CONCLUSIONS

In light of this literature review, there is evidence of an association between smoke exposure from wildfires and small increases in mortality. Evidence from one study suggests that bushfire smoke is also associated with out-of-hospital cardiac arrests. Furthermore there is evidence from several studies of an association between wildfire smoke and increased morbidity, namely hospitalisations, ED visits and outpatient visits.

It is not possible to determine from just one good quality Australian study evaluating both mortality and morbidity whether increased mortality attributable to environmental smoke events could ever occur in the absence of an observed increase in morbidity. Nevertheless, this one study found increased morbidity without detectable increased mortality. So we think it unlikely that increased mortality could be observed without a detectable increase in morbidity.

TABLES

Table 1. Summary of studies investigating the association between vegetation fire smoke events and mortality/morbidity worldwide.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Morgan et al., 2010 (25)	New South Wales fires	1 January 1994 -20 June 2002	Sydney	PM_{10}; bushfire days identified as days with city-wide 24 hour average PM_{10} concentrations greater than the 99^{th} percentile for the study period	Mortality (all-cause, respiratory and cardiovascular) and hospital admissions (respiratory and cardiovascular)	Time series analysis adjusting for temperature, humidity, day of week and influenza epidemic	Positive but not significant association between bushfire PM_{10} and all-cause mortality (%change: 0.80%; 95%CI: -0.24%, 1.86%, lag 0). No association with CVD or respiratory mortality. A 10 $\mu g/m^3$ increase in bushfire PM_{10} was associated with increase in hospital admissions for all respiratory diseases: 1.24% (95%CI: 0.22%, 2.27%, lag 0). Bushfire PM_{10} also associated with increased hospital admissions due to COPD (>65 yrs, lags 0-3), pneumonia (>65 yrs, lag 1) and asthma (15-64 yrs, lag 0).
Aditama, 2000 (16)	Indonesia forest fires, 1997	September 1997-June 1998	Indonesia	–	Mortality in pulmonary ward of one hospital and inpatient and outpatient counts in health offices and hospitals	Comparing cases between September 1997 and June 1998 with the same period in 1995-1996. No confounding factors included in analysis. No statistical significance testing.	Increased mortality rate 2 to 4 times that of the previous months in the pulmonary ward of Jambi hospital (no numerical results shown). Increase in cases of acute respiratory infection by 80% in South Kalimantan and 51% increase in respiratory diseases in Health Office Jambi.

Table 1 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Emmanuel, 2000 (18)	Indonesia forest fires, 1997	August-November 1997	Singapore	PM_{10}	Mortality, ED presentations, hospital admissions and outpatient visits for haze-related conditions	Time series analysis adjusting for temperature, relative humidity, rainfall and wind speed	No increase in mortality or hospital admissions (no numerical results presented). An increase in 100 $\mu g/m^3$ PM_{10} was significantly associated with a 12% increase in outpatient visits for upper respiratory tract illness, 19% for asthma and 26% for rhinitis. There were also increases in ED attendances for haze related conditions (no risks presented).
Awang et al., 2000 (17)	Indonesia forest fires, 1997	September 1997	Malaysia	–	Mortality and number of hospital cases of asthma and acute respiratory infections	Comparing September figures to June figures. No confounding factors included in analysis. No statistical significance testing.	No increase in mortality (no numerical data provided). Increased hospital cases of asthma and acute respiratory infections.

Table 2. Summary of studies investigating the association between vegetation fire smoke events and mortality worldwide.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Zu et al., 2015(34)	Quebec, Canada	July 2002	Boston and New York City, USA	$PM_{2.5}$ measured	Total mortality (natural causes), cardiovascular and respiratory mortality	Time series adjusting for temperature, week of the month, weekend and holiday; regression models performed for the year 2002 (fires) and compared with 2001 and 2003	Substantial short-term increases in $PM_{2.5}$ concentrations from forest fire smoke were not associated with increases in daily mortality in Greater Boston or New York City.
Faustini et al., 2015(33)	Southern Europe (Spain, France, Italy, Greece, Bulgaria)	2003-2010	Southern Europe (Madrid, Barcelona, Marseille, Turin, Milan, Bologna, Parma, Modena, Reggio Emilia, Rome, Palermo, Athens and Thessaloniki)	PM_{10} measured; forest fire events identified from satellite images; smoky days defined when smoke concentrations > 8 $\mu g/m^3$ estimated from satellite	Mortality (natural causes, cardiovascular and respiratory)	Poisson regression models simulating a stratified case-crossover approach adjusting for temperature, time trends, seasonality, population decreases during summer and holidays, influenza epidemics and Saharan dust advection	PM_{10} (per 10 $\mu g/m^3$) was associated with an increase in natural mortality (0.49%, 95%CI 0.14, 0.85), cardiovascular mortality (0.65%, (95%CI 0.10, 1.19) and respiratory mortality (2.13%, 95%CI 0.85, 3.42) on smoke-free days; PM_{10}-related mortality was higher on smoky days with a suggestion of effect modification for cardiovascular mortality (3.42%, 95%CI 0.64, 6.28, p value for effect modification 0.055), controlling for Saharan dust advections

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Sahani et al., 2014 (31)	Indonesia	2000-2007	Klang Valley region, west coast of Peninsular Malaysia	Haze days defined as those with daily average PM_{10} >100 μg/m^3	Mortality (natural causes and respiratory)	Case-crossover design with adjustments for temperature, humidity and PM_{10}; analysis stratified by age-group and gender	Haze days associated with increased natural mortality in ≤ 14 yrs (OR 1.41; 95%CI 1.01, 1.99, lag 2). Also increased all-age respiratory mortality at lag 0 (OR 1.19; 95%CI 1.02, 1.40) and among all males (OR 1.34, 95%CI 1.09, 1.64). Additional significant increased respiratory mortality of males ≥ 60 yrs (lag 0) and females 15-59 yrs (lag 5).
Shaposhnikov et al., 2014 (32)	Moscow, 1 June - 31 August 2010	2006-2010	Moscow	PM_{10} measured; temperature and PM_{10} measured to evaluate their combined effect on mortality; heatwave period 6 Jul - 18 Aug 2010; wildfires reported to have occurred during the heatwave but no specification of wildfire days or differentiation of background from wildfire PM_{10}	Mortality (non-accidental)	Time-series analysis with interaction term between PM_{10} and temperature adjusting for humidity, time trend, day of week, season and ozone	The interaction between temperature and PM_{10} was estimated to contribute to 2200 deaths during the heatwave. Relative increases in mortality per 10 μg/m^3 PM_{10} were 0.43% (95%CI: 0.09%, 0.77%) at temperature ≤18°C, 0.77% (0.40%, 1.13%) at temperature = 22°C and 1.44% (0.94%, 1.94%) at temperature = 30°C.

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Heo et al., 2014(29)	Biomass burning from Russia, Mongolia and China	2003-2007	Seoul, South Korea	$PM_{2.5}$ from biomass burning identified by positive matrix factorisation receptor model, an advanced factor analysis technique	Mortality (all non-accidental, CVD and respiratory)	Time series adjusting for temperature, humidity, day of the week, holiday and influenza epidemics	CVD mortality increased by 1.9% (95%CI: 0.0, 3.7) per IQR increase in biomass burning. No significant effects on all-cause and respiratory mortality
Linares et al., 2014 (30)	Spain	2004-2009	Madrid, Spain	$PM_{2.5}$ and PM_{10}; days with advection of particles from biomass combustion were supplied by the Spanish Ministry of Agriculture, Food and Environment	Mortality (natural, circulatory and respiratory)	Time series stratified by days with or without advection; adjustments for temperature, O_3, NO_2, season and influenza epidemics	On days with advection a 10 $\mu g/m^3$ increase in PM_{10} was associated with natural cause mortality at lag 2 (RR: 1.035, 95%CI: 1.011, 1.060); no other association of PM_{10} or $PM_{2.5}$ on days with advection. On days without advection $PM_{2.5}$ was associated with natural, circulatory and respiratory mortality.
Nunes et al., 2013 (28)	Brazilian Amazon	2005	Brazilian Amazon	$PM_{2.5}$ modeled (estimated with input from satellite observations); no defined fire days or source of $PM_{2.5}$	Mortality (CVD in elderly >64 years)	Multivariate linear regression adjusting for human development index, primary care units and ICU beds	Significant associations between CVD mortality rates and annual %hours with $PM_{2.5} > 25\ \mu g/m^3$ (β=0.01; no CI reported)

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Marlier et al., 2013 (24)	Fires in the regions of the Association of Southeast Asian Nations (ASEAN): Brunei, Cambodia, Indonesia, Lao, Malaysia, Myanmar, Philippines, Singapore, Thailand and Vietnam	1997-2006	Association of Southeast Asian Nations	$PM_{2.5}$ modeled	Mortality (cardiovascular disease)	Combined satellite-derived fire estimates, atmospheric modelling and a previously published equation (82) to estimate the risk of CVD mortality	During the 1997 Indonesian fires, there was additional exposure owing to fires of 5,240,000 person-years above the annual WHO 25 $\mu g/m^3$ interim target with estimated increase in adult CVD mortality burden by 10,800 annual deaths (95%CI 6,800-14,300)
Johnston et al., 2012 (23)	Global	1997-2006	Global	$PM_{2.5}$ estimated by combining outputs from chemical transport model and satellite observations	Mortality (all-cause)	Daily burden of mortality estimated using previously published concentration-response coefficients for the association between $PM_{2.5}$ and all-cause mortality	339,000 annual deaths attributed to wildfires (IQR 260,000-600,000); Sub-Saharan Africa (157,000 deaths) and Southeast Asia (110,000) are the most affected regions

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Analitis et al., 2012 (22)	Attica Prefecture fires, Greece	1998-2004	Athens	Size of forest fires defined by area burned: (1) small (10,000-1 million m^2 burned), (2) medium (>1 million to 30 million m^2 burned), and (3) large (>30 million m^2 burned)	Mortality (natural causes, CVD, respiratory)	Generalised additive models testing the association between forest fire size and mortality, adjusting for temperature, humidity, wind speed and direction, time trend, day of the week and holiday	Small fires not associated with mortality. Medium-size fires were associated with a 4.9% increase (95%CI: 0.3, 9.6%) and 16.2% (1.3, 33.4%) in total and respiratory mortality, respectively. The 1 large fire had the strongest health effect with a 49.7% (37.2, 63.4%), 60.6% (43.1, 80.3%), and 92.0% (47.5, 150.5%) increase in total, CVD, and respiratory mortality.
Kochi et al., 2012 (27)	California, USA	1999-2003	Counties of Los Angeles, San Diego, Riverside, Orange and San Bernardino in California	Satellite imagery defined as "smoke-affected areas" all studied counties; wildfire period defined 24 October-06 November 2003; non-wildfire in 2003 period defined as the 2 weeks prior to wildfire period	Mortality (cardio-respiratory)	Poisson regression and difference-in-difference model to estimate the mortality effects of the 2003 wildfire event using mortality data from the wildfire period and non-wildfire period from 2003, as well as the same periods from control years of 1999-2002. No adjustment for temperature or humidity.	Estimated 3.08 excess cardio-respiratory deaths daily in San Bernardino County during wildfire period

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Johnston et al., 2011 (26)	New South Wales fires	1994-2007	Sydney	PM_{10}; extreme pollution event was defined as any day when PM_{10} concentration exceeded the 99^{th} percentile of the time series (47.3 $\mu g/m^3$) and further identified as smoke or dust event	Mortality (non-accidental, CVD and respiratory)	Case-crossover analysis adjusting for temperature, humidity and influenza epidemic	A 5% increase in non-accidental mortality associated with smoke events (OR 1.05, 95%CI: 1.00, 1.10). No association with respiratory or cardiovascular mortality.
Hänninen et al., 2009 (21)	Russia	14 days (26 August - 8 September 2002)	Finland	$PM_{2.5}$ and PM_{10}	Mortality	Time series analysis including only $PM_{2.5}$ as exposure and adjusting for time trend	Positive but not significant increase in daily mortality (RR:0.8%, 95%CI: -3.5%, 5.3%) per 10 $\mu g/m^3$ increase in same-day $PM_{2.5}$
Vedal and Dutton, 2006 (20)	Denver, 2002 (2 wildfire smoke days, 9 June and 18 June 2002)	2 years (2001-2002)	Denver, USA	$PM_{2.5}$ and PM_{10}	Mortality (total non-accidental and cardio-respiratory)	Comparison of daily mortality with same month in previous year and with 2 control counties not affected by fires; descriptive analysis only with no statistical tests performed	No acute increase in mortality could be attributed to the abrupt increases in PM concentrations

Table 2 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Sastry, 2002 (19)	Indonesia forest fires, 1997	1994-1997	Kuala Lumpur and Kuching, Malaysia	PM_{10}; high-pollution days defined as those with PM_{10} > 210 $\mu g/m^3$ or by low-visibility (< 0.91 km)	Mortality (all-cause, non-traumatic, CVD, respiratory, and other)	Time series analysis adjusting for temperature, humidity and time trends; results presented as relative risk (standard error); PM_{10} measurements only in Kuala Lumpur; only 13 days high-pollution days included in 1996-1997 and 14 low-visibility days between 1994-1997	Increased non-traumatic mortality in Kuala Lumpur on high pollution days (PM_{10} > 210 $\mu g/m^3$): RR 1.697 (SE: 0.367), $p<0.05$ for 65-74 yr age-group and in CVD mortality on low-visibility days for 65-74 yr age-group (RR 2.016, SE 0.257, $p<0.01$). Increased respiratory mortality in Kuching on low-visibility days for all ages (RR 2.049, SE 0.650, $p<0.05$).

Table 3. Summary of studies investigating the association between vegetation fire smoke events and morbidity in Victoria.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Dennekamp et al., 2015 (76)	Alpine region, Victoria, Australia	July 2006 - June 2007 (Fire season 01/11/2006 to 31/03/2007)	Melbourne	$PM_{2.5}$, PM_{10} and CO; fire-hours in Melbourne were identified as those when the hourly $PM_{2.5}$ >50 µg/m^3 and the hourly CO >50 ppm and the back trajectories for air masses at 1000m elevation were in the sector where the forest fires were occurring.	Out-of-hospital cardiac arrest (OHCA)	Case-crossover adjusted for temperature and humidity	Greater increases in OHCA in men were observed with IQR increases in 48-hour lagged $PM_{2.5}$, (8.05%; 95%CI: 2.30, 14.13%; IQR=6.1 µg/m^3), PM_{10} (11.1%; 1.55, 21.48%; 13.7 µg/m^3) and CO (35.7%; 8.98, 68.92%; 0.3 ppm). No significant association between OHCA and air pollutants among women. During 174 'fire-hours' (i.e. hours in which Melbourne's air quality was affected by forest fire smoke) during 12 days of the 2006/2007 fire season, 23.9 (3.1, 40.2) excess OHCA were estimated due to elevations in $PM_{2.5}$.
Tham et al., 2009 (54)	Victoria, Australia	7 months (October 2002-April 2003)	Melbourne and Gippsland region	PM_{10} monitored	ED presentations and hospital admissions for respiratory disease	Time series analysis adjusting for temperature, humidity and day of the week	9.1 µg/m^3 increase in PM_{10} associated with a 1.8% (95%CI: 0.4, 3.3%) increase in respiratory ED presentations in Melbourne. No association with hospital admission after adjustment for confounders.

