

APSR RESPIRATORY UPDATES



Volume 10 Issue 5

Newsletter Date: May 2018

APSR EDUCATION PUBLICATION



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Articles selected and commented on by: **Tara Kuhn and Zoran Ristovski** (International Laboratory for Air Quality & Health, Queensland University of Technology); **Ian Yang, Annalicia Vaughan and Hannah O'Farrell** (UQ Thoracic Research Centre, The University of Queensland & The Prince Charles Hospital); Brisbane, Australia.



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Respiratory and cardiovascular responses to walking down a traffic-polluted road compared with walking in a traffic-free area in participants aged 60 years and older with chronic lung or heart disease and age-matched healthy controls: a randomised, crossover study

Authors: Sinharay R, Gong J, Barratt B et al.

Reference: The Lancet, 2017, Volume 391, Issue 10118, Pages 339-349

URL: [https://doi.org/10.1016/S0140-6736\(17\)32643-0](https://doi.org/10.1016/S0140-6736(17)32643-0)

Comments: A study conducted in London between October 2012 and June 2014 has found evidence that walking in a polluted, high density traffic area loses its benefits of decreasing arterial stiffness in healthy participants. The study focuses on 119 participants, 60 years and older. 40 were healthy controls, 40 had chronic obstructive pulmonary disease (COPD) and 39 had ischaemic heart disease (IHD). Participants spent two hours walking in a randomly chosen location, either at a traffic dense end of Oxford Street, London, or through Hyde Park, where after their walk they were monitored for cough, sputum, wheeze, shortness of breath, lung function (FEV1/FVC), and arterial stiffness. All participants who walked through Hyde Park showed a notable increase in lung function and decrease in arterial stiffness. Healthy participants showed significantly higher lung function after walking in Hyde Park than on Oxford Street. Participants with COPD reported significantly higher cough, sputum, shortness of breath, and wheeze after walking down Oxford Street, attributed to by an increase of PM2.5, PM10, black carbon, ultrafine particles and NO2 in the air. In IHD patients who were not on medication showed a significant increase in arterial stiffness on Oxford street when compared to Hyde Park. This study provides clear evidence that the benefits of pulmonary and cardiovascular function are lost in a polluted environment, and that fossil fuel combustion particles are particularly toxic to those with pulmonary and cardiovascular diseases.

Association of ambient pollution with inhaler use among patients with COPD: a panel study

Authors: Magzaman S, Oron A, Locke E et al.

Reference: Occupational and Environmental Medicine. 2018. Epub ahead of print

URL: <http://oem.bmj.com/content/75/5/382>

Comments: Epidemiological research has shown that short term exposure to ambient air pollution is associated with COPD exacerbations. Previous research has evaluated the relationship between pollution and exacerbations in relation to emergency department visits, hospitalisation and mortality rates. However, COPD is associated with symptoms such as cough, dyspnoea or sputum production for which patients may not seek care, but can de-

crease lung function and diminish quality of life. This study has investigated the relationship between ambient air pollution exposure and worsening COPD symptoms by assessing the frequency of inhaler use. Thirty five COPD patients received a portable electronic inhaler sensor to record the date, time and location for short-acting beta-2-agonist (SABA) use over a 3-month period. Ambient air pollution data and meteorological data were collected from a centrally located federal monitoring system. Four criteria pollutants (PM_{2.5}, PM₁₀, O₃ and NO₂), two particulate matter species (elemental carbon and organic carbon), estimated coarse fraction of PM₁₀ (PM_{10-2.5}), and four multipollutant air quality measures were each examined. This study found that the average SABA use was 2.8 puffs/day. After controlling for supplemental oxygen use, long acting anticholinergic use, dyspnoea and influenza season, increases in PM₁₀, NO₂ and PM_{10-2.5}, were significantly related increase SABA use. Multipollutant models indicated that total pollution and weather were also significantly associated with increased SABA use. These findings suggest that air pollution has a significant impact on the respiratory symptoms of COPD patients. Further research is needed to understand the relationship between air pollution and worsening symptoms that do not lead to ED visits and hospitalisation but could adversely impact quality of life.

