference to COPD, the results showed a significant association between ERAs for COPD exacerbations and environmental increases in sulphur dioxide, carbon monoxide and particulate matter (PM10).
**Associations of ambient air pollution with chronic obstructive pulmonary disease hospitalization and mortality.**

**Authors:** Gan WQ, FitzGerald JM, Carlsten C, et al.  
**Comments:** Recent research has suggested that ambient air pollution is associated with COPD. However, there is a scarcity of longitudinal studies to support this assertion. The aim of this study was to investigate the relationships between long-term exposure to ambient air pollution and the risk of COPD hospitalization and mortality. This study was conducted using a large population-based cohort of 467,994 subjects. The individual's exposure to ambient air pollutants was estimated by the person’s post-code and recorded air pollutant levels for that area. There was a five-year exposure period and a four-year follow-up period for this study. During the follow-up period, COPD hospitalizations and deaths were identified from a linked health database. Overall, this study showed that long-term exposure to elevated fine particulate matter derived from traffic emissions, specifically carbon black, was associated with a 6% increase in risk of COPD hospitalizations and 7% increase in COPD mortality. Exposure to woodsmoke was also associated with increased COPD hospitalizations. These results provide further evidence that exposure to air pollution is associated with increased risk of COPD.

**Evaluation of interventions to reduce air pollution from biomass smoke on mortality in Launceston, Australia: retrospective analysis of daily mortality, 1994-2007.**

**Authors:** Johnston FH, Hanigan IC, Henderson SB, et al.  
**Reference:** BMJ. 2013; 345: e8446  
**URL:** [http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3541469/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3541469/)  
**Comments:** Relatively few large studies of public health interventions for improving ambient air quality have been reported. This epidemiological study was undertaken in the city of Launceston, in the southern state of Tasmania in Australia, where government interventions were implemented to reduce the use of indoor wood stoves. These campaigns and regulations were designed to systematically replace indoor wood heaters with electric heaters. The interventions were associated with a reduction in the annual mean concentration of PM10 from 23.7 μg/m3 in 1997-2000, to a level of 18.4 μg/m3 after the intervention in the period 2001-07. There was a corresponding trend to decreased annual mortality in the population of the city, compared to another city which had no intervention. In males there was significantly reduced annual mortality for all cause (~11.4%), cardiovascular (~17.9%) and respiratory (~22.8%) mortality, including...
during winter. The authors concluded that reduced air pollution from biomass smoke was associated with reduced annual mortality in males and with reduced cardiovascular and respiratory mortality during winter months. This study suggests that coordinated interventions to reduce air pollution from use of biomass fuel have substantial public health benefits.

New Impact factor and ranking for Respirology released June 2013

Edited By: Peter Eastwood
Impact Factor: 2.781
ISI Journal Citation Reports ©
Ranking: 2012: 18/50 (Respiratory System)
Online ISSN: 1440-1843

Indoor air pollution and risk of lung cancer among Chinese female non-smokers.

Reference: Cancer Causes Control. 2013; 24: 439-50
URL: http://link.springer.com/article/10.1007%2Fs10552-012-0130-8
Comments: Although smoking is the highest risk factor in developing lung cancer, lung cancer incidence in some populations of non-smokers, such as Chinese females, is high. Indoor air pollution from passive smoking, a high frequency of cooking and solid fuel usage for cooking and heating has been proposed as a contributing risk factor in these instances. This study investigates the effects of household particulate matter (PM) and sources of indoor air pollution exposure on the risk of lung cancer, using newly diagnosed lung cancer patients in Taiyuan city compared to an age- and gender-matched control population. There was a high correlation between high ambient PM and poor housing ventilation in lung cancer incidence in non-smoking females. In particular, ultrafine PM (PM1; with particles ≤0.1μm) levels were higher in the living room, bedroom and kitchen of case houses compared to controls (p<0.05). This was supported by a trend in dose response, where a 10μg/m3 increase in concentration of PM increased the risk of lung cancer (p<0.01). In addition, total suspended PM concentration was correlated with exposure to solid heating fuel, lack of ventilator usage and particularly solid cooking fuel (p<0.05). Thus, the results from this study indicate that PM concentration is an indicator of indoor air pollution exposure and may be a contributing risk factor for lung cancer.
Long-term Residential Exposure to Air Pollution and Lung Cancer Risk

Authors: Hystad P, Demers PA, Johnson KC, et al.
URL: http://www.ncbi.nlm.nih.gov/pubmed/23676262
Comments: There is increasing evidence that exposure to air pollution may increase the risk of lung cancer. This study investigates the effects of ambient air pollutants on lung cancer incidence in Canada over 20 years. The cases selected for the study were taken from cancer registries spanning 1994-1997, compared against appropriately matched population controls, with models developed to estimate annual fine particulate matter (PM2.5), nitrogen dioxide (NO2) and ozone (O3) exposures and proximity to highways and major roads. For every 10μg/m3 increase in concentration of PM2.5, NO2 and O3, the adjusted odds ratios (ORs) of lung cancer incidence increased (1.29, 1.11 and 1.09 respectively), indicating that ambient air pollutants increase lung cancer incidence. However, there were no clear dose response trends. There was also an increasing trend in lung cancer incidence with those living closer to highways and major roads, although further evidence would be required to confirm this link. Finally, no associations were established between lung cancer incidence and specific histological subtype. Overall, the results from this study suggest that air pollution exposure increases the risk of lung cancer and further exploration into this area is warranted.

Respiratory health effects of diesel particulate matter.