Table 4. Summary of studies investigating the association between vegetation fire smoke events and morbidity in Australian states other than Victoria.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
New South Wales							
Johnston et al., 2014 (63)	New South Wales, Australia	1 July 1996 - 30 June 2007	Sydney	Smoke days defined as a day with either mean daily $PM_{2.5}$ > 27 $\mu g/m^3$ or PM_{10} > 47 $\mu g/m^3$	ED visits (non-trauma, all respiratory, asthma, COPD, pneumonia, bronchitis, all cardiovascular, ischaemic heart disease, arrhythmias, cardiac failure (CF) and cerebrovascular diseases	Case-crossover analysis adjusting for temperature, humidity, influenza epidemics and public holidays	Smoke days were associated with same day increases in ED attendances for all non-trauma conditions (OR: 1.03, 95%CI: 1.02, 1.04), respiratory conditions (1.07, 1.04, 1.10), asthma (1.23, 1.15, 1.30), and COPD (1.12, 1.02,1.24). Ischaemic heart disease ED visits were increased at a lag 2 (1.07, 1.01, 1.15) while arrhythmias had an inverse association at a lag 2 (0.91, 0.83, 0.99); increase in CF attendances were present for the 15–65 year age group at lag 2 (1.37 1.05, 1.78).
Martin et al., 2013 (61)	New South Wales bushfires, mostly in the Blue Mountains, Australia	1994-2007	Sydney, Newcastle and Wollongong	Smoke event days defined as: days with daily city-wide average $PM_{2.5}$ and PM_{10} exceeding the 99^{th} percentile of the daily distribution for the study	Hospital admissions: all non-trauma, cardiovascular, asthma, COPD, and other respiratory admissions	Time-stratified case-crossover design adjusting for temperature, humidity, influenza epidemics and public holidays	In Sydney, smoke events were associated with a same day increase in all non-trauma hospital admissions (OR: 1.02, 95%CI: 1.00, 1.03), respiratory admissions (1.06, 1.02, 1.09), COPD (1.13, 1.05, 1.22) and asthma admissions (1.12,1.05, 1.19). In the other cities with smaller populations, associations with all respiratory

				period. Smoke event days were compared with non-smoke event days.			admissions were more variable and tended to be greatest on the day after the smoke event; although associations tended to be positive, they were less consistent and lacked precision. No significant associations with CVD health outcomes in any city.
Smith et al., 1996 (39)	New South Wales, Australia	January 1994	Western Sydney	PM_{10}	ED presentations for asthma at seven hospitals	Comparisons between case and control periods and time series analysis not adjusted for temperature and humidity	No association between asthma presentations and PM_{10} from bushfire smoke.
Cooper et al., 1994 (37)	New South Wales, Australia	January 1994	Sydney	PM_{10}	ED presentations for asthma at three inner-city hospitals	No details given	No increase in asthma presentations compared with before the event.
Kolbe and Gilchrist, 2009 (52)	New South Wales and Victoria, Australia	38-day period in January-February 2003	Albury, New South Wales	PM_{10} monitored	Seeking medical attention because of the smoke	Telephone survey	5% reported seeking medical attention because of the smoke
Queensland							
Chen et al., 2006 (78)	Brisbane, Australia	3.5 years (1 July 1997-31 December 2000)	Brisbane	PM_{10} measured	Hospital admissions for respiratory diseases	Time series analysis adjusted for average temperature, day of the week, seasonality, long-term trends (years) and influenza	Comparing hospital admission on high PM_{10} days (>20 $\mu g/m^3$) to low PM_{10} days (<15 $\mu g/m^3$) showed a 19% (95%CI: 9, 30%) increase in respiratory hospital admissions on bushfire days and 13% (6, 23%) on background days.

Table 4 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Northern Territory							
Crabbe 2012 (59)	Darwin, Australia	1993-1998	Darwin	PM_{10}	Hospital admissions for respiratory and cardiovascular diseases (CVD).	Time series analysis adjusted for temperature, humidity, season, time trend, day of the week, holidays, influenza (1995 only)	Significant effect in respiratory admissions restricted to 1995 controlling for influenza epidemics (RR: 1.025, 95%CI: 1.000, 1.051, lag 1). No effect on CVD admissions.
Hanigan et al., 2008 (50)	Darwin, Australia	Fire seasons (April - November) in each year from 1996 to 2005	Darwin	PM_{10} estimated using a predictive model based on visibility data	Hospital admissions for cardiovascular and respiratory diseases	Time series analysis adjusted for temperature, humidity, day of the week holidays, influenza epidemics, indigenous status, ICD change	Increase of same-day 10 $\mu g/m^3$ PM_{10} associated with 4.81% (95%CI: -1.04%, 11.0%) increase in respiratory admissions. Significant Association for Indigenous Australians (9.4%, 1.04, 18.5%). No significant effect on CVD.
Johnston et al., 2007 (48)	Darwin, Australia	Fire seasons (April - November) in 2000, 2004 and 2005	Darwin	PM_{10} measured	Hospital admission for respiratory and cardiovascular diseases (CVD)	Case-crossover analysis adjusted for weekly influenza rate, days with rainfall >5 mm, temperature and humidity for same day and previous 3 days and public holidays	An increase of 10 $\mu g/m^3$ in PM_{10} associated with hospital admissions for all respiratory conditions (OR: 1.08, 95%CI: 0.98, 1.18) and significantly with COPD admissions (1.21, 1.00, 1.47). For Indigenous Australians larger effect sizes, in particular for COPD (1.98, 1.10, 3.59). Significant association between PM_{10} and CVD admissions only for Indigenous Australians (1.71, 1.14, 2.55; ischaemic heart disease, lag 3).

Table 4 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Johnston et al., 2002 (42)	Northern Territory, Australia	April - October 2000	Darwin	PM_{10}	ED presentations for asthma	Time series analysis adjusting for weekly influenza and weekday versus weekend	Increased asthma presentations with 10 $\mu g/m^3$ increase in PM_{10} (Rate ratio: 1.20, 95%CI: 1.09, 1.34).

Table 5. Summary of studies investigating the association between vegetation fire smoke events and morbidity in Southeast Asia and Europe.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Southeast Asia							
Mott et al., 2005 (44)	Indonesia	January 1995 - December 1998	Kuching, Malaysia	No pollutant measurements included in analysis	Hospital admission to seven hospitals for respiratory disease	Time series analysis comparing 1997 forest fires period with forecasted estimates using pre-fire periods, adjusting for monthly seasonal components	Increase in respiratory hospitalisations during the bushfire period, particularly due to asthma.
Brauer and Hisham-Hashim, 1998 (40)	Indonesia	August-September 1997	Kuala Lumpur, Malaysia	–	Hospital admissions to major hospitals for asthma and respiratory infection	Comparing September admissions to August. No adjustment for temperature / humidity. No statistical significance testing.	Increase in hospital admission for asthma and acute respiratory infection.
Chew et al., 1995 (38)	Indonesia	September-October 1995	Singapore	PM_{10}	ED presentations for acute asthma in children <12 years in two large hospitals in Singapore	Time series analysis adjusted for meteorological variables and "other factors" (not further specified)	Increase in daily PM_{10} associated with increase in asthma emergency presentations (no risks presented in paper).
Europe							
Ovadnevaite et al., 2006 (46)	Lithuania	August-September 2002	Vilnius City, Lithuania	–	Presentations to eight health centres for respiratory diseases in Vilnius	Comparing increase in presentations from 1 - 18 September 2002 to July 2002	Number of presentations for respiratory diseases on average over all eight health centres was 3.1 times higher in September compared to July (ranging from 1.5 times in one health centre to 20.5 times in another).

Table 6. Summary of studies investigating the association between vegetation fire smoke events and morbidity in North America.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Resnick et al., 2015 (65)	Arizona, USA	1 May - 8 July 2011	Albuquerque, New Mexico, USA	$PM_{2.5}$	ED visits for respiratory and cardiovascular diseases (CVD)	Poisson regression comparing cases during acute exposure period and post-acute exposure period with pre-wildfire (reference period)	During acute exposure period mean (range) $PM_{2.5}$ was 31.3 (10.0-70.0) µg/m^3. There was increase in ED visits due to asthma in 65+ age group (risk rate 1.73, 95%CI 1.03-2.93), due to CVD in all ages (1.08, 1.00-1.16), due to diseases of the pulmonary circulation (2.64, 1.42-4.90) and cerebrovascular disease (1.69, 1.03-2.77) in 20-64 age group. Increased ED visits due to diseases of the circulatory system (1.56, 1.00-2.43) in 65+ age group.
Tse et al., 2015 (66)	Southern California, USA	2003 and 2007	Southern California	Postal codes used to define children who were “closer to the fires” or “farther away”	ED visits and/or hospitalisation for asthma exacerbation	Comparison between the frequency of children with the outcome before and after the fires in 2003 and 2007; analyses stratified by BMI	No significant change in ED visits or hospitalisations before and after the fires.

Table 6 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Yao et al., 2014 (64)	British Columbia, Canada	Forest fire season (1 April to 30 September) of 2003 to 2010	British Columbia	$PM_{2.5}$ measured and modeled	Outpatient physician visits for asthma, upper and lower respiratory infections, otitis media and cardiovascular diseases (CVD)	Time series analysis adjusting for temperature, day of week, month and year	A 10 µg/m^3 increase in modelled $PM_{2.5}$ was associated with increased physician visits for asthma [rate ratio: 1.06, 95%CI: 1.04, 1.08], COPD (1.02, 1.00, 1.03), lower respiratory infections (1.03, 1.00, 1.05), and otitis media (1.05, 1.03, 1.07). Similar effect sizes with measured $PM_{2.5}$. Effects of $PM_{2.5}$ were small for CVD visits, and significantly protective for measured $PM_{2.5}$ on all fire season days. Modeled $PM_{2.5}$ had a marginal effect on CVD visits on the most extreme fire days (1.01; 1.00, 1.02).
Yao et al., 2013 (62)	British Columbia, Canada	Forest fire season (24 July - 29 August) 2010	British Columbia	$PM_{2.5}$ measured and forecasted	Outpatient physician visits for asthma	Time series analysis adjusting for temperature, day of week, holidays, week of study	30 µg/m^3 increase in measured $PM_{2.5}$ associated with asthma related physician visits (rate ratio: 1.10, 95%CI: 1.00-1.21)
Dohrenwend et al., 2013 (60)	Southern California, USA	1 October - 6 November 2007	San Diego county, USA	Air quality index (AQI), comprised 4 pollutants (PM, 0_3, CO and SO_2). An AQI of 100	Respiratory ED visits at a single community ED in San Diego	Frequency of ED visits compared pre- and during fire period; AQI index provided for different cities in San Diego county during the fire period	AQI >100 in >50% of cities for 4 consecutive days during the fire period associated with average number of visits for asthma during fire period (increased by 2.6 visits per day, p=0.04).

				corresponds to 150 μg/m^3 of PM_{10} (24 hour average). Fire period 21 October - 06 November 2007.			
Rappold et al., 2012 (58)	Pocosin Lakes National Wildlife Refuge, North Carolina, USA	6 weeks (1 June - 14 July 2008)	40 mostly rural counties in North Carolina	$PM_{2.5}$ estimates based on smoke dispersion simulation	ED visits for congestive heart failure (CHF) in > 44 years old and asthma in >18 years old	Time series analysis stratified by the top and bottom 50% counties in each of 6 criteria measuring community health. No adjustment for temperature or humidity.	100 μg/m^3 increase in $PM_{2.5}$ was associated with 66% increase in asthma ED visits (95%CI: 28%, 117%) in lag 0, and 42% increase in CHF ED visits (5%, 93%) in lag 1. Difference in risk between bottom and top ranked counties by Socio-Economic Factors was 85% and 124% for asthma and CHF respectively.
Henderson et al., 2011 (56)	British Columbia, Canada	92 days (1 July - 30 September 2003)	Southeastern corner of the province of British Columbia, Canada	PM_{10} measured	Physician visits and hospital admissions for respiratory and cardiovascular diseases (CVD)	Population-based cohort study. Logistic regression with repeated measures was used to estimate the independent fixed effects of a 30 μg/m^3 increase in PM_{10} on health outcomes. Adjustments for temperature, day of week and week.	A 30 μg/m^3 increase in PM_{10} was associated with respiratory physician visits (OR: 1.05, (95%CI: 1.03, 1.06), with asthma-specific visits (1.16, 1.09, 1.23) and respiratory hospital admissions (1.15, 1.00, 1.29). Associations with CVD outcomes were largely null.

Table 6 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Rappold et al., 2011 (57)	Pocosin Lakes National Wildlife Refuge, North Carolina, USA	2 weeks (1-14 June 2008)	Eastern North Carolina counties	Plume (defined as satellite measured AOD ≥ 1.25). Exposed counties had a minimum of 25% of the geographic area covered with plume on at least 2 days.	ED visits for CVD and respiratory diseases	Time series without adjustments. Results reported as cumulative relative risk over lag days 0-5 after exposure.	Exposed counties ED visits for respiratory diseases increased 66% (95%CI: 1.38, 1.99), for asthma 65% (1.25, 2.17), for COPD 73% (1.06, 2.83), pneumonia and acute bronchitis 59% (1.07, 2.34), for heart failure-related condition increased 37% (1.01, 1.85). ED visits associated with cardiopulmonary symptoms were significantly increased [1.23; 1.06, 1.43)] in the exposed counties.
Schranz et al., 2010 (55)	San Diego, USA	12 days (14-19 and 21-26 October 2007)	Emergency Departments of University of California, San Diego hospitals in San Diego	$PM_{2.5}$	ED visits and hospitalisations (all causes)	Comparison of the number of patients presenting to the ED during the first 6 days of the firestorm with the 6-day period prior to the start of fires.	5.8% decline in mean ED visits (from 154.8 to 145.8). Hospitalisations higher during the fire period (19.8% vs. 15.2%, p = 0.01). Number of patients presenting with shortness of breath increased (6.5% vs. 4.2%; p = 0.03) and due to smoke exposure (1.1% vs. 0%, p = 0.001).

Table 6 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Delfino et al., 2009 (51)	Southern California	1.5 months (1 October - 15 November 2003)	Southern California	$PM_{2.5}$ (spatially resolved particle mass data)	Hospital admissions (respiratory and cardiovascular)	Time series adjusting for temperature, humidity, trends, seasonality, fungal spores, weekend, and surface pressure gradient. Analyses stratified per wildfire period and pre-wildfire period. Estimates calculated for the 2-day moving average of $PM_{2.5}$.	10 $\mu g/m^3$ increase in $PM_{2.5}$ associated with greater effect on the wildfire period on hospital admissions due to asthma for age ≥ 65 yrs (10.1% increase, 95%CI: 3.0%, 17.8%) and age 0-4 years: (8.3% increase, 2.2%, 14.9%). No significant effect on CVD admission during or after wildfire period.
Lee et al., 2009 (53)	Northern California, USA	12 weeks (17 August - 4 November 1999),	Hoopa Valley Indian Reservation, California	PM_{10} monitored	Visits to the Medical Clinic in Hoopa Reservation for respiratory diseases, CVD, diabetes and headache	Multivariate logistic regression to study the association between clinic visits during the fires and PM_{10} levels, controlling for age, residence, gender and clinic visits in 1998	PM_{10} associated with increased risk of clinic visit due to: asthma (OR: 1.77, 95%CI: 1.51, 2.09), coronary artery disease (1.48, 1.11, 1.97) and headaches (1.74, 1.32, 2.29). Total number of clinic visits increased by 15% during fire period.
CDC, 2008 (49)	San Diego, USA	22-26 October 2007	San Diego	–	ED visits for respiratory diseases to 6 hospitals in San Diego County	Comparison of ED visits during the fire period (22-26 October 2007) with 20 weekdays during 24 September-October 19, 2007. No adjustments for temperature humidity.	Mean number of visits per day increased for respiratory syndrome (117.8 to 148, $p<0.01$), asthma (21.7 to 40.4, $p<0.01$) and dyspnea (16.3 to 23.6, $p<0.01$).