Occupational exposure to diesel engine exhaust and alterations in immune/inflammatory markers: a cross sectional molecular epidemiology study in China

Authors: Bassig B, Dai Y, Vermeulen R et.al.

Reference: Carcinogenesis, 2017, 38(11), pg 1104-1111

URL: <https://doi.org/10.1093/carcin/bgx081>

Comments: Diesel engine exhaust (DEE) is classified as a human carcinogen. A recent nested case-control study has identified 10 immune/inflammatory markers associated with future lung cancer risk. The aim of this study was to identify specific marker associated with both lung cancer risk and occupational exposure to DEE, in order to provide insight into the underlying biologic processes by which DEE causes lung cancer. The study consisted of 54 males workers exposed to DEE while working at a diesel engine manufacturing facility and 55 unexposed male control workers from the same local area as the exposed workers. The exposed workers were exposed to DEE for an average of 20 years. Blood samples were collected from the participants after their work shift. 64 immune/inflammatory markers from blood plasma were measured. Of the 10 immune/inflammatory markers associated with lung cancer risk, the plasma levels of 9 markers were altered in the DEE exposed workers. After correcting for multiple comparisons, DEE exposure was associated with decreased levels of CRP and increased levels of MIP-1D. In former and never smok-

ers, an increase in plasma MCP-1 level was also associated with DEE exposure. These immune markers have been associated with both lung cancer risk and long term occupational exposure to DEE. The results of this study may provide some insight into the underlying biologic mechanisms of the DEE-lung cancer association, but will require replication in the larger studies of DEE exposure.

Association between Household Air Pollution Exposure and Chronic Obstructive Pulmonary Disease Outcomes in 13 Low- and Middle-Income Country Settings

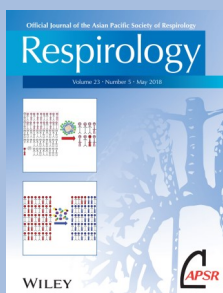
Authors: Siddharthan T, Grogby M, Goodman D et al

Reference: American Journal of Respiratory and Critical Care Medicine, 2018, Vol. 197, No. 5

URL: <https://doi.org/10.1164/rccm.201709-1861OC>

Comments: Combustion of biomass for energy in households is a major cause of indoor air pollution worldwide. Pooled data from 12,396 adults in low to middle income countries of Latin America, Sub-Saharan Africa, and Southeast Asia examined exposure to household air pollution and health outcomes in COPD. ~9% of this study population had COPD. The risk of having COPD was 41% higher (odds ratio 1.41, 95% confidence interval 1.18-1.68) in those with exposure to household air pollution, compared to those without exposure. The association was stronger in women than in men. It was estimated that in these populations, 13.5% of COPD prevalence was linked to household air pollution, compared to 12.4% related to cigarette smoking. By their very nature, these types of observational studies are frequently cross-sectional, rely on self-reported biomass burning, and differ in consistency of results across different studies. Nevertheless this study points to the need for further evidence about the risk of developing COPD with long term exposure to household air pollution.

APSR PUBLICATIONS



Edited By: Philip Bardin and Paul Reynolds

Impact Factor: **3.256**

ISI Journal Citation Reports ©

Ranking:2015: **18/59** (Respiratory System)

Online ISSN: 1440-1843



Edited By: Christopher Lai

Online ISSN: 2051-3380

Chronic obstructive pulmonary disease associated with biomass fuel use in women: a systemic review and meta-analysis

Authors: Sana A, Somda S, Meda N et al.