Authors: Ristovski ZD, Miljevic B, Surawski NC, et al.
Reference: Respirology 2012;17:201-12.
URL: http://onlinelibrary.wiley.com/doi/10.1111/j.1440-1843.2011.02109.x/abstract;jsessionid=BEBC9B52A9F5319B3AC1EBC0D1BE6526.d02t01
Comments: Particulate matter (PM) emissions involve a complex mixture of solid and liquid particles suspended in a gas, where it is noted that PM emissions from diesel engines are a major contributor to the ambient air pollution problem. Whilst epidemiological studies have shown a link between increased ambient PM emissions and respiratory morbidity and mortality, studies of this design are not able to identify the PM constituents responsible for driving adverse respiratory health effects. This review in Respirology explores in detail the physico-chemical properties of diesel particulate matter (DPM), and identifies the constituents of this pollution source that are responsible for the development of respiratory disease. In particular, this review shows that the DPM surface area and adsorbed organic compounds play a significant role in manifest-
Comments: Due to definite supply of fossil fuels, there is an increasing need for alternative energy sources and in that context, a variety of biomass-based diesel (i.e. biodiesel) is already being introduced in diesel engines. While diesel exhaust emissions are known to cause adverse effects on human health and the International Agency for Research on Cancer (IARC) has recently evaluated diesel exhaust as carcinogenic to humans, there is very limited information on the toxicological properties of the emissions resulting from these new fuels. In this study, the toxicological effects of particulate matter (PM) generated by a heavy-duty diesel engine fuelled with conventional diesel and two biofuels (namely, rapeseed methyl-ester and hydrotreated vegetable oil) were investigated. Tests were performed with and without an oxidation catalyst mounted on the exhaust pipe. Mice macrophage cell line (RAW 264.7) was exposed to several doses of exhaust PM and the cellular parameters investigated included production of inflammatory mediators (TNFα and MIP-2), cytotoxicity (MTT, cell cycle, apoptosis, necrosis), genotoxicity (Comet assay) and formation of reactive oxygen species (ROS). Considerable differences in the toxic potential between emission PM collected from the diesel engine powered by different fuel and oxidation catalyst combinations. The oxidation catalyst decreased substantially PM mass emissions with conventional diesel and vegetable oil biodiesel, and in most cases, also the toxic activities of the PM were reduced.

In most parameters, the toxicological potencies of emission PM with the use of vegetable oil biodiesel were weaker than those obtained with conventional diesel and the same trend was observed in fewer parameters with rapeseed methyl ester biodiesel. The authors point out that the results of this study show different trends comparing to the results from their similar study, just performed on a different engine, concluding that the engine type seems to affect the toxicity of emitted PM. Furthermore, they emphasize that the harmfulness of the exhaust emissions can-
not be determined merely on the basis of emitted PM mass, i.e. that even a low PM mass emissions might harbor a significant health-relevant toxic potential.

Responses of lung cells to realistic exposure of primary and aged carbonaceous aerosols.

Authors: Künzi, L., Mertes, P., Schneider, S. et al.
URL: http://www.sciencedirect.com/science/article/pii/S1352231012011235
Comments: One of the main contributors to poor air quality in major cities are combustion sources. Among them exhaust from diesel powered vehicles and wood burning are becoming the major contributors to the ambient particle loading due to the increased number of diesel cars and the reintroduction of wood as a renewable energy source. Once the particles from these major sources are emitted in the urban environment they usually spend significant amount of time airborne before they are inhaled into the human body. During this time a number of photochemical oxidation processes can significantly modify the chemical and physical properties of the carbonaceous combustion particles.

Most of the cell exposure studies so far were done on particle extracts from filters and very often at unrealistically high concentrations. In this study the authors exposed airway epithelial cells and luminal macrophages to both freshly emitted and photochemically aged carbonaceous particles under realistic atmospheric conditions. The exhaust from a passenger diesel car and a wood stove, burning beech logs, was introduced into a large (27 m³) Teflon chamber and illuminated with UV lights to simulate atmospheric photochemical ageing processes. The single exposure of the cells induced small particle specific response, with diesel exhaust affecting the cells more than wood combustion. The exposure to aged particles induced the same response as long as it was from the same source. The authors clearly demonstrated the possibility of a cell exposure system that would closely mimic realistic atmospheric conditions.
Hazardous components and health effects of atmospheric aerosol particles: reactive oxygen species, soot, polycyclic aromatic compounds and allergenic proteins.

Authors: Manabu Shiraiwa, Kathrin Selzle, Ulrich Pöschl

Comments: This review is aimed to identify hazardous substances that are commonly associated with ambient pollution and related to that, various adverse health effects including allergies, asthma, cardiovascular and respiratory diseases. It investigates formation and chemical transformation of reactive oxygen species (ROS), soot, polycyclic aromatic hydrocarbons (PAHs) and allergenic proteins. Also, reactive oxygen intermediates (ROIs) have been introduced as newly discovered products in the reaction of ozone with aerosol particles. These compounds are assumed to have a very important role in the formation of toxic organic aerosols.

During the process of chemical aging, primarily generated components are being oxidised in the presence of ozone, nitrogen dioxide, hydroxyl, nitrite and halogen radicals. These processes are altering chemical composition as well as their volatility, hygroscopicity, toxicity and allergenicity. In addition, recent investigations indicate that allergenic proteins are prone to nitration by ozone and nitrogen dioxide which may lead to inflammation and explain elevated number of reported allergic reactions among population in polluted areas. The better understanding and assessment of negative health effects of aerosols requires multidisciplinary research that will eventually yield an integrated knowledge which will further allow optimized air quality control and improved medical treatments.

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