Table 6 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Kunzli et al., 2006 (45)	Southern California, USA	October - November 2003	Southern California	PM_{10} and retrospective reporting on smell of fire indoors	Physician visits during the 2-week fire period for respiratory, pharyngeal and eye symptoms	Mixed effects models adjusted for gender, ethnicity, educational levels of the parents, asthma status	Children who reported smell of fire smoke indoors for 1-5 days had OR 1.33 (95%CI 1.02, 1.74) for visiting a doctor; those with ≥ 6 days had an OR 2.03 (1.53, 2.71).
Moore et al., 2006 (77)	British Columbia, Canada	1993-2003	Regions of Kelowna and Kamloops in British Columbia	PM_{10} and $PM_{2.5}$	Physician visits due to respiratory disease	Comparing three-week forest fire period with aggregated rates of same weeks in 10 previous years. No adjustment for temperature and humidity.	Significant increase in weekly physician visits during the fire period compared to same weeks in previous year for Kelowna. No significant effects were found for Kamloops (where PM concentrations were lower).
Viswanathan et al., 2006 (47)	California, USA	October-November 2003	San Diego County	PM_{10}	ED presentations for respiratory diseases to 15 hospitals in San Diego County	Comparing 2 weeks during and following the fire to 1 week before the fire. No adjustment for temperature/humidity. No statistical significance testing.	Increase in ED presentations during the fire period for asthma, respiratory problems (without fever) and smoke inhalation.
Mott et al., 2002 (43)	Northern California, USA	14 August - 4 November 1999	Hoopa Valley Indian Reservation, California, USA	PM_{10} measured	Clinic visits for respiratory problems	Frequency of clinic visits during fire period in 1999 compared with same period in 1998; correlation between weekly PM_{10} levels and weekly number of visits	Clinic visits for respiratory problems increased 52% during fire period compared with same period in 1998. PM_{10} correlated with counts of clinic visits in the same period in 1999 (r=0.74) and 1998 (r=-0.63)

Table 6 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Sorensen et al., 1999 (41)	Florida, USA	June-July 1998	Volusia and Flagler counties, Florida	–	ED presentations and hospital admissions at eight hospitals for respiratory conditions	Comparing bushfire period (1 June - 6 July 1998) to the same period the year before. No adjustment for temperature/humidity.	ED presentations during the fire period increased 91% for asthma (from 77 to 147), 132% for bronchitis with acute exacerbation (from 28 to 65), 37% for chest pain (from 218 to 299). Minimal changes in number of hospital admissions.
Shusterman et al., 1993 (36)	California, USA	1 week (20-26 October 1991)	San Francisco Bay area, California	–	ED presentations to 9 hospitals with complaint (respiratory, ocular or headache) caused or exacerbated by the fire	Description of diagnosis during ED presentation. No statistical significance testing.	Most frequent diagnosis of patients seen in ER was bronchospastic reaction to smoke.
Duclos et al., 1990 (35)	California, USA	August 1987	California	Total suspended particles and PM_{10}	ED presentations to 15 hospitals in 6 Californian counties due to respiratory disease, coronary disease, otitis, conjunctivitis, headache or panic reactions	Compared 2.5 weeks of bushfire smoke to a control period. No adjustment for temperature/humidity	Increase in ED presentations during the fire period for asthma, COPD, laryngitis, sinusitis and other respiratory infections.

Table 7. Summary of studies investigating the association between vegetation fire smoke events and morbidity in South America.

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Sugar cane field fires							
Arbex et al., 2014 (68)	Araraquara, Brazil (sugarcane field fires)	1 Feb 2005 to 31 Jul 2007	Araraquara, Brazil	Total suspended particles (TSP)	ED visits for pneumonia	Time series analysis adjusting for temperature, humidity and day of week. Analysis performed for (1) whole study period, (2) burning and (3) non-burning periods.	ED visits for pneumonia increased 29.4% (95%CI 14.1-50.9) in the 2 days following IQR (46.1 μg/m^3) elevation in TSP (whole study period). The magnitude of effect during the burning periods was similar to that during the entire period. During the non-burning periods effect was non-significant.
Arbex et al., 2010 (69)	Araraquara, Brazil (sugarcane field fires)	23 Mar 2003 to 27 Jul 2004	Araraquara, Brazil	Total suspended particles (TSP)	ED visits for hypertension	Time series analysis adjusting for temperature, humidity, day of week comparing sugarcane harvest and non-harvest periods.	10 μg/m^3 increase in the TSP 3-day moving average lagged in 1 day associated with hypertension-related hospital admissions during harvest (12.5% increase, 95%CI: 5.6, 19.9%) and non-harvest (9.0%, 4.0, 14.3%).
Arbex et al., 2007 (67)	Araraquara, Brazil (sugarcane field fires)	23 March 2003 to 27 July 2004	Araraquara	Total suspended particles (TSP)	Hospital admissions for asthma	Time series analysis adjusting for temperature, humidity, day of the week, stratified by sugarcane burning and non-burning periods.	10 μg/m^3 increase in the 5-day moving average (lag1–5) of TSP concentration associated with increase of 11.6% (95%CI 5.4 to 17.7) in asthma hospital admissions. In non-burning periods: 9.7% (2.6 to 17.2); in burning periods: 12.7% (2.2 to 24.3).

Table 7 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Cançado et al., 2006 (70)	Sugarcane field fires in Piracicaba, Brazil	April 1997 – March 1998	Piracicaba	$PM_{2.5}$ and PM_{10} measured	Hospital admissions due to respiratory diseases	Time series adjusting for temperature, humidity and season. Analyses performed for the whole period and stratified per burning and non-burning periods.	10.2 μg/m^3 (IQR) in $PM_{2.5}$ and 42.9 μg/m^3 (IQR) in PM_{10} associated with increases of 21.4% (95%CI: 4.3-38.5) and 31.03% (1.25-60.21) in child and elderly respiratory hospital admissions. Effects during burning period were higher than during non-burning period.
Forest fires in the Brazilian Amazon region							
Silva, et al., 2013 (75)	Brazilian Amazon	2005	Cuiaba, Brazil	$PM_{2.5}$ modeled; dry season between July and December 2005 (when forest fires occurred)	Hospitalisation due to respiratory diseases in children < 5 yrs and elderly ≥ 65 years	Time series analysis adjusting for temperature, humidity, time trend, weekdays, holidays. Analysis stratified by all year of 2005 or dry season; results presented for moving average $PM_{2.5}$ (MA)	In 2005 increased hospitalisation of children for 10 μg/m^3 increase in $PM_{2.5}$ (9.1%, 95%CI: 1.8%, 18.1%, MA1), and 12% (0.2, 25.5%, MA5). During the dry season the increase was 11.4% (1.7, 22.2%, MA1) and 21.6% (4.9, 41.1%, MA5). No significant associations with hospitalisations in elderly.
do Carmo et al., 2013 (74)	Brazilian Amazon	2004-2009	Porto Velho, Brazil	$PM_{2.5}$ modeled; no clearly defined fire days period or differentiation of forest fire from background $PM_{2.5}$	Hospitalisation for respiratory causes in children	Bayesian analysis; time series adjusting for temperature, humidity, weekdays and holidays	Increases of 10 μg/m^3 in $PM_{2.5}$ exposure associated with 5.6 % (95%CI: 3.64, 7.31) increase in hospital admissions due to respiratory diseases at lag 2.

Table 7 Continued

Study	*Location of fire*	*Study period*	*Study area*	*Exposure*	*Health outcome and study population*	*Analytical methodology*	*Study result*
Andrade Filho et al., 2013 (73)	Manaus, Brazilian Amazon	2002-2009	Manaus, Brazil	$PM_{2.5}$ modeled	Hospitalisations due to respiratory diseases in children < 9 years	Correlation and multiple linear regression, adjusting for humidity and rainfall	$PM_{2.5}$ correlated negatively with hospitalisations (r= -0.168, p<0.01). Average weekly and monthly number of fires not correlated with hospitalisations. Weekly mean $PM_{2.5}$ associated with weekly mean number of hospitalisations (β=-1.60, p=0.003) in regression analysis.
Ignotti et al., 2010 (72)	Brazilian Amazon	2004 and 2005		$PM_{2.5}$ modeled; % of hours/year with $PM_{2.5}$ >80 $\mu g/m^3$ as indicator of exposure [named % of annual hours (AH %)]	Hospitalisation for respiratory diseases	Time series adjusting for mean number of blood tests per 100 inhabitants (indirect indicator of health service quality) and Human Development Index. No meteorological adjustment	1% increase in annual hours of $PM_{2.5}$ >80 $\mu g/m^3$ associated with 5%, 8% and 10% increases in hospitalisations for 5-64 yrs, <5 yrs, and ≥ 65 yrs age groups, respectively
Ignotti et al., 2010 (71)	Brazilian Amazon	2005	Municipalities of Alta Floresta and Tangará da Serra, Mato Grosso, Brazil	$PM_{2.5}$ modeled	Hospitalisation for respiratory diseases in children < 5 yrs and elderly >64 yrs	Time series adjusting for temperature, relative air humidity and temporal trend and stratified by whole year and dry season (when fires occurred)	Significant associations in Alta Floresta only. Increased risks for 10 $\mu g/m^3$ increase in average daily $PM_{2.5}$. Children: RR 4.7% (95%CI: 0.6-9.1) for lag 3 and 4.2% (0.1-8.5) for lag 4 during whole year. In dry season RR 6% (1.4-10.8) and 5.1% (0.6-9.8) respectively. Elderly RR 4.3% (0.25-8.6) for lag 0 and 5.5% (0.56-10.6) for lag 4.

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Dr Diogenes Ferreira has been a research fellow at the Department of Epidemiology & Preventive Medicine since September 2014. He graduated in Medicine from the Federal University of Goiás, Brazil and completed Residency Training in Allergy and Immunology at the University of Campinas, Brazil. He received his PhD from the University of São Paulo, Brazil studying the pathology of fatal asthma. His interest in the epidemiology of asthma and allergies brought him to Monash in 2014 to perform post-doctoral research on the 20-year follow-up of European Community Respiratory Health Survey conducted in Melbourne.

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MONASH University

GROUP OF EIGHT

School of Public Health and Preventive Medicine
The Alfred Centre
[redacted]

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IN THE MATTER OF
THE HAZELWOOD MINE FIRE INQUIRY

STATEMENT OF MICHAEL ABRAMSON

I, Michael Abramson of [redacted] say as follows:

The Hazelwood Mine Fire Health Study ("the Study")

1. I am a Professor of Clinical Epidemiology and Deputy Head of the Department of Epidemiology and Preventive Medicine, School of Public Health and Preventive Medicine at Monash University ("Monash").

2. On 30 October 2014, Monash was commissioned by the Department of Health and Human Services ("DHHS") to undertake the Study.

3. I am one of two Principal Investigators for the Study.

4. The Study is a long-term health study into the potential health effects of the Hazelwood mine fire in 2014 ("Fire"). Those potential effects include cardiovascular and respiratory disease, low birth weight, psychological impacts and the development of cancer.

5. The Study has been funded through DHHS but is conducted by a team of independent researchers led by Monash University through the School of Public Health and Preventive Medicine and the School of Rural Health. A number of other research organisations are involved.

6. A Community Advisory Committee has been established to ensure that the study hears directly from and works in partnership with Latrobe Valley community members, health and community service providers and local government. Local and national experts will be called upon to contribute to our Clinical Reference Group and Scientific Reference Group during the course of the Study.

7. The general aims of the study are to provide answers to the follow questions:
    a. Is there evidence that people who were heavily exposed to smoke from the Fire are more likely to have developed heart and lung conditions or to develop them in the future, when compared with another similar community with less exposure to the Fire?
    b. Is there evidence of any impact of smoke exposure during pregnancy or infancy on the health and development of children in the Latrobe Valley compared to otherwise similar infants and children with less

exposure to the Fire?

c. Is there evidence that people who were heavily exposed to smoke from the Fire have a higher level of psychological distress than otherwise similar people with less exposure to the Fire and is this associated with particular vulnerable groups?
d. Is there evidence that people who were heavily exposed to smoke from the Fire are more likely to develop cancers over a long period of time than otherwise similar people with less exposure to the Fire?

8. The Study has been divided up into multiple research streams which will commence at different times. Initial pilot work was undertaken in 2014 in respect of a number of streams. The streams are:
    a. Community Wellbeing – mid 2015;
    b. Latrobe ELF (Early Life Followup) Study – mid 2015;
    c. Older People – May 2015;
    d. Schools Study – July 2015;
    e. Adult Survey – late 2015;
    f. Follow up health and psychological assessments – likely 2017;
    g. Linkage to health records including hospital, ambulance and cancer – 2016 onwards.

9. Different streams will cover different towns. For example, the Latrobe ELF Study will cover the entire Latrobe Valley, as will the Schools Study. The Community Wellbeing Study will cover an ever larger area.

10. However, the Adult Survey and associated risk assessments (including respiratory and cardiovascular sub-streams) will focus only on the residents of Morwell. Air pollution modelling provided to us by the CSIRO (**Attachment 1**) shows that Morwell was the town most exposed to fine particulate matter during the Fire.

11. We have requested access to the Victorian Electoral Roll to identify suitable adult participants for the Adult Survey. All adults resident in Morwell at the time of the fire will then be invited to participate. From that group, researchers will recruit a sample of people to participate in the study. It is expected it will take at least a year to recruit all the participants required. We hope to obtain 7,500 participants from Morwell.

12. The Adult Survey will use Sale as the comparison population. Sale has been selected because it is another rural community with a comparable socio-demographic profile to Morwell and a large enough population. Air pollution modelling shows there was minimal if any exposure to smoke from the Fire in Sale which makes it appropriate as a comparison town. We hope to obtain 4,000 participants from Sale.

13. The scope of the Study has been largely set but it would be possible to expand it to include other groups, for example, emergency responders to the fire such as fire fighters and police who were stationed in Morwell during the fire. Currently the Adult Survey does not cover this group unless they were also residents of Morwell during the Fire.

14. From a scientific point of view, it would be of great interest to involve this group, particularly in the respiratory and cardiac parts of the study. If they were included, it would be possible to find a comparison group by recruiting fire fighters and police who were not deployed to the Fire. If the study were to be expanded to include this group, further funding would be required.

15. In the long-term, we expect the Study will contribute to answering the question as to whether the Fire contributed to an increase in deaths in those exposed to the smoke.

16. There are a number of published studies which show an association between deaths and exposure to fine particulate matter ($PM_{2.5}$). 'Association' in this context means that the two things vary together – but not necessarily that one causes the other. For example, there are studies which show a small increase in the risk of cardiovascular deaths after $PM_{2.5}$ exposure.

17. However, none of these studies have examined the health effects of exposure as a result of an open cut brown coal mine fire – let alone one with a similar size, duration and proximity to a town to the Fire. Most have looked at urban air pollution and some at smoke from bush fires.

18. As far as we are aware, there has been no comparable fire in a brown coal mine overseas or in Australia. In this sense, the Study will be the first of its kind in terms of the data obtained regarding health effects including any association with an increase in deaths over time.

19. We have recently become aware of some unpublished research completed on the health effects from a black coal mine fire in the United States. We have requested copies of this research but not yet received it. It is unclear at this stage whether it will be directly comparable with the Fire or not.

20. One key way in which the Study is designed to provide, in the future, information about whether the Fire leads to an increase in deaths, is through the planned linking to the National Death Index. This will occur at some point in the future.