Reference: BMJ Open Respiratory Research 2018;5:e000246

URL: <http://dx.doi.org/10.1136/bmjresp-2017-000246>

Comments: The global prevalence of COPD is growing in women more so than in men. One-thirds of the world population use biomass fuel for cooking and heating. Individual epidemiological studies have shown a variation of findings with regards to the relationship between biomass smoke exposure and COPD prevalence in women. Therefore, this study has performed a systemic review and meta-analysis to further investigate the relationship between COPD in women and biomass smoke exposure. A total of 24 studies were included. Of these studies, 5 were case-control studies and 19 were cross-sectional studies. This study found that biomass-exposed individuals were 1.38 times more likely to be diagnosed with COPD than non-exposed. Furthermore, there was a significant association between COPD and biomass smoke exposure in woman living rurally as well as in the urban environment. This study strengths current evidence for the association between biomass smoke and COPD prevalence in women. Future studies should investigate the potential health benefits of mixed/alternative fuel use for cooking/heating on respiratory health, specifically the reduction of COPD.

Air Pollution and Mortality in the Medicare Population

Authors: Di Q, Wang Y, Zanobetti A et al.

Reference: New England Journal of Medicine, 2017; 376:2513-2522

URL: <https://www.nejm.org/doi/full/10.1056/NEJMoa1702747>

Comments: Thresholds for safe ambient air pollutant levels in the outdoor environment are well-established in health care policy, with goals for air quality to ensure levels are below these thresholds. However, previous evidence suggests that there may not actually be a 'safe' threshold level, and that the dose-response of health effects of air pollutant exposure is linear. This cohort study of Medicare beneficiaries in the US further adds to this evidence. Survival was determined for 60,925,443 persons in the US (from years 2000 to 2012, with 460,310,521 person-years of follow-up), and compared to particulate matter and

ozone levels measured as annual averages at their registered location of residence (by postcode). Increases in particulate matter levels (measured as PM_{2.5}) and ozone levels were associated with statistically significant increases in mortality. These associations were also found, at similar effect size, in air pollutant levels that were below the published national standards. Certain populations within the cohort with lower socioeconomic status had higher risk overall. These data suggest that even exposure to lower levels of air pollution can have health effects. Consequently further efforts to reduce outdoor air pollution need to be enacted.

The Lancet Commission on pollution and health

Authors: Landrigan P, Fuller R, Acosta N et al

Reference: The Lancet, 2018, Volume 391, Issue 10119, 462 - 512

URL: [https://doi.org/10.1016/S0140-6736\(17\)32345-0](https://doi.org/10.1016/S0140-6736(17)32345-0)

Comments: This Lancet Commission on pollution and health, published in 2018, summarises key issues in a range of pollutants that impact on human health and disease, with air pollution being one of the prominent areas highlighted. Key messages from this Lancet Commission include the disproportionate impact of pollution as an environmental cause of morbidity and mortality in low and middle-income countries. The bulk of air pollution arises from combustion in traffic, industry, heating and cooking, with fossil fuel combustion contributing to outdoor air pollution, and biomass combustion contributing to indoor air pollution in many countries. The series of comprehensive reviews within this Commission advocate for effective pollution control in all settings, which would lead to improved outcomes for pollution-related health conditions, better quality of life for people, enhanced economic outcomes and overall better planetary health.

Damp housing, gas stoves, and the burden of childhood asthma in Australia

Authors: Knibbs L, Wildeuohannes S, Marks G et al

Reference: Medical Journal of Australia, 2018; 208 (7): 299-302.

URL: [doi: 10.5694/mja17.00469](https://doi.org/10.5694/mja17.00469)

Comments: Triggers in the indoor environment are potential risk factors for development of asthma in children. This modelling study of Australian children aged 14 years or

younger estimated dampness (visible mould in the households, outside of the bathroom) and gas stove exposure (presence of gas stoves in households). Based on associations from meta-analyses, it was estimated that the population attributable fraction for childhood asthma from damp housing was ~8%, and ~12% for exposure to gas stoves. This study suggests that in the Australian setting, there is a substantial burden from damp housing and gas stoves to the development of childhood asthma. Ameliorating indoor environmental exposures – including improving indoor housing conditions to reduce dampness, and increasing ventilation of gas stove emissions – could help to reduce the burden of asthma in children.

Chronic electronic cigarette exposure in mice induces features of COPD in a nicotine-dependent manner

Authors: Garcia-Arcos I, Geraghty P, Baumlin N et al.