21. The National Death Index is a compilation of data from various State based registries which includes information regarding cause of death. Having access to this (along with the data we have ourselves obtained in the Adult Survey) would allow us to exclude accidental deaths, for example, from a car accident and focus on cases where chronic disease is identified as the cause of death. This would permit an examination over a longer period of specific causes of death among residents of Morwell.

22. We are developing exposure metrics to assess individual exposures to smoke from the Fire. It will then be possible to conduct an analysis to determine whether there is any association between smoke exposure and causes of death such as cardiovascular, respiratory diseases or cancer. It will also be possible to adjust for confounding factors such as sex, age, socioeconomic status,

tobacco smoking and occupational exposures.

23. Because we will be drawing from the data obtained from the Adult Survey, which is limited to Morwell residents, the Study will not provide information as to whether or not there was an increase in deaths in surrounding areas or in persons who worked in Morwell during the Fire but did not reside there.

24. The Study's current scope also will not look backwards to analyse deaths or other health impacts which have occurred during or after the Fire and the commencement of the various study streams. One reason for this is because it is not possible for us to exclude other confounders, such as cigarette smoke or adverse effects from the work environment, in the absence of a person completing the Adult Survey. In particular, we consider that the effects of cigarette smoke must be allowed for in order to detect any effects of the Fire.

25. Unfortunately, the data linkage and statistical analysis cannot take place during the timeframe of the Inquiry. This work will take a number of years to complete.

The Rapid Health Risk Assessment and the Literature Review on Mortality and Morbidity associated with Environmental Smoke Events

26. On 5 February 2015, Monash University was asked to conduct a literature review as part of an updated Rapid Health Risk Assessment. I was one of the joint authors of that review.

27. On 5 May 2015, we provided DHHS with a review titled "Updated Literature Review on Mortality and Morbidity associated with Environmental Smoke Events". A copy of the review is **Attachment 2**.

28. Mortality refers to deaths; morbidity refers to symptoms or disease including hospital admissions, emergency department and outpatient visits**.**

29. We were asked to review the literature available domestically and internationally to determine whether increased mortality could be attributed to an environmental smoke event in the absence of any observed increase in morbidity.

30. As outlined above, there is no study which deals with a comparable environmental smoke event to the Fire. The studies we reviewed dealt with the mortality and morbidity associated with wildfires (bushfires).

31. In relation to morbidity, we searched for studies which looked at hospital admissions, emergency department visits and outpatient visits to a physician.

32. We concluded that while it was not possible to definitively answer the question, in large part because of the limits of the studies we reviewed, it was unlikely that increased mortality could be observed without a detectable increase in morbidity.

33. We were not asked to undertake any statistical analysis and at that time were not provided with any data showing numbers of deaths in the Latrobe Valley during the Fire. Nor were we provided with any data showing numbers of hospital admissions, emergency department visits, outpatient visits to a physicians or visits to 'pop-up clinics' in the Latrobe Valley during the Fire.

From:	Heffernan, Emily (AU)
To:	"Philip McCloud [redacted])"
Cc:	Fox, Chris (AU)
Subject:	RE: Hazelwood Mine Fire Inquiry [KWM-Documents.FID1770821]
Attachments:	WIT.0001.001.0005.pdf

Dear Philip,

Further to our letter dated 6 October 2015, please find **attached** a report of Professor Abramson & others dated 12 March 2014 entitled "*Final Report Rapid Health Risk Assessment Report (**RHRA**)*".

Kind regards,

Emily Heffernan | Senior Associate
King & Wood Mallesons
[redacted]

"RAL-2"

MONASH University

M8 Alliance

Medicine, Nursing and Health Sciences

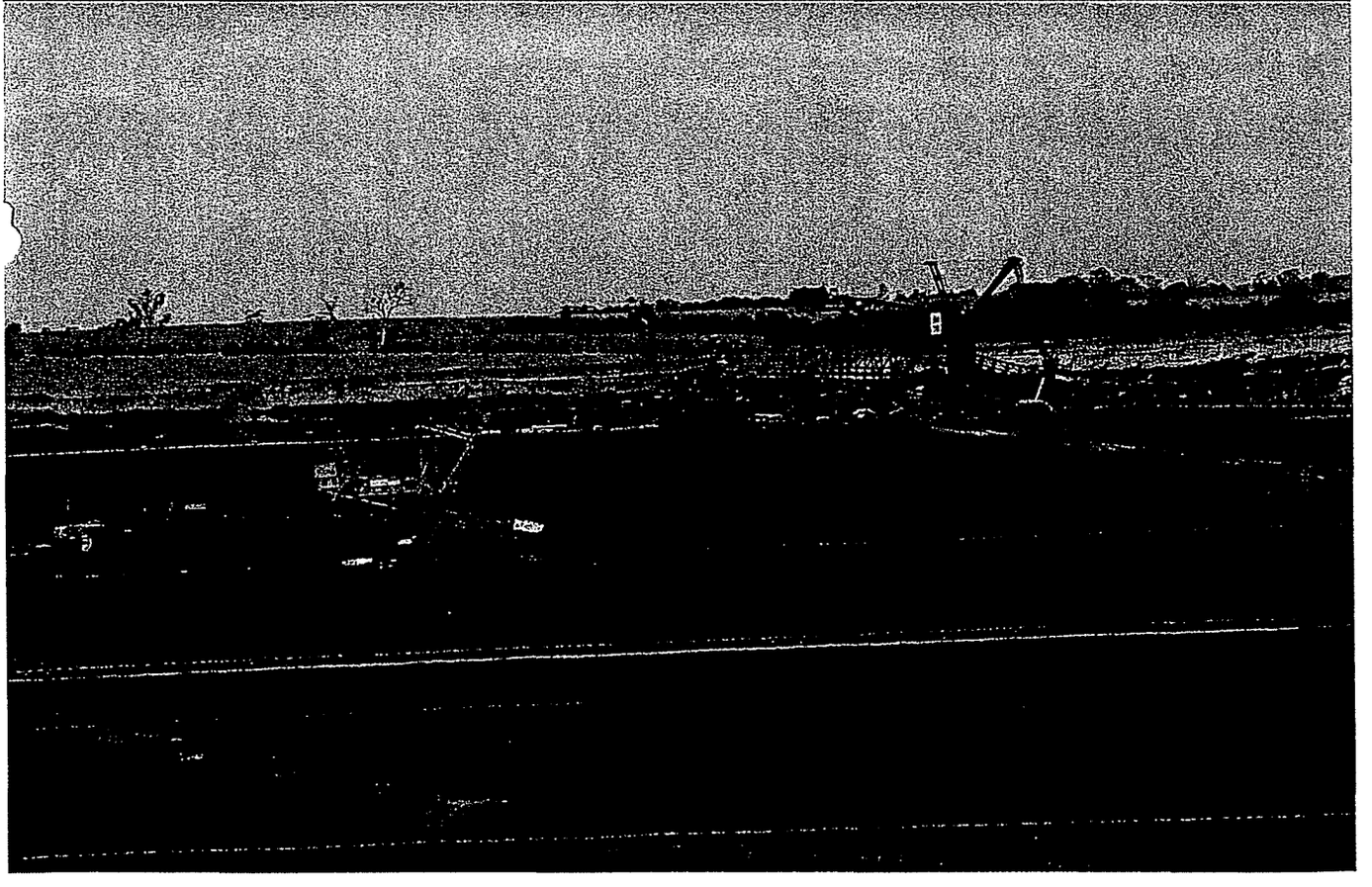
School of Public Health & Preventive Medicine

Final Report
Rapid Health Risk Assessment (RHRA)

Prepared for the Department of Health

12 March, 2014

Authors: Professor Michael Abramson, Dr Martine Dennekamp, Professor Malcolm Sim, Associate Professor Manoj Gambhir, Professor Brian Priestly, Dr Fay Johnston (Menzies Research Institute, University of Tasmania), Dr Lisa Demos, Professor John McNeil.



Contents

Glossary of Terms

Ambient air	Outdoor air
Acute Lower Respiratory Infection (ALRI)	Acute lower respiratory infections usually includes acute bronchitis and bronchiolitis, influenza and pneumonia
Carbon monoxide (CO)	A poisonous gas produced by incomplete oxidation of fossil fuels. Carbon monoxide is poisonous by virtue of its capacity to bind to haemoglobin much more easily than oxygen
Carboxy-haemoglobin (COHb)	A compound produced by the exposure of haemoglobin to carbon monoxide. Carbon monoxide is inhaled into the lungs, absorbed through the alveoli, and bound to haemoglobin in the blood, blocking the sites for oxygen transport
Case-crossover study	An epidemiological study comparing ambient conditions during a period when an individual suffered an acute health effect with a period when no such effect
Chronic obstructive pulmonary disease (COPD)	Long-standing disease of the airways of the lung associated with increased production of phlegm and shortness of breath and often caused by cigarette smoking
Cohort study	An epidemiological study involving subjects exposed to pollutant(s) suspected of being related to the development of the disease under investigation. The entire study population is followed over time
Electrocardiogram (ECG)	A recording, from electrodes placed on the chest and limbs, of electrical changes originating in the muscle of the heart
ESCAPE	European Study of Cohorts for Air Pollution Effects
Ischaemic heart disease (IHD)	Disease of the heart caused by a reduction in the blood flow to the myocardium (the muscle of the heart wall) due to narrowing of the coronary arteries

Meta-analysis	Statistical analysis that allow the results of epidemiological studies to be combined
National Environment Protection Measure (NEPM)	A special set of national objectives designed to assist in protecting or managing particular aspects of the environment. The concept of NEPMs is unique and is provided for under the National Environment Protection Acts (NEPC Acts). NEPMs can be made about a variety of environmental matters as prescribed by the NEPC Acts, including ambient air quality
Polycyclic Aromatic Hydrocarbons (PAHs)	A group of organic contaminants that form from the incomplete combustion of hydrocarbons, such as coal. Many of these can cause cancer
$PM_{2.5}$	The concentrations (expressed in $\mu g/m^3$) of particles of less than 2.5 µm in the air
PM_{10}	The concentrations (expressed in $\mu g/m^3$) of particles of less than 10 µm in the air
Sulphur dioxide (SO_2)	An acidic gas formed by oxidation of sulphur found in fossil fuel
Time-series study	A study using an epidemiological method involving the relationship between outcome (e.g. number of deaths or hospital admissions in a population) and explanatory variables (e.g. pollutant concentrations) using measures of these variables at regular (usually daily) time intervals
Volatile Organic Compounds (VOCs)	Organic chemical compounds whose composition makes it possible for them to evaporate under normal indoor atmospheric conditions of temperature and pressure
µm	Abbreviation for micrometre or micron (a unit of length). 1µm = one thousandth of a millimetre
95% Confidence interval (95%CI)	The degree of uncertainty associated with a sample statistic, i.e. 95% Cl means that there is a 95% chance that the true value lies between the two values

FINAL REPORT OF RAPID HEALTH RISK ASSESSMENT

Executive Summary

This Rapid Health Risk Assessment was commissioned by the Department of Health to help understand the potential long term health effects on the Morwell community of short to medium-term exposure to coal mine fire smoke, including particulates and gaseous pollutants.

The specific aspects of the brown coal fire smoke which are addressed in this review are:

1. identifying the hazards of most concern in the coal mine smoke;
2. characterisation of the level of risk to the Morwell community, including vulnerable subgroups;
3. indicating how the risks would change with persisting exposure up to a year
4. identifying reports of long-term health outcomes from comparable incidents;
5. Providing recommendations for other relevant air toxics which should be measured; and
6. Identifying factors increasing risk.

The main health outcomes requested to be included in the review were heart and lung conditions, cancer and birth outcomes. Health risks to mine workers, firefighters and other emergency workers were not specifically asked to be included in this review.

The fire in the Hazelwood brown coal mine fire involved exposure of a nearby township to plumes of smoke which persisted over a period. To date there are no published health studies done specifically in relation to exposure to smoke from fires in open cut brown coal mines, similar to that at Morwell. As there was little published literature on the health effects of medium term high exposures from coal fire smoke on long term health outcomes, this review extrapolates and predicts effects on health by using what is known about health effects from urban air pollution and from biomass smoke.

The studies of urban air pollution used in the assessment of PM2.5 risks typically involved exposure to other air pollutants as well as particles. Despite statistical approaches that attempt to isolate the impact of PM2.5 alone, these adjustments are often limited and this potential confounding must be considered in assessing the health effects of fine particle exposure.

The principal risks to the health of the Morwell community from brown coal fire smoke arise from exposure to fine particulates ($PM_{2.5}$) and carbon monoxide (CO). There does not appear to be any significant risk from sulphur dioxide (SO_2). The potential risks from other air toxic hazards are currently unknown but are unlikely to substantially alter the conclusions in this Report. Available air quality data have been derived from two stationary monitoring sites in Morwell East and Morwell South. It has not been possible to allow for variability in personal exposures in assessing risks to health in this review.

Regarding possible causes of death from the inhalation of coal fire smoke, a meta-analysis of landmark cohort studies of urban air pollution has estimated an excess of risk of 6.2% (95%CI 4.1 – 8.4%) per 10 $\mu g/m^3$ of $PM_{2.5}$ for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality. In addition, four studies have found associations between forest fire smoke and all-cause mortality. Most large cohort studies have not found a significant increase in the risk of all cause mortality associated with CO alone.

Regarding health outcomes not resulting in death, the European Study of Cohorts for Air Pollution Effects (ESCAPE) recently found that a 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$. The most convincing evidence for adverse cardiovascular effects is from short term CO exposures that result in blood carboxy-haemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with heart disease.

Regarding cancer, the International Agency for Research on Cancer recently concluded that particulate air pollution was a Class 1 human carcinogen. The ESCAPE lung cancer meta-analysis found a pooled hazard ratio for all lung cancers of 1·18 (0·96–1·46) per 5 $\mu g/m^3$ $PM_{2.5}$. The review also found that indoor smoke from black coal fires is a risk factor for lung cancer, particularly among non-smoking women in China.

Regarding birth outcomes, the ESCAPE meta-analysis of mother-child cohort studies found that a 5 $\mu g/m^3$ increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (OR 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit. Two studies have found small but significant declines in birth weight for babies antenatally exposed during wildfires. There is also some evidence that antenatal CO exposure can be associated with low birthweight and preterm birth.

Based on these findings about the types of health outcomes related to air pollutants, epidemiological modelling undertaken as part of this review found that for combined $PM_{2.5}$ exposures around 250 $\mu g/m^3$ in Morwell South and for exposures around the National Environment Protection Measure (NEPM) in the rest of Morwell, no additional deaths would be expected even if the exposure continues for 6 weeks. However, if this level of exposure had persisted for 3 months this level of $PM_{2.5}$ might be expected to result in some additional deaths from IHD (0.5 additional deaths), Stroke (0.2), COPD (0.1), Lung Cancer (0.1) and Acute Lower Respiratory Infection (ALRI) (0.2).

The most vulnerable subpopulations in relation to health risks from exposure to $PM_{2.5}$ include children (< 5 years old), the elderly (> 65 years old), those with chronic heart and lung diseases such as Ischaemic Heart Disease (IHD), asthma and Chronic Obstructive Pulmonary Disease (COPD), and pregnant women. In addition to these well documented vulnerable groups, other factors may also increase the risk of health effects. These would include lower socioeconomic status. In addition the confounding effects of other known risk factors for these chronic diseases need to be considered. These would include cigarette smoking, obesity, hypertension, high cholesterol, diabetes, physical inactivity, occupational exposures etc.

The review has found that other relevant air toxics, apart from $PM_{2.5}$ and CO, should be measured. Measurements are currently being made of Polycyclic Aromatic Hydrocarbons (PAHs), dioxins and furans, formaldehyde and other carbonyls, chemical composition of particles, Volatile Organic Compounds (VOCs) and gaseous mercury. The results of these measurements were not available at the time of this report. It is not considered that any other air toxics need to be measured.

1. Background

This Rapid Health Risk Assessment (RHRA) was commissioned to assist the Department of Health to understand the potential long term health effects on the community of short to medium-term exposure (intermittent over weeks to months) to coal mine fire smoke, including particulates and gaseous pollutants.

The specific aspects of the risks to human health relating to the Morwell coal fire which the Department of Health has specifically asked to be addressed in this review are:

- *Characterisation of the level of risk to Morwell community posed by brown coal fire smoke:*
  - identifying the hazards and incorporating variable exposures experienced by differing community members
  - explicit reference to subpopulations with differing vulnerabilities
- *How does the risk change with persisting exposure; i.e. for periods of 3 weeks, 6 weeks, 3 months, 6 months, 9 months, or 1 year.*
- *Reports of long-term health outcomes from comparable incidents or circumstances*
- *Health outcomes of relevance:*
  - *Mortality*
  - *Morbidity*
  - *Cancer*
  - *Birth outcomes*
- *Recommendations for other relevant air toxics which should be measured*
- *Factors increasing risk*

The fire in the Hazelwood brown coal mine fire is unprecedented in that a township only a few hundred metres from the mine that has caught fire, exposing inhabitants to sometimes intense plumes of smoke over a period of several weeks. Unfortunately, to date there appear to be no published health studies done specifically in relation to exposure to smoke from fires in open cut brown coal mines, similar to that at Morwell.

The other unique aspect is that the exposure to the coal mine smoke has resulted in intermittent and on occasion high concentrations of pollutants, lasting for several weeks and which may persist for some months. As there is little published literature on the health effects of medium term high exposures on long term health outcomes, this review extrapolates and predicts effects on health by using what is known about health effects from urban air pollution and from biomass smoke.

In summary, without the availability of established scientific evidence related to health outcomes for this precise situation, this review relies on estimates of the scope of potential health effects based on knowledge and judgements related to what is known from similar air pollution research.

Of the criteria pollutants measured by the EPA, particles < 2.5 μm diameter ($PM_{2.5}$) and carbon monoxide (CO) are the ones that have most significantly increased. These have exceeded current National Environment Protection Measures (NEPM) for ambient air quality as a result of the smoke from the brown coal fire. Although other pollutants that may be elevated will be discussed, this report will mainly focus on the health risks from $PM_{2.5}$ and CO.

The focus of this review was requested to be the health risks on the Morwell community, rather than health risks related to the mine workers, firefighters and other emergency personnel.

2. Particulate Matter

2.1 Long term health effects of $PM_{2.5}$ urban air pollution

There is a well established association between short term $PM_{2.5}$ exposure and acute effects on cardiovascular and respiratory health. However there is now also evidence of long term $PM_{2.5}$ exposure and effects on the respiratory and cardiovascular systems. These studies show an increase in mortality and exacerbations of existing conditions. To date, the only association that has been found with the development of a disease has been with respiratory illness in children.

There are quite a few landmark studies on long term effects of urban air pollution on health. These include the:

- Harvard Six Cities Study(1-3) - a cohort study following 8111 adults in six US cities (1976 - 2009)
- American Cancer Society Study(4-6) - a cohort study of 552,800 adults from 51 US cities (1982 - 1998)
- Women's Health Initiative Observational Study(7) - cohort of 65,000 postmenopausal women from 36 US cities (1994-1998)
- Netherlands Cohort Study(8) - cohort of 120,000 men and women from 204 municipalities (1997 - 1996)
- Nurses' Health study(9) - cohort of 66,000 women from the US (1992-2002)
- Canadian National Cohort(10) - 2.1 million Canadians (1991-2001)
- European Study of Cohorts for Air Pollution Effects (ESCAPE) - several European Cohort Studies - results published recently(11)

Table 1 summarises the populations, follow-up periods, mean $PM_{2.5}$ concentrations proportional changes in risk of all-cause mortality and cardiovascular mortality associated with a 10$\mu g/m^3$ increase in $PM_{2.5}$.

The long term health effects in the above studies focus almost exclusively on mortality. Although the effect estimates in the above studies varies considerably, there is a clear association between $PM_{2.5}$ and all-cause mortality and mortality of respiratory and cardiovascular causes. The estimated cardiovascular effects tend to be stronger than the respiratory effect estimates. A review looking at several landmark studies on mortality found a pooled effect estimate of 6% for all-cause mortality expressed as excess risk per 10 $\mu g/m^3$ increase in $PM_{2.5}$ exposure(12). The landmark studies relate to long-term exposures to ambient air concentrations, that are generally much longer in duration that the elevated exposures for Morwell residents. There is likely to be a real difficulty in extrapolating the results of the above studies to the shorter-term exposures associated with this RHRA.

It is important to note, that very recently the International Agency for Research on Cancer (IARC) has classified ambient particulate air pollution as a known human carcinogen (Class 1)(13). Thus long term exposures to $PM_{2.5}$ are also likely to result in additional cases of lung cancer.

Table 1. Cohort studies on particulate matter <2.5μm($PM_{2.5}$) and mortality from all causes and cardiovascular diseases, adapted from Hoek et al (12)

Study	Population	Follow-up period	Mean Concentration $PM_{2.5}$ ($\mu g/m^3$)	% change in risk (95%CI) in all-cause mortality associated with a 10$\mu g/m^3$ increase PM	% change in risk (95%CI) in CV mortality associated with a 10$\mu g/m^3$ increase PM
Harvard six cities (1)	8111 adults (6 US cities)	1976 - 1989	18 (range 11–30)	13 (4, 23)	18 (6, 32)
Harvard six cities (2)	8096 adults (6 US cities)	1979 - 1998	15 (range 10–22)	16 (7, 26)	28 (13,44)
Harvard six cities (3)	8096 adults (6 US cities)	1974 - 2009	16 (range 11–24)	14 (7, 22)	26 (14, 40)
American Cancer Society (ACS) study (4)	552,800 adults (51 US cities)	1982 - 1989	18 (range 9–34)	26 (8, 47)	
ACS study (5)	500,000 adults (51 US cities)	1982 - 1998	18 (SD: 4)	6 (2, 11)	9 (3, 16)
ACS sub-cohort study (6)	22,905 subjects (LA area)	1982 - 2000	Range ~9 – 27	17 (5, 30)	26 (1, 60)
Women's Health Initiative Observational Study (7)	65,893 postmenopausal women (36 US metropolitan areas)	1994 - 1998	14 (range 3–28)		76 (25,147)
Netherlands Cohort Study (8)	120, 852 subjects (204 municipalities)	1987 - 1996	28 (range 23–37)	6 (–3, 16)	4 (–10, 21)
Nurses' Health Study (9)	66,250 women (US north eastern metropolitan areas)	1992 - 2002	14 (range 6–28)	26 (2, 54)	-
Medicare national cohort (14)	13.2 million US elderly Medicare recipients	2000 - 2005	13 (SD 4)	4 (3, 6)	-
California teachers study (15)	45,000 females	2002 - 2007	18 (7–39)	6 (–4, 16)	19 (5, 36)
Health professionals follow-up study (16)	17,545 highly educated US men	1989 - 2003	18 (SD 3)	–14 (–28,2)	3 (–17, 26)
Vancouver cohort (17)	452,735 residents 45–85 yr	1999 - 2002	4 (0 – 10)		7 (-14, 32)
US trucking industry cohort (18)	53,814 men (4 trucking companies)	1985 - 2000	14.1 (SD 4.0)	10 (2, 18)	5 (–7, 19)
Canadian national cohort (10)	2.1 million Canadians > 25 yr	1991 - 2001	9 (2 – 19)	10 (5, 15)	15 (7, 24)

Mortality

For the studies in table 1 above, pooled effect estimates expressed as percent excess risk per 10 $\mu g/m^3$ $PM_{2.5}$ was 6.2% (95%CI: 4.1 – 8.4%) for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality.(12) A meta-analysis of 33 time-series and case-crossover studies conducted in China showed that each 10 $\mu g/m^3$ increase in $PM_{2.5}$ was associated with a 0.38% (95%CI: 0.31, 0.45) increase in total mortality, 0.51% (95%CI: 0.30, 0.73) in respiratory mortality and 0.44% (95%CI: 0.33, 0.54) in cardiovascular mortality.(19)

A prospective cohort of 2.1 million adults from the 1991 Canadian census mortality follow-up study was exposed to relatively low mean (±SD) $PM_{2.5}$ exposure levels of 8.7 ± 3.9 $\mu g/m^3$.(20) In fully adjusted models a 10-$\mu g/m^3$ elevation in $PM_{2.5}$ exposure was associated with an increase in risk of diabetes-related mortality (HR 1.49; 95%CI 1.37-1.62). The change in risk to the population persisted at $PM_{2.5}$ concentrations <5 $\mu g/m^3$.

Even within concentration ranges well below the present European annual mean limit value of 25 $\mu g/m^3$, long-term exposure to fine particulate air pollution was associated with natural-cause mortality in the 367,251 subjects from the ESCAPE study.(11) A significantly increased hazard ratio (HR) was found for $PM_{2.5}$ of 1.07 (95%CI 1.02-1.13) per 5 $\mu g/m^3$. This remained significantly raised even when only participants exposed to pollutant concentrations lower than the European annual mean limit (HR 1.06, 95%CI 1.00-1.12) or below 20 $\mu g/m^3$ (1.07, 95%CI 1.01-1.13) were included.

Cardiovascular and Cerebrovascular Events

The cardiovascular effects of short- and long-term exposure to $PM_{2.5}$ have been comprehensively reviewed for the American Heart Association.(21) The scientific statement concluded that exposure to $PM_{2.5}$ over a few hours to weeks could trigger cardiovascular disease–related mortality and nonfatal events. Longer-term exposure (eg. a few years) increased the risk of cardiovascular mortality to an even greater extent than exposures over a few days. It reduced life expectancy within more highly exposed segments of the population by several months to a few years. Reductions in $PM_{2.5}$ levels were associated with decreases in cardiovascular mortality within a time frame as short as a few years.

The ESCAPE Study recruited 100,166 people (Finland, Sweden, Denmark, Germany, Italy) free from cardiovascular events and followed them for an average of 11.5 years from 1997 – 2007.(22) A 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (HR 1.13, 95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$ (1.18, 1.01 to 1.39 for a 5 $\mu g/m^3$ increase in $PM_{2.5}$).

The Multi-Ethnic Study of Atherosclerosis (MESA) reported that 2.5 $\mu g/m^3$ higher levels of residential $PM_{2.5}$ during the follow-up period were associated with 5.0 µm/year (95%CI 2.6 to 7.4 µm/year) greater intima-medial thickness (IMT) progression among persons in the same metropolitan area as well as slowed IMT progression with reductions in $PM_{2.5}$.(23)

In the Women's Health Initiative an increase of 10$\mu g/m^3$ in $PM_{2.5}$ concentration was associated with a 24% increase in the risk of a cardiovascular event (HR 1.24; 95%CI 1.09 to 1.41), 76% increase in the risk of death from cardiovascular disease (HR 1.76; 95%CI, 1.25 to 2.47) and an increased risk of

a cerebrovascular event (HR 1.35; 95%CI 1.08 to 1.68).(7) However not all studies have shown an increase in cerebrovascular events.

Lung Disease

The review by Hoek and co-workers concluded that the long-term exposure to $PM_{2.5}$ was more clearly associated with mortality from cardiovascular disease (particularly ischaemic heart disease) than from non-malignant respiratory diseases (pooled estimate 3%, 95% CI –6, 13%).(12)

The ESCAPE meta-analysis for Chronic Obstructive Pulmonary Disease (COPD) combined data from the ECRHS, NSHD, SALIA and SAPALDIA cohorts(24). There were 6550 and 3692 subjects with NO_2 and $PM_{2.5}$ data respectively. Exposure assessment was conducted with land use regression models and back-extrapolation. Surrogate measures were traffic intensity on the nearest road and load on roads within 100m of residence. COPD prevalence and incidence were defined both by the Global initiative for Obstructive Lung Disease (GOLD) criteria and the lower limit of normal. Logistic regression models adjusted for age, sex, height, BMI, education and smoking. Meta-analysis was performed with random effects models. There were weak but nonsignificant positive associations between NO_2, $PM_{2.5}$ and the prevalence / incidence of COPD. However significant overall associations were found with COPD and traffic intensity in females and never-smokers.

The ESCAPE lung cancer meta-analysis included 312,944 subjects from 17 cohort studies in 9 European countries and had a mean follow-up of 12.8 years. The hazard ratio (HR) for $PM_{2.5}$ for all lung cancers was 1·18 (0·96–1·46) per 5 $\mu g/m^3$ and 1·55 (1·05–2·29) for adenocarcinomas of the lung. (25) An analysis of birth cohort studies from ESCAPE which included Germany, Sweden, the Netherlands, and the United Kingdom measured lung function at 6–8 years of age (n = 5,921).(26) Annual average exposure to air pollution at the birth address and current address was estimated by land-use regression models. Associations of lung function with estimated air pollution levels and traffic indicators were estimated for each cohort. Changes in forced expiratory volume in 1 sec (FEV_1) were –1.77% (95% CI: –3.34, –0.18%) for a 5-$\mu g/m^3$ increase in $PM_{2.5}$.

Birthweight

Pooled data from 14 population-based mother-child cohort studies in 12 European countries (ESCAPE) included 74,178 women who had singleton deliveries between 1994 and 2011. The endpoint was low birth-weight at term defined as weight <2500 g at birth after 37 weeks of gestation.(27) A 5 $\mu g/m^3$ increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (adjusted odds ratio [OR] 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit of 25 $\mu g/m^3$ (OR for 5 $\mu g/m^3$ increase in participants exposed to concentrations of less than 20 $\mu g/m^3$ 1·41, 95%CI 1·20-1·65). The population attributable risk estimated for a reduction in $PM_{2.5}$ concentration to 10 $\mu g/m^3$ during pregnancy corresponded to a decrease of 22% (95% CI 8-33%) in cases of low birthweight at term.

Australian Studies

The findings of these international cohort and time series studies have been replicated in Australia. Studies investigating the effects of $PM_{2.5}$ on hospital admissions found strong associations in

Melbourne, Sydney and Brisbane for admissions for respiratory and cardiovascular disease, asthma (especially in children <14 years) and those with COPD.(28) The strongest associations found in these studies were in the elderly and children. A study conducted in four Australian cities (Brisbane, Melbourne, Perth and Sydney), found significant associations between particles and all-cause mortality. Meta-analyses carried out for three cities yielded estimates for the increase in the daily total number of deaths of 0.9% (-0.7% to 2.5%) for a 10 $\mu g/m^3$ increase in $PM_{2.5}$ concentration(29).

2.2 Long term health effects of biomass smoke

The studies reporting on the health effects of biomass smoke may be more relevant to the current situation than those which examined urban particulate pollution. Unfortunately there are very few studies to date that have investigated the health effects of bushfire, peat or sugar cane smoke on long term health. This is in part because it is very difficult to measure long term health effects from short to medium term exposures.

There is an established association between short term exposure to bushfire smoke and effects on respiratory health – mainly measured by Emergency Department (ED) presentations and hospital admissions. Only a limited number of studies have looked at health outcomes other than emergency presentations and admissions. There is a suggestion of effects on cardiovascular presentations and admissions. However we have recently found that during bushfire smoke in Melbourne, there was a significant increase in the number of out-of-hospital cardiac arrests attended by ambulance personnel(30). These do not show up in hospital data as most arrests are fatal and are not recorded in the hospital system. Although the studies have only looked at short term health effects, this does not mean that the effects on the individual are short term as well. For example if someone has a cardiac arrest and survives with brain damage, the effects will be long lasting for that individual.