Reference: Thorax. 2016. 71: 1119-1129

URL: <http://dx.doi.org/10.1136/thoraxjnl-2015-208039>

Comments: E-cigarettes are electronic devices that can be filled with liquid that can contain a concentration of nicotine or flavouring in propylene glycol (PG) or vegetable glycerine (VG). This liquid is heated which produces a vapour that is then inhaled into the lungs. Potential implications for prolonged exposure to e-cigarette vapour may include impaired immune defence, increase in inflammation and oxidative stress as well as airway hyperactivity. Due to the high rate of smokers that will relapse despite nicotine replacement therapy (93% within 6 months of quitting), it becomes evident that smoking cessation is difficult. Chronic obstructive pulmonary disease (COPD) is a disease associated with smoke exposure, and is predicted to become the 3rd leading cause of death worldwide, creating an enormous health care burden. Therefore, some health care advocates argue that e-cigarettes are a safer alternative for individuals who cannot quit traditional smoking methods. This study used in vitro and in vivo models to evaluate the biological impact e-cigarette usage had on mouse lungs and normal human bronchial epithelial cells (NHBE) from a series of human subjects. 12 week old A/J mice were whole body exposed for 4 months (5 days/ week) to 0.4mL PBS or e-cigarette vehicle (50:50 PG & VG) containing 0 or 18mg/mL nicotine. NHBE cells isolated from 15 non-smoking donors were differentiated at ALI with cells exposed to 36 puffs of vaporised e-liquid with 0 or 36 mg/mL of nicotine using a VC-1 exposure system, with a 70mL puff volume applied every 30 seconds. This

study found that inhaled nicotine in e-cigarettes triggered effects associated with COPD pathogenesis and progression by causing airspace enlargement, mucous cell hypertrophy, release of inflammatory mediators and altered epithelial ciliary function. Furthermore, this study provided direct evidence for the harmful effects e-cigarettes. In particular, nicotine was a key driver of the pathogenic changes observed in the lung. Future research is needed to compare the pulmonary consequences of inhaled nicotine versus nicotine absorbed through products such as nicotine patches.

Electronic cigarette use and indoor air quality in a natural setting

Authors: Soule EK, Maloney SF, Spindle TR et al.

Reference: Tobacco Control, 2017; 26: 109-112

URL: <http://dx.doi.org/10.1136/tobaccocontrol-2015-052772>

Comments: Second hand smoke produced by tobacco cigarettes has been linked to causing a number of diseases such as COPD and cancers. A number of countries and cities worldwide have enacted laws that restrict cigarette smoking from public areas including hospitals, restaurants and bars to prevent non-smoking individuals risk of negative health consequences from second hand smoke. E-cigarettes have gained popularity in recent years, with these devices being capable of producing aerosols that contain toxicants that include nicotine and volatile organic compounds. Previous studies have found that e-cigarette aerosols influence indoor air quality and currently a number of countries globally have varied restrictions on e-cigarette use in indoor areas. This study aimed to investigate e-cigarettes effects on indoor air quality in a natural setting. PM_{2.5} was measured at a two-day e-cigarette event in which e-cigarettes use was permitted, in a hotel meeting room, an indoor restaurant within the event hotel and an indoor restaurant adjacent to the hotel but not hosting an e-cigarette event. Measurements were collected in the event room the day before the event, during the two days of the event with two time points on each day for at least 30 minutes, and the day after the event. This study showed that the median PM_{2.5} concentration in the event room increased from a range of 1.92-3.20µg/m³ to 311.68-818.88µg/m³. Therefore, PM_{2.5} was 125-330 times higher in a room where e-cigarette use was active compared to a similar room with no e-cigarette usage. Overall this study concluded that e-cigarettes have the potential to generate high concentrations of PM_{2.5}, which has the potential to cause second hand exposure harm to non e-cigarette users. Policy makers should take into consideration that e-cigarettes users should be required to comply with existing clean indoor air regulations like cigarette smokers.



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Compiled by Dr Christel Norman, Respirology Editorial Office, Perth, Australia

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