Susceptible populations

Studies specific to severe episodes of pollution due to forest and peat fires are relatively few in number and only a small proportion have studies individuals by risk factors such as age, and underlying health status(31-38). The findings from these studies are consistent with those of the wider air pollution literature i.e. that those most susceptible to forest fire smoke are the elderly and those with pre-existing heart or lung disease.

Biomass smoke and mortality

Only fairly recently have studies been adequately powered and able to investigate associations between forest fire smoke and mortality. In particular four studies, two from Sydney(31, 39), one each from Athens(40) and Malaysia(32) have found associations between forest fire smoke and mortality. Larger studies are needed to determine which causes of mortality are most affected(41). However in a regional Australian town seasonally affected by biomass smoke from woodheaters, an intervention that reduced winter $PM_{2.5}$ by 40% was associated with a reduction in winter mortality especially for cardiorespiratory causes of death(42).

Biomass smoke and birth outcomes

There is emerging evidence concerning perinatal exposure to episodes of smoke pollution and adverse outcomes in the neonatal period and infancy. Holstius et al(43) and Breton et al(44) both found small but significant declines in birth weight for babies antenatally exposed during the 2003 southern California wildfires. Jayachandran(45) studied the impact of a severe episode of forest fire smoke pollution that persisted for several months in Indonesia. She found that exposure to wildfire smoke, especially in the three months prior to birth, was associated with reduced foetal and infant survival. A very recent, as yet unpublished, study of Rhesus monkeys(46) demonstrated reduced cytokine production at the age of three in monkeys who had been exposed to smoke from Californian wildfires soon after they were born, compared with monkeys of a similar age who were not exposed. This could suggest that early life exposures might have immunological effects that persist into adolescence. However this requires confirmation in human studies.

Cancer and biomass smoke

There are no studies that have investigated the relationship between bushfire smoke and cancer. However there is one study(47) that found that sugar cane cutters exhibited increased frequency of cytological abnormalities in blood lymphocytes (micronuclei/1000 cells) compared to a control group, possibly due to exposure to emissions derived from sugar cane burning. On the other hand indoor smoke from black coal fires is a well recognised risk factor for lung cancer, particularly among non-smoking women in China. A recent meta-analysis of 25 case-control studies found that household coal use was associated with an overall 2.15 (95%CI 1.61, 2.89) fold increased risk of lung cancer(48). There was variation in effects across regions, with the highest risk found in southern China and Taiwan. This variation is thought to be due to other constituents of black coal. The elevated risk persisted when the studies were stratified by gender, smoking, sample size, design and language of publication.

3. Carbon Monoxide

There are two relevant international guidelines for carbon monoxide (CO): one for outdoor(49) and one for indoor air(50). There are many studies on the health effects of carbon monoxide poisoning and short term low dose exposures.(51) Continuous low level CO exposure for up to 8 days has been reported to result in ECG changes in p waves in healthy subjects.(52) Table 2 summarises longer term cohort studies, the populations, durations of followup and mortality per 1 ppm increase in CO concentrations.

Table 2. Cohort studies of Carbon Monoxide Exposure and Mortality

Population	Follow-up period	Mortality per 1 ppm increase CO concentration (95% CI)	Reference
552,138 US adults (151 metropolitan areas)	1982-1998	All cause RR= 0.97 (0.93, 1.0) Cardiopulmonary RR= 0.95 (0.88, 0.99) Lung cancer RR= 0.90 (0.83, 0.96)	(5)
28,402 US postmenopausal women 50 -79 years (36 metropolitan areas)	1994–2003	Cardiovascular HR =0.92 (0.71, 1.21) using single-pollutant model HR= 0.93 (0.67, 1.30) using multi-pollutant model	(7)
70,000 US male military veterans (mean 51 years) with hypertension	1976-2001	All cause RR= 1.032 (0.954, 1.117) using single-pollutant model RR=1.023 (95% CI: 0.939, 1.115) after adjustment for NO2 & O3	(53-55)

RR= relative risk, HR= Hazards ratio

Mortality

In a reanalysis of data from Pope and co-workers of 552,138 US adults (5) with more extensive treatment of co-variates the relative risk for death from all causes was estimated to be approximately 0.98 (95%CI: 0.92, 1.03) for an increase in ambient carbon monoxide concentration from 0.19 to 3.95 ppm.(56)

Several multi-city studies have found significant associations between increasing short-term average ambient air CO concentration and increasing mortality risk when CO is considered in single-pollutant models, with the associations attenuated when models were adjusted for other co-pollutants.(57) In a time-series study of mortality in 82 US cities during the period 1987– 1994, a 1 ppm increase in ambient air carbon monoxide concentration (lag 1 day) was associated with increased all-cause mortality of 0.46% (95%CI: 0.18, 0.73), but this was not significant after adjustment for air concentration of PM_{10} alone or PM_{10} and NO_2.(58) A time-series analysis of mortality in 19 European cities participating in the APHEA-2 (Air Pollution and Health: A European Approach) project during 1990–1997 found a significant association between CO and total non-accidental and cardiovascular mortality in single-pollutant models.(59) The estimated effect size for total mortality based on a single-pollutant model ranged from 0.59% increase (95%CI: 0.41, 1.79) to 1.20% (95%CI: 0.63, 1.77) per 1 mg/m^3 increase in CO concentration (0–1-day lag). The effect on cardiovascular mortality ranged from 0.8% increase (95%CI: 0.53, 1.07) to 1.25% (95%CI: 0.30, 2.21).

Overall, the epidemiologic evidence is consistent with a causal relationship between short-term exposure to environmentally relevant CO concentrations and all cause mortality.(60) The associations were strongest for people with existing cardiovascular disease and the elderly (>65 years).

Cardiovascular effects

Cardiovascular effects of inhalation exposures to CO have been evaluated in controlled human clinical trials, epidemiological studies, and various animal studies.(57) In general, these studies provide convincing evidence for adverse cardiovascular effects in association with short term CO exposures that result in blood carboxyhaemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with coronary artery disease. Mean ambient air carbon monoxide concentrations reported in studies that have found CO-associated adverse cardiovascular outcomes ranged from 0.5 to 10 ppm, with maximum values ranging from 2 to 50 ppm.(57) These values correspond to approximate steady-state blood COHb levels of <2% for the mean and <10% for the maximum.

Ambient daily CO levels have been associated with increases in daily mortality and hospital admissions for cardiovascular diseases.(61) Groups within the population considered to be most susceptible to the effects of CO are people with cardiovascular disease, in particular the elderly with ischaemic heart disease.(28, 61) Studies of hospital admissions and emergency department visits for ischemic heart disease (IHD) and congestive heart failure (CHF) provide the strongest evidence of ambient CO being associated with adverse CVD outcomes.(28)

Single Australian city studies have found consistent associations with CO for hospital admissions and emergency department attendances for cardiovascular outcomes(62, 63). The strongest effects were found for admissions for cardiovascular disease in the elderly (>65 years) and all age groups, admissions for ischaemic heart disease and admissions for asthma in the 0–14 year age group.

Lung Disease

Relatively few controlled clinical trials have evaluated adverse respiratory effects of CO exposure. (57) Clinical trials have been conducted in very small numbers of healthy subjects under acute exposure conditions. There is a lack of strong evidence for associations between ambient air carbon monoxide concentrations at <30 ppm and pulmonary function from epidemiological studies.

Studies of children have also yielded mixed results. However one study of 263 Western Australian children demonstrated significant associations between CO (8 hour) and symptoms such as wheeze/rattle and runny/blocked nose (lag 5 and additive exposure over 5 days).(64) These associations were observed even though air pollutant concentrations were below national standards throughout the study period.

There is some evidence for associations between increasing air carbon monoxide concentrations and increasing severity of asthma.(57) In a study of 990 children aged 5–12 years from eight North American cities a 1 ppm increase in air carbon monoxide concentration (lag=0 days) was associated with an odds ratio of 1.08 (95%CI: 1.01, 1.14) for asthma symptoms.(65) However it is quite likely

that CO is serving as a surrogate for other combustion products, rather than having direct effects on the respiratory system.

Birth and Developmental Outcomes

Carbon monoxide has been associated with birth and developmental outcomes in international studies.(60) The most compelling evidence for a CO-induced effect on birth and developmental outcomes is for preterm birth (PTB) and cardiac birth defects. There is limited epidemiologic evidence that during early pregnancy (e.g., first month and first trimester) CO is associated with an increased risk of PTB. There is evidence of ambient CO exposure during pregnancy having a negative effect on foetal growth in epidemiologic studies.(66) In the Children's Health Study, a 1.4-ppm difference in first-trimester CO exposure was associated with 21.7 g lower birth weight (95%CI 1.1-42.3 g) and 20% increased risk of intrauterine growth retardation (95%CI, 1.0-1.4).(67)

A study that estimated the average exposure of five common air pollutants during pregnancy for births in metropolitan Sydney between 1998 and 2000 reported a reduction of 7 (95%CI -5.0 to 19.0) to 29 (95%CI 7.0 to 51.0) grams in birth weight for a 1ppm increase in mean carbon monoxide levels.(68) However another study which investigated the effect of prenatal exposure to six common urban air pollutants in the Sydney metropolitan area on pre-term birth between 1998 and 2000 found no clear impact of air pollutants on gestational age.(69)

Sensitive Groups

The results of epidemiological studies have found no evidence for a threshold below which adverse health effects in sensitive groups have not been observed after exposure to CO. The most sensitive groups to the effects of CO are people with existing cardiovascular disease, including ischaemic heart disease, pregnant women and the elderly.(28)

4. Morwell Air Quality Data

The fire at the coal mine started on the 9th February 2014. The data from the EPA started in Morwell East on the 17th Feb and in Morwell South on the 21st Feb. CO monitoring started on the 19th of Feb in Morwell East and 21st Feb in Morwell South. Both CO and $PM_{2.5}$ data were provided up until the morning of the 3rd of March.

Using the hourly concentrations, the average concentration to date Morwell South for $PM_{2.5}$ was 180µg/m^3 and the median 66 µg/m^3. As can be seen from Figure 1, the data are highly skewed. For Morwell East the average and median concentrations were 32 µg/m^3 and 13 µg/m^3 respectively. For CO in Morwell South the average and median concentrations were 3.3 ppm and 1.7 ppm respectively. For Morwell East the average and median concentration was 0.5 ppm and 0.1 ppm respectively. We use these observations to extrapolate effects on health (see below).

The figures below show the hourly air concentrations of the raw data as provided by the EPA.

Figure 1: Hourly average $PM_{2.5}$ concentrations in Morwell East and Morwell South

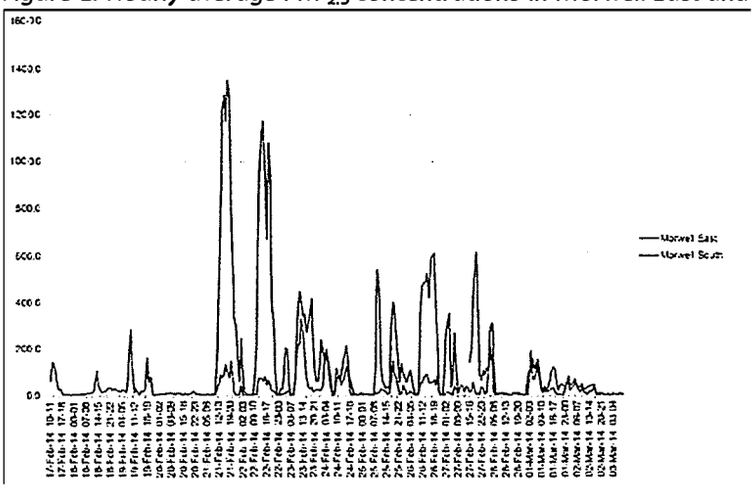
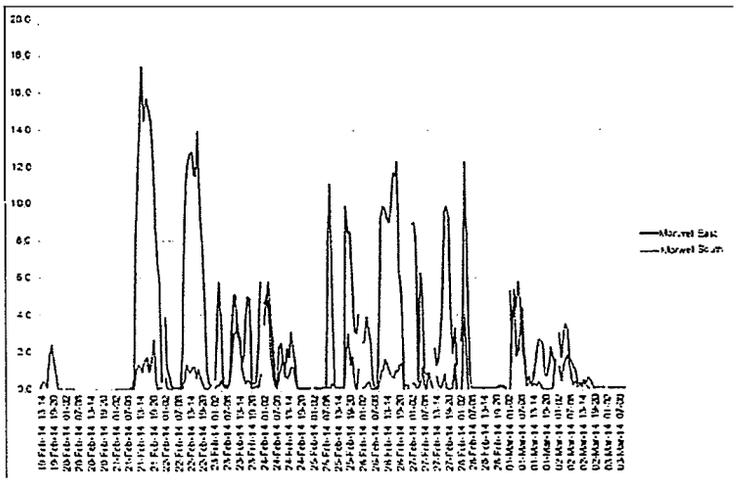


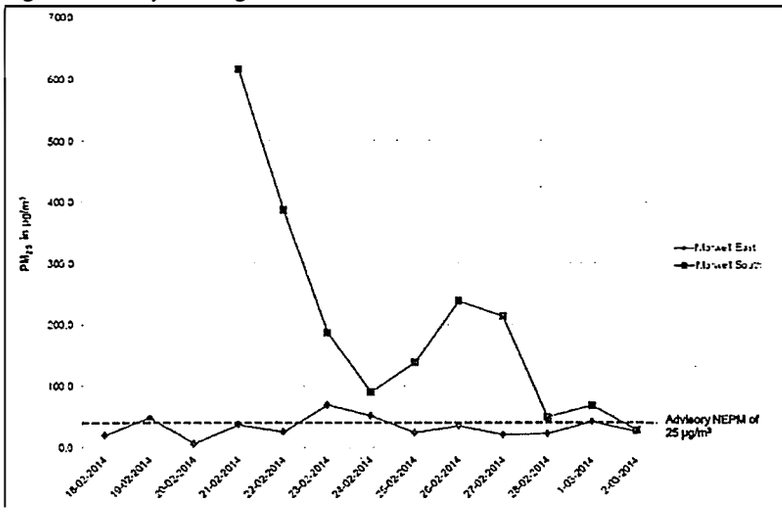
Figure 2: Hourly Average CO Concentrations (ppm) in Morwell East and Morwell South



How do the Morwell Air Quality Data compare with current standards?

The 24 hour National Environmental Protection Measure (NEPM) advisory standard for $PM_{2.5}$ is 25µg/m^3 with a maximum of 5 exceedances per year. The EPA $PM_{2.5}$ data provided for Morwell South started on the 17th of Feb, so it is quite likely exposures prior to that may have been higher. The average of the 14 days for which we have data is 33 µg/m^3 for Morwell East and for the 10 days in Morwell South the average is 202 µg/m^3. From figure 3 it is clear that exposures were very high when monitoring started. It is unfortunate that we do not know the concentrations prior to that. It is important to note, that even though there is a NEPM for $PM_{2.5}$, there is no 'safe level' below which there are no health effects expected. Numerous studies have been published where effects have been found well below the current air quality guidelines. The association between PM and effects on health has been shown to be linear.

Figure 3: Daily average PM2.5 concentrations in Morwell East and South



The 8 hour NEPM for CO is 9ppm with a maximum of 1 exceedance per year. This NEPM for CO was exceeded on 2 occasions in Morwell South for when measurements were available, i.e. the first 2 days the measurements were available, i.e. the 21st and the 22nd Feb. Therefore it is possible that the CO NEPM may have been exceeded on several days since the start of the fire on the 9th of February. Despite this the concentrations of CO are likely to have been mainly an occupational hazard rather than an environmental one. The long-term effects of exposure (at the measured levels) on health of the population of Morwell are likely to be minimal.

Sulphur dioxide (SO_2) is often a concern as a pollutant as a result from combustion. However in Victoria this is much less of an issue, mainly because brown coal has a very low sulphur content. The 1 hour NEPM for Sulphur dioxide is 200 ppb and the 24 hour NEPM 80 ppb. The sulphur dioxide measurements we have available for Morwell South (21st Feb - 7th March) confirm the low sulphur content of brown coal smoke. The hourly maximum concentration measured was 35ppb, so this even below the 24 hour NEPM. Health effects of SO_2 are unlikely to be observed at this level.

How does Morwell Air Quality compare with other cities / smoking etc?

Smoking: To put these concentrations into context, Burnett et al(70) estimated that smoking a single cigarette was equivalent to breathing a daily ambient concentration of $PM_{2.5}$ of 667 µg/m^3. The vast majority of a smoker's increase in mortality occurred at doses received before he/she was half way through the first cigarette of each day. It is also well known that cigarette smokers tend to have carboxyhaemoglobin concentrations substantially higher than non-smokers, and generally above a level considered to be the threshold for a range of adverse health effects associated with short-term exposures(51). The confounding effects of cigarette smoking would need to be factored into any analysis of carboxyhaemoglobin concentrations measured in Morwell residents during this incident.

Other Cities: *The London Smog event*: In December 1952, for 5 days London was covered by a very thick fog, due to unfavourable weather conditions trapping air pollution from homes, power plants and factories. The pollution was thought to have mainly been caused by smoke from the burning of coal. Bell and Davis(71) estimated that about 12,000 excess deaths occurred from December 1952 through February 1953 because of acute and persisting effects of the London smog. However there is an important difference between the London Smog episode and the coal mine smoke in Morwell.

Sulphur dioxide and acid aerosols are thought to have played a very important role in the death rate. And as shown above, the SO_2 in the Morwell smoke was well below the air quality standards.

As a comparison with other polluted cities in our region, the average (SD) $PM_{2.5}$ concentration in Beijing from 2007-2008 was 82 (52) $\mu g/m^3$. In addition the average concentration in Shanghai from 2007-2008 was 54 (31) $\mu g/m^3$. In Guangzuo the average concentration from 2007-2008 was 70 $\mu g/m^3$.(72) So therefore on several days in Morwell the concentration was at least as bad, or worse than the yearly average in some of the most polluted cities in China.

5. What Is The Risk And How Does Risk Change With Persisting Exposure?

Epidemiological modelling was undertaken to address this question. The population at risk has been assumed to be 14,005 which was the total population of Morwell in the 2011 census. Background mortality risks for Victoria were calculated using 2011 ABS data. Cause specific mortality rates of Ischaemic Heart Disease (IHD), Stroke, Chronic Obstructive Pulmonary Disease (COPD) and Acute Lower Respiratory Infection (ALRI) for Victoria were obtained from: http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/3303.0Chapter42011 Lung cancer rates for Victoria in 2010 were obtained from: http://www.cancervic.org.au/research/registry-statistics/cancer-in-victoria

Increases in absolute risk due to $PM_{2.5}$ exposure were calculated using the integrated risk functions recently published by Burnett et al(70). These functions provided an estimate and 95%CI for the additional number of cause-specific deaths attributable to PM_{25}. It should be noted that this study primarily related to ambient $PM_{2.5}$ exposure levels (e.g. from urban air pollution) and these tended to be considerably longer-term exposures than in the scenario we are analysing here. Therefore the additional risks attributable to $PM_{2.5}$ exposure published by Burnett et al(70) may only fully accrue when overall exposure times are longer than those we consider for Morwell. The exposure scenario is a combination of the daily average NEPM, which was the average exposure in Morwell East and the average exposure in Morwell South, where 1500 people were assumed to have been exposed. As requested, alternative durations of 3 weeks, 6 weeks, 6 months, 9 months and 1 year were modelled. The expected number of deaths for each duration of exposure and cause are summarised in Table 3. It can be seen that for this combined exposure scenario, no additional deaths would be expected even if the exposure continues for 6 weeks. However after 3 months, this level of exposure would be expected to result in some additional deaths from IHD, Stroke, COPD, Lung Cancer and ALRI.

Table 3. Modelled deaths from Ischaemic Heart Disease (IHD), Stroke, Chronic Obstructive Pulmonary Disease (COPD), Lung Cancer and Acute Lower Respiratory Infection (ALRI) in Morwell for a combined $PM_{2.5}$ exposure scenario and alternative durations of exposure

Cause of Mortality	Expected number of deaths in Morwell at Victorian rate	Duration of exposure	Additional no. deaths [95% CI]
		3 weeks	0.3 [0.2, 0.4]
		6 weeks	0.4 [0.3, 0.6]
		3 months	0.5 [0.4, 1.6]

IHD	12.9	6 months	2.4 [1.4, 4]
		9 months	2.9 [2.1, 5]
		1 year	3.7 [2.4, 6]
		3 weeks	0 [0, 0.3]
		6 weeks	0.1 [0, 0.2]
		3 months	0.2 [0, 0.4]
Stroke	3.1	6 months	0.5 [0.1, 0.8]
		9 months	0.8 [0.2, 1.2]
		1 year	1.1 [0.3, 2]
		3 weeks	0 [0, 0]
		6 weeks	0 [0, 0.1]
		3 months	0.1 [0, 0.2]
COPD	4.1	6 months	0.4 [0.1, 0.7]
		9 months	0.5 [0.3, 1.1]
		1 year	0.7 [0.3, 1.4]
		3 weeks	0 [0, 0]
		6 weeks	0.1 [0, 0.1]
		3 months	0.1 [0, 0.2]
Lung Cancer	4.8	6 months	0.6 [0.1, 1]
		9 months	0.8 [0.1, 1.5]
		1 year	1.3 [0.3, 2]
		3 weeks	0 [0, 0.1]
		6 weeks	0.1 [0.1, 0.1]
		3 months	0.2 [0.1, 0.4]
ALRI	3.6	6 months	0.6 [0.3, 1.1]
		9 months	1 [0.5, 1.5]
		1 year	1.2 [0.7, 1.9]

Other potential pollutants currently being measured:

It is difficult to estimate possible health effects of other pollutants being emitted by the brown coal fire smoke as we do not have results from the measurements currently being conducted. EPA and CSIRO have set up monitoring in Morwell to investigate these other potentially relevant pollutants, but unfortunately to date these samples have not been analysed. EPA and CSIRO are currently measuring:

- Polycyclic Aromatic Hydrocarbons (PAH), both particle and gas phase
- Particle and gas phase dioxins and furans, dioxin-like PCBs, BFRs
- Formaldehyde and other carbonyls
- Chemical composition of particles: soluble ions, elemental analysis (including metals), organic and elemental carbon
- Volatile Organic Compounds (VOC), including benzene, toluene, ethylbenzene, xylenes and 10 other common VOC
- Gaseous Elemental Mercury

Comment: Some of the above airborne pollutants (e.g. VOCs, formaldehyde and other carbonyls) are more likely to be associated with acute health impacts, such as mucous membrane irritation (eye and respiratory tract), while others (PAHs, dioxins, mercury) are more likely to be associated with longer-term or delayed health impacts, such as cancer, adverse reproductive outcomes and neurotoxicity. It is difficult to speculate on the potential for these health outcomes for the residents of Morwell in the absence of the monitoring data, and it may also be difficult to differentiate exposures to some of these pollutants from sources other than the coal fires (vehicle exhaust and other industrial or background sources).

In the case of dioxins and furans, it is well established that combustion sources rich in chlorinated aromatic compounds (e.g. bushfires, incinerators) are a significant source of airborne emissions of highly toxic chlorinated dibenzodioxins such as 2,3,7,8-tetrachlorodibenzodioxin (TCDD) and other congeners(73). However the toxicity profile of these congeners varies over a wide range, and it is usual to express the aggregate toxicity of a mixture of 'dioxin' congeners in terms of equivalent TCDD toxicity (TEQ), derived using Toxic Equivalency Factors (TEFs) that vary from 1 (for TCDD) to 0.0003 for the less toxic congeners. The profile of dioxin emissions from a brown coal fire relative to other combustion sources is unknown.

Direct inhalation of dioxins from airborne sources generally makes a very small contribution to overall intake in the general population of Australia(74), with more than 95% of exposure occurring through dietary intake of food, mainly fatty foods of animal origin (e.g. meat, dairy). This does not mean that inhalation exposure of Morwell residents to dioxins may not have been significant during the major phase of the fires, but it will be more difficult to assess in the absence of contemporaneous monitoring data. It may be necessary to extend dioxin monitoring to fatty foods sourced in the region, or to contamination of domestic surfaces, where hand-to-mouth contact by children may be a significant mode of systemic exposure.

Final remark:

The effects of biomass and peat smoke studied to date are broadly similar to urban air pollution in terms of mortality, and impacts on the heart, lungs and pregnancy, although the evidence base is much smaller. The main difference is that short term respiratory impacts could be greater with biomass smoke exposure, presumably due to the numerous respiratory irritants in the smoke.

6. Summary of major findings and conclusions

Based on the findings in this review, the following summarises the specific questions posed by the Department of Health:

- *Characterisation of the level of risk to the Morwell community posed by brown coal fire smoke*
    - identifying the hazards and incorporating variable exposures experienced by differing community members

The principal risks to the health of the Morwell community from brown coal fire smoke arise from exposure to fine particulates ($PM_{2.5}$) and carbon monoxide (CO). There does not appear to

be any significant risk from sulphur dioxide (SO_2). The potential risks from other air toxic hazards are currently unknown. Available air quality data have been derived from two stationary monitoring sites in Morwell East and Morwell South and it has not been possible to allow for variability in personal exposures in assessing risks to health in this review.

- explicit reference to subpopulations with differing vulnerabilities

The most vulnerable subpopulations include children (< 5 years old), the elderly (> 65 years old), those with chronic cardio-respiratory diseases such as Ischaemic Heart Disease (IHD), asthma and Chronic Obstructive Pulmonary Disease (COPD), and pregnant women. The likely effects on these groups are detailed in the main body of this report.

- *How does the risk change with persisting exposure; i.e. for periods of 3 weeks, 6 weeks, 3 months, 6 months, 9 months, or 1 year.*

Epidemiological modelling has been undertaken to address this question (see Table 3 on page 15). For combined $PM_{2.5}$ exposures around 250 $\mu g/m^3$ in Morwell South and around the National Environment Protection Measure (NEPM) in the rest of Morwell, no additional deaths would be expected even if the exposure continues for 6 weeks. However, after 3 months this level of $PM_{2.5}$ exposure would be expected to result in some additional deaths from IHD (0.5 additional deaths), Stroke (0.2), COPD (0.1), Lung Cancer (0.1)and Acute Lower Respiratory Infection (ALRI) (0.2).

- *Reports of long-term health outcomes from comparable incidents or circumstances*

No reports relating to any directly comparable previous incidents were able to be identified. Thus, this review has extrapolated from previous studies of the health effects of urban particulate air pollution and biomass smoke.

- *Health outcomes of relevance:*

  - *Mortality*

    A meta-analysis of landmark cohort studies of urban air pollution estimated that excess risk per 10 $\mu g/m^3$ $PM_{2.5}$ was 6.2% (95%CI 4.1 – 8.4%) for all-cause mortality and 10.6% (95%CI 5.4, 16.0%) per 10 $\mu g/m^3$ for cardiovascular mortality. Four studies have found associations between forest fire smoke and all-cause mortality. Most large cohort studies have not found a significant increase in the risk of all cause mortality associated with CO alone.

  - *Morbidity*

    The European Study of Cohorts for Air Pollution Effects (ESCAPE) recently found that a 5 $\mu g/m^3$ increase in estimated annual mean $PM_{2.5}$ was associated with a 13% increased risk of coronary events (95%CI 0.98 to 1.30). Positive associations were detected below the current annual European limit value for $PM_{2.5}$ of 25 $\mu g/m^3$. The most convincing evidence for adverse cardiovascular effects is from short term CO exposures that result in blood carboxy-haemoglobin (COHb) levels ≥ 2.4%, with effects occurring at the lowest levels in subjects with IHD.

- *Cancer*

  The International Agency for Research on Cancer recently concluded that particulate air pollution was a Class 1 human carcinogen. The ESCAPE lung cancer meta-analysis found a pooled hazard ratio (HR) for all lung cancers of 1·18 (0·96–1·46) per 5 μg/m^3 $PM_{2.5}$ and 1·55 (1·05–2·29) for adenocarcinomas of the lung. Indoor smoke from black coal fires is a well recognised risk factor for lung cancer, particularly among non-smoking women in China.

- *Birth outcomes*

  The ESCAPE meta-analysis of mother-child cohort studies found that a 5 μg/m^3 increase in concentration of $PM_{2.5}$ during pregnancy was associated with an increased risk of low birthweight at term (OR 1·18, 95%CI 1·06-1·33). An increased risk was also found for concentrations lower than the present European annual $PM_{2.5}$ limit. Two studies have found small but significant declines in birth weight for babies antenatally exposed during wildfires. There is also some evidence that antenatal CO exposure can be associated with low birthweight and preterm birth.

- *Recommendations for other relevant air toxics which should be measured*

  Measurements are currently being made of Polycyclic Aromatic Hydrocarbons (PAHs), dioxins and furans, formaldehyde and other carbonyls, chemical composition of particles, Volatile Organic Compounds (VOCs) and gaseous mercury. The results of these measurements were not available at the time of this report. However it is not considered that any other air toxics need to be measured.

- *Factors increasing risk*

  See above discussion of vulnerable subpopulations. Others would include lower socioeconomic status and indigenous people. In addition the confounding effects of other known risk factors for these chronic diseases need to be considered. These would include cigarette smoking, obesity, hypertension, hypercholesterolaemia, diabetes mellitus, physical activity, occupational exposures etc.

ACKNOWLEDGMENTS

We are very grateful to the British Columbia Centre for Disease Control for making available a confidential draft of the document: a review of 'health impacts of forest fire smoke exposure' (Reid et al, 2014) specifically for the purpose of this health risk assessment. The air quality data supplied by the EPA were raw data and therefor "EPA does not warrant that the data is error free "

AUTHOR BIOGRAPHIES

Professor Michael Abramson is Deputy Head of the Department of Epidemiology & Preventive Medicine and visiting medical officer in Allergy, Immunology & Respiratory medicine at the Alfred Hospital in Melbourne. He graduated in Medicine from Monash University in 1979 and received his PhD from the University of Newcastle in 1990 for research into occupational asthma. His current research program covers the epidemiology of asthma and chronic obstructive pulmonary disease, including genetic and environmental risk factors such as smoking, air pollution and occupation, and the role of spirometry in managing these conditions. This work has been supported by the National Health & Medical Research Council, Australian Research Council, Department of Human Services, Victorian Health Promotion Foundation, Australian Lung Foundation and Asthma Victoria.

Dr Martine Dennekamp is an occupational and environmental epidemiologist working in the field of air pollution, health and the environment. She was awarded a postdoctoral fellowship from the Centre for Air quality & health Research and evaluation (CAR), an NHMRC Centre of Research Excellence. Martine has an MSc in Environmental Sciences from the Netherlands and a PhD in Environmental Medicine from Scotland which included investigating the health effects of particulate air pollution on patients with chronic lung disease. Her environmental health research program and major interests are in the area of air quality and health, and in particular the association between health effects and smoke exposure from planned burns and bushfires, and the association between ambient air pollution and respiratory and cardiovascular health effects.

Professor Malcolm Sim is an Occupational Physician and epidemiologist who leads a team of 25 research and teaching staff in the Monash Centre for Occupational and Environmental Health (MonCOEH). His main research interests include the risks of chronic diseases from occupational and environmental exposures, with a special emphasis on occupational and environmental risk factors for chronic diseases, such as cancer and respiratory disease, exposure assessment, occupational disease surveillance, veteran health research and the health effects of environmental exposures to mobile phones, small particles, arsenic, pesticides and other environmental contaminants. He is a co-investigator on several NHMRC and ARC grants and other national and international collaborative studies. Malcolm is the Editor-in-Chief of Occupational and Environmental Medicine, a specialty journal of the BMJ.

A/Prof Manoj Gambhir is Head of the Epidemiological Modelling Unit, Department of Epidemiology & Preventive Medicine at Monash University. He has been involved in modelling and forecasting for policy in such diverse areas as neglected tropical diseases, HIV Aids and infectious diseases including influenza, pertussis, mumps, measles, rabies and cholera. He has worked at the US Federal Government Centers for Disease Control and Prevention (CDC) based in Atlanta since 2010, where he helped create the National Center for Immunization and Respiratory Diseases (NCIRD) Modelling Unit. His work there involved him in a range of dramatic health events, from targeting outbreaks of respiratory disease in the Middle East to tracing swine influenza illnesses in pig barns at US state fairs. His work at NCIRD included public health analysis and modelling for research and policy and collaboration with US government agencies, academics and the wider community on policy advice.

Professor Brian Priestly is a Professorial Fellow (now part-time) in the Department of Epidemiology & Preventive Medicine (DEPM) at Monash University and Director of the Australian Centre for Human Health Risk Assessment (ACHHRA). ACHHRA's core objective is to provide a national focus for human health risk assessment in the area of food and environment pollutants, and to contribute to workforce development by mounting training programs in health risk assessment. Brian's primary area of expertise is in toxicology. He was recently recognised as a Fellow (FACTRA) in the peer-reviewed register of the Australasian College of Toxicology & Risk Assessment. He has been active on many government technical committees and scientific advisory panels over the past thirty years. He is currently a Science Fellow advising the food (FSANZ), AgVet chemical (APVMA) regulators, as well as being an advisor to the industrial chemical (NICNAS) regulator on toxicological issues, including those relating to the regulation of nanoscale materials.

Dr Fay Johnston is an environmental epidemiologist at the Menzies Research Institute Tasmania. Her research focuses on the public health impacts of bushfires, heat events and outdoor air pollution from biomass combustion. She has studied the health impacts of landscape fire smoke for 15 years and has led several national and international research programs in this field including the global burden of disease modelling of deaths attributable to landscape fire smoke. She is lead investigator of the current Bushfire CRC/DEPI funded program of research 'Smoke impacts on community health and social perceptions' and the ARC funded 'Bushfires, smoke, and people: the risks and benefits from planned burning on the urban–rural interface'. She contributes to an international expert group developing current evidence-based guidelines for the management of severe smoke events form forest fires and was a founding member of the US NSF and NCEAS funded international pyrogeography network. She is also a public health physician and Specialist Medical Advisor, Public and Environmental Health, Department of Health and Human Services, Tasmania.

Dr Lisa Demos is a Clinical pharmacologist and her current positions include Senior Research Fellow, Department of Epidemiology & Preventive Medicine, Monash University and Senior Research Scientist with the Australian Disease Management Association. Lisa qualified as a pharmacist in 1977 and then went on to undertake post graduate studies completing her PhD in1990 in the department of Clinical Pharmacology. Over the years she has work on a very broad range of studies and projects from clinical drug trials, drug use evaluations to government reviews on water fluoridation and health effects of alcohol.

Professor John McNeil has been the head of the Monash University School of Public Health and Preventive Medicine based at the Alfred Hospital in Prahran since 1986. His research background is in epidemiology and clinical pharmacology. He has served on the senior medical staff of the Austin and Alfred Hospitals and the Monash Medical Centre. He is currently CIA of the NIH funded ASPREE study, a large scale randomized clinical trial of low-dose aspirin in persons over 70 years of age. He has a long history of involvement in clinical trials addressing preventive options in heart disease, renal disease, anesthesia, stroke and eye disease. He has published over 350 refereed publications. He has also been instrumental in the development of large-scale clinical registries to improve the measurement and benchmarking of clinical outcomes.

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MONASH University

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From:	Heffernan, Emily (AU)
To:	"Philip McCloud [redacted]"
Cc:	Fox, Chris (AU)
Subject:	FW: Hazelwood Mine Fire Inquiry [KWM-Documents.FID1770820]
Attachments:	image003.jpg image002.jpg Re Hazelwood Inquiry.msg RE Hazelwood Inquiry.msg

Dear Philip,

Further to our letter dated 6 October 2015, please find **attached** email correspondence from Professor Armstrong and Dr Flander, received from the Board today.

Kind regards,

Emily Heffernan | Senior Associate
King & Wood Mallesons
[redacted]

From: Justine Stansen [redacted]
Sent: Tuesday, 13 October 2015 3:47 PM
To: [redacted] Heffernan, Emily (AU); Fox, Chris (AU); [redacted]; Ariane Wilkinson; Felicity Millner; Robert Perry
Cc: Ruth Shann; Peter Rozen
Subject: RE: Hazelwood Mine Fire Inquiry

Dear all

I refer to my email below. Please see **attached** emails from Dr Flander and Professor Armstrong.

Kind regards

Justine Stansen
Principal Legal Advisor
Hazelwood Mine Fire Inquiry
[redacted]

From: Justine Stansen
Sent: Monday, 12 October 2015 3:43 PM
To: [redacted] 'Heffernan, Emily (AU)'; 'Fox, Chris (AU)'; [redacted]; 'Ariane Wilkinson'; 'Felicity Millner'; 'Robert Perry'
Cc: 'Ruth Shann'; 'Peter Rozen'
Subject: Hazelwood Mine Fire Inquiry

Dear all

Further to email correspondence and my letters to you sent 30 September 2015, I confirm that the Board is sitting at 9am on Thursday, 15 October 2015 at Level 11, 222 Exhibition Street, Melbourne in Court 2. The Board will hear further evidence from Associate Professor Barnett, Professor Armstrong, Professor Gordon and Dr Flander.

The Board has circulated to you the emails and documents sent and received from Associate Professor Barnett, Professor Armstrong and the Secretariat to date. The same has been provided to Dr Flander and Professor Gordon through Environmental Justice Australia (see **attached**). The Board has requested that Dr Flander and Professor Gordon provide a short report to the Board by tomorrow afternoon. Those reports will be circulated to all parties as soon as they are received. Professor Armstrong has been requested to provide any further comments to the last email received from Associate Professor Barnett by tomorrow afternoon also. Counsel Assisting intends to tender each of documents and correspondence at the further hearing.

The Board has received correspondence from some of the parties in relation to the further hearing. The letter received from solicitors for GDF Suez dated 6 October 2015 was circulated to the parties on 8 October 2015 together with a response from Associate Professor Barnett to questions posed in that letter. The Board has also received correspondence from the solicitors for Dr Lester. Copies of those letters, together with my response, are **attached**.

It has been suggested to the Board by the solicitors for Dr Lester that it also hear from Professor Abramson in the hearing on Thursday. The Board declines that suggestion on the basis that at no stage has Professor Abramson reviewed the Births Deaths and Marriages data and conducted any statistical analysis of same (see T363:21-29). It is not appropriate for Professor Abramson to give evidence on the subject matter.

The solicitors for Dr Lester also ask for clarification on two further matters:

1. What communication took place between 11 and 15 September 2015 between the Secretariat and Associate Professor Barnett; and
2. What communications were had with Associate Professor on or around 31 August 2015 in relation to the provision of the Births Deaths and Marriages data.

In answer to the first question, copies of all email correspondence have been provided. As is

referenced in the email from Associate Professor Barnett sent 15 September 2015, Counsel Assisting had a brief conversation on that day in which she indicated that the work completed by Associate Professor Barnett should be sent to the Secretariat rather than having it published due to the sensitivity of the data upon which it relied.

In answer to the second question and as noted in my letter dated 9 October 2015 to the solicitors for Dr Lester, it became apparent at the joint expert meeting that Associate Professor Barnett had not been provided with this data when undertaking his earlier reports. In order to ensure that each of the experts had the same data, a copy of the data was downloaded on a USB and provided to Associate Professor Barnett at the completion of the meeting. Associate Professor downloaded the data and handed back the USB. No correspondence was provided.

I will circulate in advance a list of the documents that Counsel Assisting will seek to tender at the further hearing.

Please contact me if you have any question in relation to the further hearing.

Kind regards

Justine Stansen
Principal Legal Advisor
Hazelwood Mine Fire Inquiry
[redacted]

From: Louisa Flander
To: Justine Stansen
Subject: Re: Hazelwood Inquiry
Date: Tuesday, 13 October 2015 3:28:59 PM
Attachments: image001.jpg

Dear Justine

My short report on the fourth paper (Barnett, 25 September 2015) follows:

1. The methods used in this analysis appear to be correct.
2. The results presented in this analysis appear to be correct, subject to the following reservations about the way results are presented:
3. I have concerns about the lack of communication of uncertainty around estimates in this report. I suggest the following statements may be a fuller and more accurate representation of the results:

    1. The analysis shows 99% confidence that there were more than zero additional deaths associated with the 45 days of the fire, beyond the usual fluctuations captured in the model.
    2. The analysis shows 95% confidence (in the form of credible intervals) that between 2 and 46 additional deaths were associated with the 45 days of the fire, beyond the usual fluctuations captured in the model (Table 2).
    3. The best estimate of the analysis is 23 additional deaths associated with the 45 days of the fire, beyond the usual fluctuations captured in the model (Table 2).

4. The increase in explanatory power in this analysis may be due to the inclusion of the postcode as an extra predictor variable. One of the 'usual fluctuations' captured in this model is that daily deaths tend to be higher in postcodes 3825 (Moe) and 3840 (Morwell), and tend to be lower in postcodes 3842 (Churchill) and 3844 (Traralgon) across the entire data set.
5. Temperature may also be a useful variable for explaining fluctuations, but given its absence from Table 2 and its presentation in Figure 3, I cannot tell whether it is statistically significant in this analysis. I do not see any representation of uncertainty around the relative risk values plotted in Figure 3.
6. Given that the results are based on a much larger dataset compared to previous reports, covering the period from 1 January 2009 to 31 December 2014, some discussion of the effect this may have on the improved estimate of additional mortality is warranted.

Kind regards
Louisa

Dr Louisa Flander
[redacted]

Senior Research Fellow,
Centre for Epidemiology & Biostatistics
Melbourne School of Population & Global Health,
Room [redacted]
[redacted]

[redacted]

From: Justine Stansen <[redacted]>
Date: Monday, 12 October 2015 3:54 pm
To: Louisa Flander <[redacted]>
Subject: RE: Hazelwood Inquiry

Thank you Louisa.

From: Louisa Flander [redacted]
Sent: Monday, 12 October 2015 3:52 PM
To: Justine Stansen
Subject: Re: Hazelwood Inquiry

Dear Justine

I will provide a short report (in the form of my email response) in relation to the Barnett report dated 25 September 2015, by 4pm 13 October,

Kind regards
Louisa

Dr Louisa Flander
[redacted]

Senior Research Fellow,
Centre for Epidemiology & Biostatistics
Melbourne School of Population & Global Health,
Room 321/207 [redacted]
[redacted]
[redacted]

From: Justine Stansen [redacted]
Date: Monday, 12 October 2015 12:54 pm
To: Louisa Flander [redacted]
Subject: RE: Hazelwood Inquiry

Dear Louisa

Further to my email below, please see **attached** correspondence from Associate Professor Barnett and Professor Armstrong.

The Board would be grateful if you could provide a short report in relation to the fourth report of Associate Professor Barnett dated 25 September 2015 and any other matter you think would be useful to the Board. The Board is interested in your opinion as to whether you agree or disagree with the methodology used and conclusions reached by Associate Professor Barnett. It would be grateful if your report could be provided by 4pm, tomorrow (13 October 2015). Please let me know if you can accommodate this request.

If you have any questions, please do not hesitate to contact me.

Kind regards

Justine Stansen
Principal Legal Advisor
Hazelwood Mine Fire Inquiry
[redacted]

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From: Justine Stansen
Sent: Thursday, 1 October 2015 10:43 AM
To: [redacted]
Subject: Re: Hazelwood Inquiry

Thanks Louisa

Sent by Outlook for Android

On Wed, Sep 30, 2015 at 3:12 PM -0700, "Louisa Flander" [redacted] wrote:

Dear Justine
I confirm receipt of the additional materials, and my availability for 15 October 2015,
Kind regards
Louisa

Dr Louisa Flander
[redacted]

Senior Research Fellow,
Centre for Epidemiology & Biostatistics

Melbourne School of Population & Global Health,
[redacted]

From: Justine Stansen [redacted]
Date: Wednesday, 30 September 2015 8:45 pm
To: Louisa Flander [redacted]
Subject: Hazelwood Inquiry

DearLouisa

I refer to Term of Reference 6 and the recent public hearings held on 1-3 and 9 September 2015. During the course of those hearings two reports prepared by Associate Professor Barnett were tendered.

On 11 September 2015, Associate Professor Adrian Barnett contacted the Secretariat and indicated that he was undertaking further analysis of the daily death data provided to him prior to the hearing and that he intended to produce a further report that he wished to publish.

On 15 September 2015, Associate Professor Barnett provided that third report to the Board. On 17 September 2015, the Board sought the views of Professor Armstrong concerning the third report of Associate Professor Barnett. Professor Armstrong's comments in relation to the third report were provided to the Board on 18 September 2015 and were forwarded to Associate Professor Barnett by the Board in an email dated 24 September 2015. On 25 September 2015, Associate Professor provided a fourth report to the Inquiry.

Copies of the correspondence described above and the third and fourth reports of Associate Professor Barnett are **attached**. Copies of the reports and the correspondence will also be provided to all experts who gave evidence at the hearing in relation to Term of Reference 6.

The Board will holding a short further hearing to consider this additional evidence held on **15 October 2015 from 9.00 am** in Melbourne. The hearing will take place on level 11, 222 Exhibition St Melbourne. The Board requests that all experts who gave evidence in the early September hearing appear again as witnesses as a panel and will be questioned about this new material by Counsel Assisting and any other party.

I would be grateful if you could confirm that you are available to appear at the hearing on 15 October 2015.

If you have any questions about the above, please contact me.

Kind regards

Justine Stansen
Principal Legal Advisor
Hazelwood Mine Fire Inquiry
[redacted]

From: Bruce Armstrong
To: Justine Stansen
Subject: RE: Hazelwood Inquiry
Date: Monday, 12 October 2015 10:10:37 PM
Attachments: image001.jpg

Thank you Justine. I have no further comments.

Bruce

BRUCE ARMSTRONG
Emeritus Professor, School of Public Health
THE UNIVERSITY OF SYDNEY
Senior Adviser
THE SAX INSTITUTE
Chairman
BUREAU OF HEALTH INFORMATION

CONTACT INFORMATION
[redacted]

From: Justine Stansen [redacted]
Sent: Monday, 12 October 2015 2:17 PM
To: Bruce Armstrong
Subject: Hazelwood Inquiry

Dear Bruce

Please see attached email from Associate Professor Barnett. If you have any additional comments to make in relation to the attached, could you please provide them by 4pm tomorrow (13 October 2015).

Many thanks

Justine Stansen
Principal Legal Advisor
Hazelwood Mine Fire Inquiry
[redacted]

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